Colleen E. Carney
Jack D. Edinger

Insomnia
and Anxiety

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Series in Anxiety and Related Disorders

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Martin M. Antony, Professor, Department of Psychology,
Ryerson University, Toronto, Ontario, Canada

Colleen E. Carney · Jack D. Edinger

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Colleen E. Carney
Department of Psychology
Ryerson University
Toronto ON
Canada
ccarney@psych.ryerson.ca

Jack D. Edinger
Department of Psychiatry and Behavioral
Sciences
Duke University Medical Center
Durham, NC
USA
jack.edinger@duke.edu

For Shannon and Sydney
                           Colleen E. Carney

In loving memory of my parents who
always encouraged my best efforts and
to my wife Wanda who tolerated those
well over the years.
                           Jack D. Edinger

Acknowledgements

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Chapter 1
Anxiety and Insomnia: An Overview

Abstract Living chronically with insomnia can generate considerable anxiety and interfere with the quality of life. Of the most commonly reported health-related problems, insomnia is rated as among the most frequently reported complaints (Acta Psychiatrica Scandinavica 96:287–294, 1997). While insomnia is a prevalent, distressing, and significant disorder in its own right, it is a problem that occurs frequently in the context of another serious psychological disorder. For example, the presence of insomnia is associated with the eventual development of an Anxiety Disorder in one quarter of insomnia sufferers (Journal of the American Medical Association 262:1479–1484, 1989). Both insomnia (Sleep 17:630–637, 1994; Journal of the American Medical Association 247:997–1103, 1982; American Journal of Psychiatry 145:346–349, 1988) and anxiety disorders (Journal of Abnormal Psychology 99:308–312, 1990) often occur with another comorbid disorder, which would be expected to further compound their costliness. Anxiety and insomnia exert complex dynamic effects upon each other. It is for this reason that we write a book for clinicians to understand the overlap and develop effective treatment strategies to address sleep problems in this context. In this first chapter, we consider diagnostic considerations for insomnia when it cooccurs with anxiety symptoms and anxiety disorders and discuss the limitations of viewing insomnia as a secondary symptom when it cooccurs with other conditions such as anxiety disorders. We also propose a Cognitive Behavioral model of insomnia and anxiety. Thus, this chapter provides a preliminary introduction to insomnia and the cooccurrence of anxiety and anxiety disorders, whereas the subsequent chapters provide a more in-depth exploration of this complex and poorly understood relationship.

It is understandably distressing to experience a sustained impairment in the ability to initiate or maintain sleep. Living chronically with this condition known as insomnia can generate considerable anxiety and interfere with the quality of life. Of the most commonly reported health-related problems, insomnia is rated as among the most frequently reported complaints (Canals, Domenech, Carbajo, & Blade, 1997). Prevalence rates for chronic insomnia vary by age and are somewhere between 10%

and 20% of the general population (Foley, Monjan, & Brown, 1995; Mellinger, Balter, & Uhlenhuth, 1985). Although insomnia diagnoses occur across all age groups, such diagnoses are alarmingly common in middle-aged to older adults (Ohayon, 2002; Ohayon, Zulley, Guilleminault, Smirne, & Priest, 2001). While insomnia is a prevalent, distressing, and significant disorder in its own right, it is a problem that occurs frequently in the context of another serious psychological disorder. For example, the presence of insomnia is associated with the eventual development of an Anxiety Disorder in one quarter of insomnia sufferers (Ford & Kamerow, 1989). Both insomnia (Buysse et al., 1994; Coleman et al., 1982; Jacobs, Reynolds III, Kupfer, Lovin, & Ehrenpreis, 1988) and anxiety disorders (Sanderson, Di Nardo, Rapee, & Barlow, 1990) often occur with another comorbid disorder, which would be expected to further compound their costliness. For example, those with comorbid anxiety and insomnia have a poorer reported quality of life than those with anxiety only (Ramsawh, Stein, Belik, Jacobi, & Sareen, 2009). Anxiety and insomnia symptoms and disorders cooccur frequently and exert complex dynamic effects upon each other. It is for this reason that we write a book for clinicians to understand the overlap and develop effective treatment strategies to address sleep problems in this context.

In this chapter, we consider diagnostic considerations for insomnia when it cooccurs with anxiety symptoms and anxiety disorders, and we discuss the limitations of viewing insomnia as a "secondary" symptom when it occurs comorbid to other conditions such as anxiety disorders. We also propose a Cognitive Behavioral model of insomnia and anxiety. The purpose of this chapter is to provide a preliminary introduction to insomnia and the cooccurrence of anxiety and anxiety disorders, whereas the subsequent chapters provide a more in-depth exploration of this complex and poorly understood relationship.

What is Insomnia?

Primary Insomnia (PI), in the absence of psychiatric or other medical disorders, is costly in its own right. For example, it substantially increases health care utilization and related costs and accounts for as many as 3.5 disability days per month (Hajak & SINE Study Group Study of Insomnia in Europe, 2001; Simon & VonKorff, 1997; Weissman, Greenwald, Nino-Murcia, & Dement, 1997). PI has been linked to several other poor outcomes including decreased occupational productivity and increased occupational accidents, increased alcohol consumption, and a general sense of poor health-related quality of life (Gislason & Almqvist, 1987; Johnson, Roehrs, Roth, & Breslau, 1998; Katz & McHorney, 1998). There is a cause for special concern for insomnia in older adults as insomnia occurs in a staggering 57% of this age group (Foley et al., 1995), and it is linked to serious falls even after controlling for age, gender, the use of prescription medications, depression, and visual or mobility impairments (Brassington, King, & Bliwise, 2000). Given these considerations, PI warrants timely, effective, and enduring treatment, yet it is well-documented that insomnia too often goes undetected and untreated (Leger, 2005).

Diagnostic Considerations for Insomnias

The disorder called Primary Insomnia was first described in the revised, third edition of the American Psychiatric Association's Diagnostic and Statistical Manual – DSM-III-R (American Psychiatric Association, 1987) and remains in the present-day updated version of this manual (American Psychiatric Association, 1997). The diagnostic criteria require that the sleep complaint (e.g., sleep onset or sleep maintenance difficulties, or nonrestorative sleep) is predominant, and lasts at least one month. There are no established cutoffs for how quantitatively disrupted sleep must be to receive this diagnosis. The convention often employed is that the time spent awake at the beginning of the sleep period or in the middle of the night is greater than 30 min, and that such onset or maintenance problems occur three or more nights per week (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003). Despite this convention, the DSM has not yet adopted these guidelines. Interestingly, an attempt to quantify a frequency was unsuccessful because of wide variability in insomnia complaints (Lineberger, Carney, Edinger, & Means, 2006). Thus, someone with insomnia could have only one impressively poor night of sleep (e.g., spending 3 or more hours awake on bed once or twice per week), moderately disturbed sleep (e.g., half the nights are characterized by an hour or so of sleep loss), or frequent mild disruptions (e.g., taking 30 min to fall asleep every night). Unlike many other sleep disorders (e.g., sleep-disordered breathing, periodic limb movement disorder and some parasomnias), PI is a subjective condition, that is, the diagnosis can be made from clinical interview and self-reported tools, rather than expensive overnight polysomnographic studies (American Sleep Disorders Association, 1995).

In addition to the presence of a subjective sleep complaint, the disturbance must cause clinically significant distress or impairment in social, occupational, or other areas of functioning. This criterion would typically reflect one or more of the daytime symptoms of insomnia listed in the Research Diagnostic Criteria for Insomnia (Edinger et al., 2004), such as: fatigue/malaise, attention/concentration problems, negative mood, social/vocational dysfunction or poor school performance, somatic symptoms such as tension headaches and/or gastrointestinal symptoms in response to sleep loss, motivation/energy/initiative reduction, daytime sleepiness, and/or worry about sleep. Whilst some clinicians are not accustomed to thinking about such symptoms as part of insomnia; in actuality, insomnia is best characterized as a disorder with manifestations throughout the 24-h period.

The remaining criteria for insomnia relate to exclusions, or differential diagnosis. For instance, the insomnia cannot occur *exclusively* during another sleep disorder (e.g., narcolepsy, breathing-related sleep disorder, circadian rhythm disorder or parasomnia), or another mental disorder (e.g., major depressive disorder, Generalized Anxiety Disorder). Lastly, it cannot be due to the physiologic effects of a substance (e.g., medication or drug of abuse) or a general medical condition. These criteria specify that a PI diagnosis is assigned when the insomnia does not occur *exclusively* during the course of another primary sleep or mental disorder and is not the direct

result of a general medical disorder or substance use/abuse. The word exclusively is important, as it is possible and common to have a comorbid condition accompanying the insomnia. Thus, an insomnia diagnosis can, and should be made in the presence of another mental, medical, or sleep disorder, if there is a prominent insomnia complaint that causes distress or impaired functioning. The following case examples exemplify how individuals with PI and coexisting anxiety disorders may present clinically:

> Mr. B was a 43-year-old professor who complained of sleep onset insomnia for the past 11 years. About 8 years ago, he began suffering from panic attacks. He acknowledged that there might be some relation between the panic attacks and his insomnia (e.g., he was occasionally more likely to experience a panic attack after a poor night of sleep). He was treated with medication and ceased having panic attacks, but his insomnia persisted. The panic attacks later returned and he continues to suffer from both conditions.

> Ms. K was a 23-year-old student who suffered from OCD but denied any relation of this condition to her sleep. She did not report any nighttime rituals that interfered with her ability to fall asleep, and she did not appear to be any more likely to sleep poorly on a day, characterized by significant anxiety and compulsive behaviors. There were periods of apparent remission of obsessions and compulsions, but her sleep problem persisted.

The Case of Comorbid Insomnia: More Nosological Issues

As noted in the previous section, PI can occur seemingly independently from a coexisting disorder and also can persist during periods of remission from another coexisting disorder. If the insomnia is a prominent clinically significant complaint and does not occur *exclusively* during the comorbid disorder, a diagnosis of PI is made. In cases wherein clinically significant insomnia occurs exclusively within the context of a cooccurring disorder (i.e., the person is complaining about his/her sleep and resultant daytime symptoms, and the insomnia waxes and wanes with the comorbid condition), it may be appropriate to diagnose Insomnia Related to Another Axis I Disorder (IMD).

The relationship between insomnia and anxiety is complex. Insomnia can cause anxiety, and experiencing a chronic anxiety problem also can interfere with the ability to sleep. Inducing worry in nonworrying good sleepers by telling them that they will have to give a speech upon waking significantly increases the time it takes to fall asleep (sleep onset latency) (Gross & Borkovec, 1982). Stress-induced worry also decreases slow (delta) wave intensity in the first sleep cycle of normal subjects (Hall, Buysse, Reynolds, Kupfer, & Baum, 1996), and this physiological change tends to make sleep subjectively "lighter" and more objectively disrupted. Similarly, inducing poor sleep artificially with the administration of a stimulant increases next-day anxiety (Bonnet & Arand, 1992). It is fairly common for those with clinical levels of anxiety to report insomnia, and the rate of comorbid anxiety disorders in those with insomnia is approximately 24% (Ford & Kamerow, 1989).

Whereas previous psychiatric conceptualizations viewed insomnia as a mere symptom of psychiatric disorders, contemporary viewpoints recognize the ample

evidence for insomnia as an often comorbid, treatment-worthy condition. Anxiety disorders often precede the onset of insomnia (Johnson, Roth, & Breslau, 2006; Ohayon & Roth, 2003), but insomnia also can occur prior to the onset of anxiety disorders suggesting a possible etiological role of sleep disturbance for some with such conditions. For example, the presence of insomnia is predictive of future anxiety disorders ascertained one to 45 years after the baseline interview even after other significant predictors were statistically controlled (Ford & Kamerow, 1989; Livingston, Blizard, & Mann, 1993).

Traditionally, PI and Insomnia Related to a Mental Disorder (IMD) or so-called secondary insomnia have been considered separate disorders. However, recently the need for this diagnostic distinction has been debated. Distinguishing between primary and secondary insomnias may have led to a traditional clinical misconception that the insomnia of those with comorbid conditions is merely a product of the larger comorbid disease process (e.g., a secondary condition) that fails to merit separate diagnostic or treatment attention. Arguably, such a conceptualization has also led to a lack of proper treatment attention and possible diagnostic problems. Clinical practice studies reveal a lack of attention for sleep complaints, and a misunderstanding of the predominant features of various insomnia diagnoses (Haponik, Frye, Richards, Wymer, & Hinds, 1996). For example, in a survey of over 3,000 general practitioners, the most common symptom used to identify depression was the presence of a sleep complaint or insomnia (Krupinski & Tiller, 2001). In the same study, only 28% of those diagnosed with MDD actually met DSM-IV criteria for this condition; the remaining patients were incorrectly diagnosed with MDD when they actually had untreated insomnia. It is unclear as to how often this occurs in anxiety disordered clients. We examined this question more broadly by examining those with insomnia with an Axis I Mental Disorder (IMD) and those with insomnia only (PI) (Carney, Edinger, Krystal, Stepanski, & Kirby, 2005). One third of these insomnia patients were diagnosed by sleep specialists via clinical interview with Insomnia Related to a Mental Disorder. These sleep experts did not have access to Structured Clinical Interview for DSM Disorders (SCID) information – instead they had access to a range of sleep related information such as sleep logs, polysomnography, and a sleep history questionnaire. Of those diagnosed with IMD, almost one third did *not* have a SCID-verified disorder on Axis I (despite the fact that a mental disorder is needed to give an IMD diagnosis). These sleep experts did not have access to the insomnia patient's scores on instruments such as the Beck Anxiety Inventory (BAI) or the Penn State Worry Questionnaire (PWSQ), but a multivariate analysis of these measures suggested that these two groups indeed differed (that is, the Axis I group had significantly higher scores that were all above the clinical cutoff for the measures, and those without an Axis I disorder had statistically lower scores and the means were below the clinical cutoffs for these measures). Interestingly, the groups did not differ on a measure of worry about sleep; the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS). The DBAS worry scores were in the pathological range, which suggests that the high level of worry about sleep may be interpreted by some to be a sign of more global pathology when it is in fact, merely a daytime symptom of insomnia (Fig. 1.1).
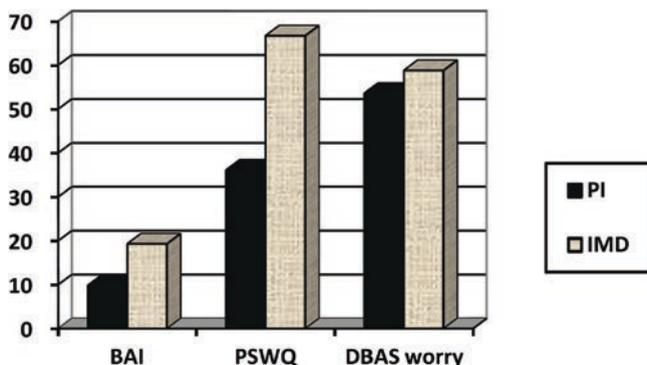
Fig. 1.1 Anxiety measure scores are not pathological unless there is a comorbid Axis I disorder

Other studies have also suggested that there can be misconceptions about insomnia sufferers. For example, in a multisite evaluation of factors considered in differential diagnosis for insomnia (Nowell et al., 1997), the presence of a mental disorder tended to preclude consideration of concomitant sleep disorder diagnoses such as Primary Insomnia, or Psychophysiological Insomnia, whereas the presence of conditioned arousal or poor sleep habits tended to preclude consideration of an insomnia "related to a mental disorder" diagnosis. Although conditioned arousal and poor sleep habits could play a significant role in the insomnia of those with comorbid problems such as anxiety disorders, this possibility tends to be ignored by clinicians.

The utility of a primary versus secondary distinction also may be questionable in light of the following evidence: (1) insomnia can precede Axis I disorders (Ford & Kamerow, 1989; Weissman et al., 1997); (2) insomnia can effectively be treated without necessarily treating the comorbid disorder (Lichstein, Wilson, & Johnson, 2000); (3) insomnia may be a risk factor for other disorders, including anxiety disorders (Ford & Kamerow, 1989); and (4) as many as half of individuals complain of residual, persistent insomnia after remission from the so-called "primary" disorder (e.g., PTSD) (Zayfert & DeViva, 2004). Thus, distinguishing between a primary versus secondary insomnia may be more of a theoretical than a practical enterprise (Lichstein, 2000).

Moreover, the relegation of insomnia to a secondary condition has dictated treatment practices that are questionable. Conventional wisdom was to treat the presumed primary condition and the insomnia would purportedly resolve. However, clinically significant levels of so-called "residual insomnia" remain in about half of people successfully treated for their primary disorder only. For example, rates of residual insomnia in depression have been reported as high as 53%, and there does not appear to be any difference in residual insomnia rates between CBT and pharmacotherapy for depression (Carney, Segal, Edinger, & Krystal, 2007). The residual insomnia rate for PTSD is similarly high (Zayfert & DeViva, 2004). Also, insomnia treatment in those with comorbid Axis I disorders has typically

shown improvement in the accompanying condition, and combined approaches have shown some superiority to treating the so-called primary condition only (Edinger, Wohlgemuth, Krystal, & Rice, 2005; Manber et al., 2008; Pollack et al., 2008). Thus, the problem of residual insomnia and support for the superiority of combination (sleep and the comorbid condition) treatment approaches casts doubt on the practice of ignoring insomnia.

A particularly useful way of thinking about insomnia and comorbid conditions is that insomnia can be partially, independently, or speciously related to the other condition (Lichstein et al., 2000). This means that the etiological significance of insomnia in those with anxiety disorders can vary considerably. In some, insomnia may truly represent an *absolute* secondary sleep difficulty caused by an anxiety process and eliminated by successful anxiety disorder treatment. In others, the insomnia/anxiety relationship is only *partial* as the two conditions have some, albeit not total independence. In such cases, successful anxiety-targeted therapy might be expected to reduce but not eliminate presenting insomnia symptoms. Still in others, the insomnia/anxiety relationship is *specious* in that the comorbidity of two essentially independent conditions is mistaken for causality. In this latter case, anxiety-focused therapies would be expected to have little, if any, effect on presenting sleep difficulties. This conceptualization has heuristic appeal for explaining various etiologic pathways and treatment outcomes for sleep difficulties presented by those with anxiety disorders. However, in practice, it is often difficult to identify the various subtypes this taxonomy proposes given the frequent unreliability of patients' historical reports and the lack of any objective assay to discriminate them from each other. Moreover, the manner in which insomnia and anxiety interact remains poorly understood, so it appears most appropriate to consider insomnia a comorbid condition that may often warrant special recognition and separate treatment attention in those with anxiety.

Cognitive Behavioral Model of Insomnia and Anxiety

Now, we turn from nosology to a proposed cognitive-behavioral model of etiology for insomnia and anxiety. If we integrate the ideas included in several conceptual models of insomnia (Harvey, 2002; Lichstein, 2000; Spielman, Caruso, & Glovinsky, 1987), we can envisage multiple different interacting or independent paths of risk for developing diagnoses or symptoms of insomnia in the context of anxiety. We will discuss Spielman's model more thoroughly in the next chapter, but etiology in insomnia is commonly regarded in diathesis-stress model terms. That is, there are predisposing/diathetic factors in insomnia that in the absence of a precipitating stressor would not be expected to result in exceeding the clinical threshold of insomnia. However, the occurrence of a physical, environmental, or psychological stressor could be disruptive enough to precipitate an episode of insomnia. Such stressors could include experiencing a positive or negative life event, an environmental change such as the introduction of a noisy roommate, multiple time-zone

travel, interpersonal conflict, or a physical or mental health condition (such as an anxiety disorder). An important element of Spielman's model is the consideration that reactions to the sleep problem tend to become important perpetuating factors in insomnia. Negative thoughts and emotions, selective attention and monitoring of sleep-threat related stimuli, and safety behaviors that undermine sleep regulation and reinforce maladaptive beliefs about sleep all may sustain an insomnia problem (Harvey, 2002). Thus, the model in Fig. 1.2 captures the idea that perpetuating and precipitating factors are important in the initial onset of sleep disruption, but the core of the model focuses on perpetuating factors in insomnia like a repetitive thought process and maladaptive coping behaviors. Certainly, the presence of an anxiety disorder could be a stressor that causes the initial sleep disruption to occur. In such a case, we might expect the sleep disruption to resolve when the stressor resolves; that is, we might not expect chronic insomnia. We would expect chronic insomnia when perpetuating factors are activated. For example, whereas an anxiety disorder may be the original impetus (stress) for sleep disruption, an individual's subsequent efforts to compensate for lost sleep could have a negative effect on the sleep regulatory system and perpetuate a chronic (and comorbid) insomnia. In such a case, we might anticipate the problem of residual insomnia after remission from the treated anxiety disorder. Although the original stressor may have been removed (i.e., via recovery from the anxiety disorder), there remains important untreated perpetuating factors that sustain the insomnia problem. We say untreated because as you will see in subsequent chapters, anxiety disorder treatments do not address the thoughts and behaviors that lead to sleep dysregulation. Cognitive Behavior
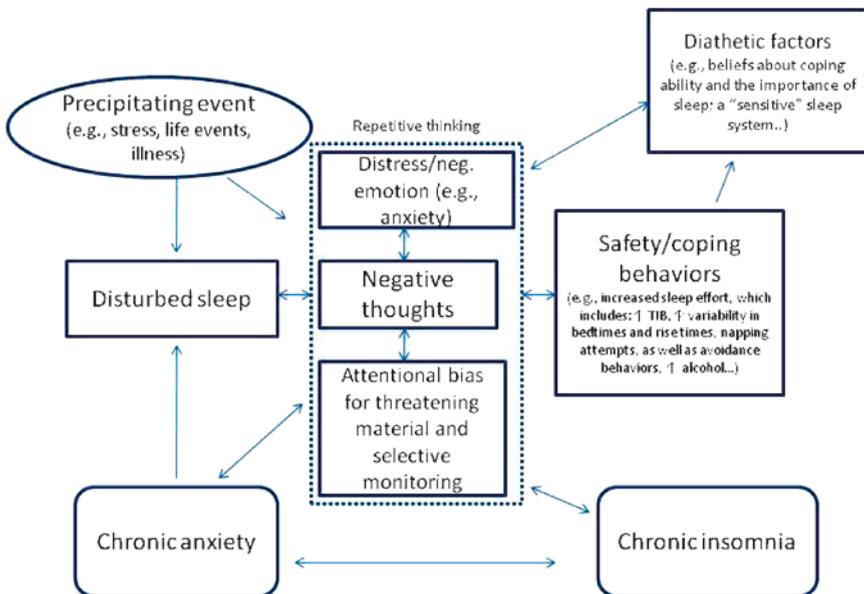
Fig. 1.2 Proposed model for relation between anxiety and insomnia

Therapy for insomnia primarily targets the perpetuating causes of insomnia, as these proximal factors become the primary reason for the insomnia's continuation. Some more distal, predisposing factors (e.g., maladaptive beliefs about sleep) may also act as perpetuating factors, and are thus also targeted. Lastly, in cases wherein the initial stressor was not an anxiety disorder and a chronic insomnia ensues (via perpetuating factors), the experience of chronic distress/anxiety, worry about sleep and functioning could potentially act as a stressor that could activate a more general anxiety process and lead to the development of an anxiety disorder.

Summary

There is a close relationship between insomnia and anxiety. Unfortunately, our understanding of this relationship has been hindered by faulty assumptions (i.e., insomnia is always a mere symptom). This is unfortunate given that insomnia can be effectively treated in the context of a variety of comorbid disorders. Subsequent chapters will elaborate further on the perpetuating factors of insomnia in anxiety disorders and how to treat them with CBT for insomnia.

References

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1997). *Diagnostic and statistical manual of mental disorders, 4th edition, text revision (DSM-IV-TR)* (4th ed.). Washington, DC: American Psychiatric Association.

American Sleep Disorders Association. (1995). Practice parameters for the use of polysomnography in the evaluation of insomnia. *Sleep, 18*(1), 55–57.

Bonnet, M. H., & Arand, D. L. (1992). Caffeine use as a model of acute and chronic insomnia. *Sleep, 15*(6), 526–536.

Brassington, G. S., King, A. C., & Bliwise, D. L. (2000). Sleep problems as a risk factor for falls in a sample of community-dwelling adults aged 64–99 years. *Journal of the American Geriatrics Society, 48*, 1234–1240.

Buysse, D. J., Reynolds, C. F., III, Kupfer, D. J., Thorpy, M. J., Bixler, E., Manfredi, R., et al. (1994). Clinical diagnoses in 216 insomnia patients using the International Classification of Sleep Disorders (ICSD), DSM-IV and ICD-10 categories: A report from the APA/NIMH DSM-IV field trial. *Sleep, 17*, 630–637.

Canals, J., Domenech, E., Carbajo, G., & Blade, J. (1997). Prevalence of DSM-III-R and ICD-10 psychiatric disorders in a Spanish population of 18 year olds. *Acta Psychiatrica Scandinavica, 96*, 287–294.

Carney, C. E., Edinger, J. D., Krystal, A. D., Stepanski, E. J., & Kirby, A. (2005). *Are Primary Insomnia patients pathologically worried or primarily worried about sleep?* Paper presented at the Associated Professional Sleep Societies, Salt Lake City, UT.

Carney, C. E., Segal, Z. V., Edinger, J. D., & Krystal, A. D. (2007). A comparison of rates of residual insomnia symptoms following pharmacotherapy or cognitive behavioral therapy for major depression. *The Journal of Clinical Psychiatry, 68*(2), 254–260.

Coleman, R. M., Roffwarg, H. P., Kennedy, S. J., Guilleminault, C., Cinque, J., Cohn, M. A., et al. (1982). Sleep wake disorders based on polysomnographic diagnosis: A national cooperative study. *Journal of the American Medical Association, 247*, 997–1103.

Edinger, J. D., Bonnet, M., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., et al. (2004). Derivation of research diagnostic criteria for insomnia: Report on an American Academy of Sleep Medicine work group. *Sleep, 27*, 1567–1596.

Edinger, J. D., Wohlgemuth, W. K., Krystal, A. D., & Rice, J. R. (2005). Behavioral insomnia therapy for Fibromyalgia patients: A randomized clinical trial. *Archives of Internal Medicine, 165*, 2527–2535.

Foley, D. J., Monjan, A., & Brown, S. L. (1995). Sleep complaints among elderly persons: An epidemiologic study of three communities. *Sleep, 18*, 425–432.

Ford, D., & Kamerow, D. (1989). Epidemiologic study of sleep disturbances in psychiatric disorders. *Journal of the American Medical Association, 262*, 1479–1484.

Gislason, T., & Almqvist, M. (1987). Somatic diseases and sleep complaints. An epidemiological study of 3, 201 Swedish men. *Acta Medica Scandinavica, 221*, 475–481.

Gross, R., & Borkovec, T. (1982). Effects of cognitive intrusion manipulation on the sleep onset latency of good sleepers. *Behaviour Therapy, 13*, 112–116.

Hajak, G., & SINE Study Group Study of Insomnia in Europe. (2001). Epidemiology of severe insomnia and its consequences in Germany. *European Archives of Psychiatry & Clinical Neuroscience, 251*, 49–56.

Hall, M., Buysse, D. J., Reynolds, C. F., Kupfer, D. J., & Baum, A. (1996). Stress-related intrusive thoughts disrupt sleep onset and contiguity. *Sleep Research, 25*, 163.

Haponik, E., Frye, A., Richards, B., Wymer, A., & Hinds, A. (1996). Sleep history is neglected diagnostic information. *Journal of General Internal Medicine, 11*, 759–761.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research & Therapy, 40*, 869–893.

Jacobs, E. A., Reynolds, C. F., III, Kupfer, J. D., Lovin, P. I., & Ehrenpreis, A. B. (1988). The role of PSG in the differential diagnosis of chronic insomnia. *American Journal of Psychiatry, 145*, 346–349.

Johnson, E. O., Roehrs, T., Roth, T., & Breslau, N. (1998). Epidemiology of alcohol and medication as aids to sleep in early adulthood. *Sleep, 21*, 178–186.

Johnson, E. O., Roth, T., & Breslau, N. (2006). The association of insomnia with anxiety disorders and depression: Exploration of the direction of risk. *Journal of Psychiatric Research, 40*(8), 700–708.

Katz, D. A., & McHorney, C. A. (1998). Clinical correlates of insomnia in patients with chronic illness. *Archives of Internal Medicine, 158*, 1099–1107.

Krupinski, J., & Tiller, J. W. (2001). The identification and treatment of depression by general practitioners. *Australian and New Zealand Journal of Psychiatry, 35*, 827–832.

Leger, D. & Poursain, B. (2005). An international survey of insomnia: under-recognition and under-treatment of a polysymptomatic condition. *Current Medical Research and Opinion, 21*(11), 1785–1792.

Lichstein, K. L. (2000). Secondary insomnia. In K. L. Lichstein & C. M. Morin (Eds.), *Treatment of late-life insomnia* (pp. 297–319). Thousand Oaks, CA: Sage.

Lichstein, K., Durrence, H., Taylor, D., Bush, A., & Riedel, B. (2003). Quantitative criteria for insomnia. *Behavioral Research and Therapy, 41*, 427–445.

Lichstein, K. L., Wilson, N. M., & Johnson, C. T. (2000). Psychological treatment of secondary insomnia. *Psychology and Aging, 15*, 232–240.

Lineberger, M., Carney, C., Edinger, J., & Means, M. (2006). Defining insomnia: Quantitative criteria for insomnia severity and frequency. *Sleep, 29*, 479–485.

Livingston, G., Blizard, B., & Mann, A. (1993). Does sleep disturbance predict depression in elderly people?: A study in inner London. *British Journal of General Practice, 43*, 445–448.

Manber, R., Edinger, J. D., Gress, J. L., San Pedro-Salcedo, M. G., Kuo, T. F., & Kalista, T. (2008). Cognitive Behavioral Therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia. *Sleep, 31*(4), 489–495.

Mellinger, G. D., Balter, M. B., & Uhlenhuth, E. H. (1985). Insomnia and its treatment: Prevalence and correlates. *Archives of General Psychiatry, 42*, 225–232.

Nowell, P. D., Buysse, D. J., Reynolds, C. F., Hauri, P. J., Roth, T., Stepanski, E. J., et al. (1997). Clinical factors contributing to the differential diagnosis of primary insomnia and insomnia related to mental disorders. *American Journal of Psychiatry, 154*, 1412–1416.

Ohayon, M. M. (2002). Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Medicine Reviews, 6*, 97–111.

Ohayon, M., & Roth, T. (2003). Place of chronic insomnia in the course of depressive and anxiety disorders. *Journal of Psychiatric Research, 37*, 9–15.

Ohayon, M. M., Zulley, J., Guilleminault, C., Smirne, S., & Priest, R. G. (2001). How age and daytime activities are related to insomnia in the general population: Consequences for older people. *Journal of the American Geriatric Society, 49*, 360–366.

Pollack, M., Kinrys, G., Krystal, A., McCall, V. W., Roth, T., Schafer, K., et al. (2008). Eszopiclone coadministered with escitalopram in patients with insomnia and comorbid Generalized Anxiety Disorder. *Archives of General Psychiatry, 65*(5), 551–562.

Ramsawh, H. J., Stein, M. B., Belik, S. L., Jacobi, F., & Sareen, J. (2009). Relationship of anxiety disorders, sleep quality, and functional impairment in a community sample. *Journal of Psychiatric Research, 43*(10), 926–933.

Sanderson, W. C., Di Nardo, P. A., Rapee, R. M., & Barlow, D. H. (1990). Syndrome comorbidity in patients diagnosed with a DSM-III-R anxiety disorder. *Journal of Abnormal Psychology, 99*, 308–312.

Simon, G. E., & VonKorff, M. (1997). Prevalence, burden, and treatment of insomnia in primary care. *American Journal of Psychiatry, 154*, 1417–1423.

Spielman, A. J., Caruso, L. S., & Glovinsky, P. B. (1987). A behavioral perspective on insomnia treatment. *Psychiatric Clinics of North America, 10*, 541–553.

Weissman, M. M., Greenwald, S., Nino-Murcia, G., & Dement, W. C. (1997). The morbidity of insomnia uncomplicated by psychiatric disorders. *General Hospital Psychiatry, 19*, 245–250.

Zayfert, C., & DeViva, J. C. (2004). Residual insomnia following cognitive behavioral therapy for PTSD. *Journal of Traumatic Stress, 17*(1), 69–73.

Chapter 2
Considerations for Assessment

Abstract Before we consider complex issues such as the relation between sleep and anxiety, the sleep-related features of anxiety disorders, and the way sleep problems with co-existing anxiety are treated, it is imperative to consider diagnostic and assessment issues. Thus, this chapter provides an overview of assessment and measurement. While a comprehensive review of assessment techniques across all sleep and anxiety disorders is beyond the scope of this book, we focus on commonly used and supported measures and techniques for assessment, including physiologic, clinical, and structured interviews, and self-reported measures of sleep and anxiety.

This book focuses on the relation between sleep and anxiety, the sleep-related features of anxiety disorders, and the way the sleep problems are treated when there is coexisting anxiety. Before we consider these diagnostic features and treatment issues, it may be prudent to begin with an overview of assessment and measurement. A thorough review of assessment techniques across all sleep and anxiety disorders is beyond the scope of this book, but we provide information on commonly used measures and techniques for assessment.

Assessment of Sleep

When thinking about the assessment of sleep, many people conjure an image of a patient sleeping in a laboratory with various wires coming out of his or her head. Indeed, a polysomnogram (PSG) involves sleeping overnight in a laboratory, while electroencephalogram (EEG), electrocardiogram (EKG), pulse oximetry, air flow and electromyogram (EMG) equipment monitor brain, heart, respiration, and muscle activity, respectively. While it is often assumed that overnight PSG studies are used to asses insomnia, the measurement of insomnia most typically involves structured or unstructured interviews and self-report instrumentation. In fact, the standards in the field dictate that polysomnography (PSG) should not be used in routine assessment for insomnia (American Sleep Disorders Association, 1995).

Among the reasons for not using PSG is the fact that many people with insomnia will simply sleep very little – which is expensive verification for what they have already reported (i.e., they have difficulty sleeping). Alternatively, the opposite can occur as well. That is, some people experience what is called the "first-night effect," and they sleep considerably better than usual. The presumed mechanism of the first-night effect is conditioning. Those with psychophysiological insomnia frequently evidence conditioned arousal in their bed/bedroom, and the change in setting no longer elicits arousal during the sleep period. Thus, traditional PSG, clinically speaking, often tells us little about the person with insomnia. The exception to this is that some people with insomnia actually have another occult sleep disorder, such as sleep apnea, that would better account for their symptoms, and this could only be detected by the PSG.

In contrast to clinical practice, research using the PSG has revealed much about the sleep of those with insomnia. PSG data is used to visually score and classify 30 s periods called epochs into various stages of sleep or wakefulness, as well as to denote whether there was a significant event (e.g., a period in which the brain roused out of sleep). This is done according to accepted scoring criteria (Rechtshaffen & Kales, 1968). In insomnia, not all insomnia sufferers have disturbed sleep according to these scoring principles. In fact, there can be a discrepancy of minutes to hours between the sleep reported by people with insomnia and what is seen on the PSG. This is generally the exception to the rule and in some cases may be a subtype of insomnia called paradoxical insomnia. In paradoxical insomnia there is a large discrepancy between objective recordings of sleep and the subjective report of little to no sleep. In addition, the daytime impairment is far less than you would expect given their dramatic reports of sleep loss. Setting those with paradoxical insomnia aside, there can be some discrepancy between objective and subjective sleep. Some of this may have to do more with our measurement than with an insomnia sufferer necessarily making a perceptual error. The visual scoring method can obscure relative range and amplitudes of each band of electrical brain activity during sleep stages, as well as brief, frequent sleep stage transitions. Thus, it seems reasonable to assume that subjectively important sleep information is not captured by the traditional PSG scoring approach. In contrast, sleep EEG spectral analysis provides a microarchitectural picture of brain wave activity across sleep stages. Applications of such analyses to the study of insomnia have been promising and suggested that spectral measures effectively discriminate psychophysiological insomnia sufferers from normal sleepers and other insomnia subtypes (Freedman, 1986; Lamarche & Ogilvie, 1997; Nofzinger et al., 1999; Perlis, Smith, Andrew, Orff, & Giles, 2001). Perlis et al. (2001) have shown that the degree of relative power in the Beta (14–35 Hz) range negatively correlates with subjective–objective discrepancy measures for sleep time and sleep onset latency. This finding seems particularly intriguing since it implies that EEG spectral indices may relate to subjective insomnia sufferers' complaints. It should be noted that little has been done with spectral analysis and anxiety disorders, so much of what we will report will be nonspectral polysomnographic findings only. As such, mixed or absent findings using visually scored methods may not necessarily imply that the sleep disturbance is only subjective.

Actigraphic Measurement of Sleep

The most commonly known objective measure of sleep is the polysomnogram, but a less expensive and commonly used measure is actigraphy. An actigraph is a small, wrist-watch-like instrument that detects movement using an accelerometer. Scoring algorithms can then determine whether the activity is indicative of sleep or wakefulness. Measures of movements during sleep provide a relatively inexpensive, objective assessment of several sleep parameters, as well as objective corroboration of the subjective, self-report sleep evaluations obtained from sleep logs. Actigraphic monitoring is widely used as an objective estimate of sleep variables in insomnia research given that sleep parameters derived from actigraphs correlate well with PSG derived variables.

Subjective Assessment of Sleep

When someone with insomnia presents for treatment, she/he usually has ready answers for the clinically important questions such as:

"What time do you go to bed?"

"What time do you get up in the morning?"

"How long does it take you to fall asleep?"

"How long are you awake during the night?"

Whereas some people are able to provide reasonably accurate answers to these questions, a substantial proportion of insomnia sufferers provide answers that do not provide an accurate portrayal of their general sleep difficulty and usual sleep practices. This is usually not due to an intention to exaggerate the sleep difficulty or otherwise provide any misleading information. Moreover, it results from a natural tendency to remember the more difficult nights that led to seeking professional consultation. Moreover, sleep habits and sleep patterns are notoriously variable from one night to the next among insomnia sufferers, so the summary retrospective appraisals required by the questions shown are likely to overlook this variability, and thus conceal important treatment targets. For this reason, the evaluation of insomnia usually benefits by prospective assessment techniques fashioned to capture the sleep and associated behavioral variability that defines the insomnia disorder. The tool most commonly used for this purpose is the sleep diary. Rather than asking if particular sleep behaviors are a problem, sleep logs generally inquire about how long it took to fall asleep and the time spent awake in the middle of the night (see Fig. 2.1). We discuss face-valid retrospective measures of symptom severity later in the chapter.

In its usual format, the sleep diary is a paper and pencil instrument designed to allow the collection of information about sleep patterns prospectively over a period of several weeks. The typical sleep diary includes questions about

EXAMPLE

DAY OF THE WEEK	Monday						
CALENDAR DATE	3/24/08						
1. Yesterday I napped from ___ to ___ (note time of all naps).	2:30-3:15 PM						
2. Last night I took____ mg. of _____ or _____ of alcohol as a sleep aid	Ambien 5 mg.						
3. Last night I got in my bed at ____	11:00 PM						
4. Last night I turned off the lights and attempted to fall asleep at ____	11:30 PM						
5. After turning off the lights it took me about ____ minutes to fall sleep.	40 Min.						
6. I woke from sleep _____ times. (Do not count your final awakening)	2 Times						
7. My awakenings lasted _____ minutes. (List each awakening separately)	25 Min. 40 Min.						
8. Today I woke up at _____ NOTE this is your final awakening.	6:30 AM						
9. Today I got out of bed for the day at _____.	7:15 AM						

10. I would rate the quality of last night's sleep as:
  1 = very poor   4 = good
  2 = poor   5 = excellent
  3 = fair.
	3						

11. How well rested did you feel upon rising today?
  1 = not all rested   4 = rested
  2 = slightly rested   5 = well rested
  3 = somewhat rested
	2						

Fig. 2.1 Traditional sleep diary

nightly bed time and rising time, the perceived time it takes to fall asleep each night, the amount of time being awake during the night, and the time of the final morning awakening. Also, often included are questions about the quality of each night's sleep, daytime napping patterns, and usage of substances (caffeine, alcohol, sleep medications) that might influence sleep each night. Sleep diaries are useful for quantifying insomnia severity, aiding in diagnostic discriminations and case conceptualization, guiding the implementation of behavioral interventions, and measuring treatment outcomes (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006). Indeed, the sleep diary is such a mainstay of behavioral insomnia treatment that it is difficult to envision implementing treatment strategies such as those described later in this chapter without this invaluable tool.

A variety of paper and pencil sleep diaries are available, and these differ slightly in the type or amount of information obtained (Edinger & Carney, 2008; Espie, 2000; Monk et al., 1994; Morin, 1993; Sateia, 2002; Wohlgemuth & Edinger, 2000). Figure 2.1 shows one version commonly used in insomnia treatment studies and in clinical venues. Through the daily entries this type of diary elicits, daily bedtimes, rise times and napping patterns can be ascertained, and estimates of nightly sleep and wake time can be derived. Whereas this type of diary adapts well to the assessment of many types of insomnia problems, alternate versions may better suit the needs of certain populations or certain types of sleep difficulties. For example, The National Sleep Foundation has developed three sleep diary versions geared toward adults, teenagers, and children (NSF, 2007). The American Academy of Sleep Medicine [www.aasm.org] offers a diary that is structured in an analogue design (AASM, 2008) that instructs respondents to "color in" the blocks of time slept. This type of diary may provide a richer picture of sleep patterns for individuals who have difficulty accurately completing the traditional diary or among those who have erratic sleep patterns (e.g., shift workers). Nonetheless, the format shown in Fig. 2.1 is used in this chapter since it is well suited for initial assessment and tracking response to the types of treatment discussed later.

Thus, with sleep diaries, the clinician can determine whether particular sleep indices like sleep onset latency is a problem based on norms. For example, if someone complains that they cannot sleep on a fairly consistent basis, and their logs support this complaint, they are viewed as having subjectively disturbed sleep. How well do people estimate their sleep? This is a tricky proposition as it begs the questions, what should be the gold standard for comparison? If insomnia is a subjective disorder, should not subjective report be the gold standard? Generally speaking, people do fairly well in estimating their sleep when compared to an objective measure such as polysomnography. The most common *errors* when compared with PSG are the overestimation of time spent awake and the underestimation of the time spent sleeping (Coates & Thoresen, 1979; Means, Edinger, Glenn, & Fins, 2003). Nonetheless, sleep logs are considered quintessential to insomnia assessment (Buysse et al., 2006; Sateia, 2002).

Clinical Interview

Both structured and semi-structured clinical interviews are commonly used in clinical and research practice. To arrive at a diagnosis and to develop a case formulation for treatment, the interview focuses on the etiologic (i.e., cognitive and behavioral perpetuating) factors in the insomnia. Thus, the interview will elicit the details and history of the complaint as well as the history of any possible cooccurring medical or mental health issue. It is often helpful to attempt to develop a timeline of each condition to attempt to understand the degree to which the sleep and comorbid conditions are independent, interactive, or dependent. Interviews tend to cover these major areas: the nature and history of the sleep complaint, current stressors (including relationship discord, financial strain, or environmental factors such as a loud or unsafe sleeping environment), presence of any cardinal symptoms of another sleep disorder (e.g., loud snoring, a tendency to fall asleep involuntarily, leg twitching, restless leg symptoms), medical and psychiatric history (including medication use, surgeries, allergies, exposure to toxins, or any recent change in reproductive status), current sleep habits (including the presence of shift work or frequent time zone travel, use of sleep-interfering substances such as caffeine, cigarettes, alcohol), and treatment history. Information about or from their current bed partner also can be helpful. For example, the bed partner may exhibit loud snoring, which may be disruptive to the patient's sleep. The bed partner may also be helpful in unexpected ways. For example, a patient was complaining of rather spectacular sleep deprivation (e.g., she complained that she had not slept in the past 4 years) but she lacked any appearance of sleepiness and was quite functional during the day. When the husband was asked about his wife's sleep problem he said that the problem was her snoring. The wife had not reported that they were sleeping in separate rooms because of his complaints about her snoring. Her history of complaints led to an overnight study that revealed moderate sleep apnea. She also had insomnia and a focus in cognitive therapy was to examine the anxiety-producing consequence of her belief that she did not sleep at all (when in actuality she was sleeping, as evidenced by her snoring). She modified this belief to a more accurate and helpful realization that her sleep was lightened by the breathing disruptions, and she did indeed sleep.

In addition to an unstructured clinical interview, there are several site-specific sleep disorder interviews or published semi-structured interviews for insomnia (Savard & Morin, 2002; Spielman & Anderson, 1999) useful for guiding the practitioner through diagnostic criteria for sleep disorders including insomnia.

Self-report Measurement

Global Sleep Symptom Questionnaires

Arguably, the two most common self-report symptom questionnaires are the Insomnia Severity Index (ISI) (Morin, 1993) and the Pittsburgh Sleep Quality Index – PSQI (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The ISI is a 7-item

questionnaire of subjective insomnia symptom severity. Daytime and nighttime insomnia symptoms are rated using a 5-point (0–4) Likert scale. These symptoms include: difficulties falling asleep and/or staying asleep, waking too early in the morning; sleep dissatisfaction; degree of impairment with daytime functioning; degree to which impairments are noticeable; and distress or concern about insomnia. Morin and colleagues suggest the following ranges for interpretation of clinical significance: 0–7 (no clinical insomnia), 8–14 (sub-threshold insomnia), 15–21 (insomnia of moderate severity), and 22–28 (severe insomnia). There is good reliability and validity (using both sleep logs and electronic sleep recordings) (Bastien, Vallières, & Morin, 2001). It is a recommended assessment tool for insomnia research (Buysse et al., 2006), and its quick administration time makes it useful for clinical use too.

Another recommended measure for standard insomnia assessment (Buysse et al., 2006) is the Pittsburgh Sleep Quality Index – PSQI (Buysse et al., 1989). While the ISI is insomnia-specific, the PSQI is a more global measure of sleep disturbance across sleep disorders. It is a retrospective measure (over the past month) of sleep onset latency, sleep duration, sleep efficiency (i.e., the proportion of time in bed that is actually spent asleep), sleep quality, disturbances to sleep, medication use, and daytime dysfunction. Out of a possible total score that ranges from 0 to 21, a PSQI score of >5 appears to discriminate those with insomnia from good sleepers (Buysse et al., 1989). As such, a post-treatment PSQI score <5 has been used in some studies as indicating insomnia remission. While it is widely used and has good psychometrics, we have reported that elevated levels of anxiety may contribute to PSQI score elevations in those with comorbid disorders (Carney, Edinger, Krystal, Stepanski & Kirby, 2006). Thus, it may be prudent to interpret PSQI scores with caution in the presence of significant anxiety.

Cognitive Insomnia Questionnaires

The Dysfunctional Beliefs and Attitudes about Sleep Questionnaire – DBAS (Morin, 1993) is a cognitive measure to assess problematic levels of unhelpful beliefs about sleep. The most current version is 16 items (Morin) wherein respondents rate the degree to which they believe particular statements about sleep. Both the original 30-item version and DBAS-16 have acceptable levels of internal consistency (Cronbach's alpha values >0.80) (Morin, 1993; Morin, Vallières, & Ivers, 2007). The DBAS discriminates between good and poor sleepers and is responsive to changes in beliefs resulting from cognitive-behavioral therapy for insomnia (Carney & Edinger, 2006). Responses on specific DBAS items can also be used in therapy to orient patients to particular unhelpful beliefs and to modify the veracity of belief in them.

The Sleep Self-Efficacy Scale (SES) (Lacks, 1987) is a 9-item measure of one's level of confidence in carrying out particular sleep-related behaviors. Insomnia is often characterized by thoughts of helplessness (Morin, 1993), so it can be a worthwhile clinical enterprise to determine the level of self-efficacy/agency one has with

regards to sleep. The SES has been used in several insomnia trials and has been shown to improve (i.e., one becomes more confident in the ability to engage in effective sleep behaviors) with sleep-related improvements (Carney & Edinger, 2006) and to predict response to CBT for insomnia (Edinger et al., 2009). Another potentially useful measure is the Glasgow Sleep Effort Scale (Broomfield & Espie, 2005). This scale is a measure of sleep-related effort with promising initial psychometric support (Broomfield & Espie, 2005). While further studies are needed, the concept of sleep effort is a useful one, as it purportedly underlies maladaptive sleep beliefs (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006).

Behavioral Insomnia Questionnaires

The Sleep Hygiene Practice Scale (SHAPS) (Lacks, 1987) is a widely used measure for the presence of sleep-disruptive behaviors such as taking naps, or exercising strenuously within 2 h of bedtime. While it enjoys frequent usage, the SHAPS does not appear to have particularly strong internal consistency (Lacks, 1987) and studies establishing its validity are currently lacking. A lesser known but initially psychometrically promising tool may be the Sleep Hygiene Index (Mastin, Bryson, & Corwyn, 2006).

In addition to sleep hygiene behaviors, it may also be important to assess the presence of safety behaviors. Safety behaviors are those behaviors that are used to avoid an unwanted experience. In insomnia, an example of a safety behavior would be consuming alcohol when having difficulty sleeping. One helpful tool in this regard (i.e., to assess unhelpful safety-related sleep behaviors) is the Sleep-Related Behaviors Questionnaire (SBRQ) (Ree & Harvey, 2004). This measure was derived from Harvey's Cognitive Model (2002) that asserts the safety behaviors that perpetuate sleep problems – an observation that has been shown experimentally too (Harvey, 2002).

Daytime Insomnia Symptom Questionnaires

One final issue to consider in the assessment of sleep is the measurement of daytime impairment. The ISI is useful in that one of the items specifically queries daytime insomnia symptoms across the range of cognitive, mood, functioning domains. Additionally, one of the most frequently assessed daytime areas is fatigue. People with insomnia often complain of fatigue. The Fatigue Severity Scale (FSS) (Krupp, LaRocca, Muir-Nash, & Sternberg, 1989) is a measure of the severity of fatigue symptoms. Like the DBAS the total FSS score is a mean-item score of the responses on the 9 items; a score above 3 is indicative of significant fatigue. While there are many more comprehensive measures of fatigue available (e.g., the

Multidimensional Fatigue Inventory), the FSS is brief and has many studies that establish its strong psychometric properties in those with sleep problems (Krupp et al., 1989; Krupp, Jandorf, Coyle, & Mendelson, 1993; Lichstein, Means, Noeb, & Aguillard, 1997).

When dealing with sleep disorders, it is useful to distinguish fatigue from clinically significant sleepiness. This is because sleepiness is often associated with disorders other than insomnia such as sleep apnea, narcolepsy, or periodic limb movement disorder. Whereas people with insomnia feel very tired (e.g., fatigued); they usually do not have clinical levels of sleepiness. Sleepiness is characterized by the propensity to fall asleep unintentionally, quickly and frequently when given the opportunity. The widely used Epworth Sleepiness Scale (ESS) (Johns, 1991), is an 8-item self-report questionnaire designed to assess the propensity to fall asleep in situations such as while driving, watching TV, or sitting and talking to someone. Respondents rate how likely they would be to fall asleep in these situations using a 4-point rating scale (0 = "would never doze" to 3 = "high chance of dozing"). A score of 10 or greater is considered to indicate clinically significant daytime sleepiness. The ESS is a common tool in sleep assessment with good reliability (Johns, 1991) and validity (i.e., strong correlation with objective tests of daytime sleepiness (Johns, 1991). The most common objective test of sleepiness is the Multiple Sleep Latency Test (MSLT). The MSLT is conducted at a sleep laboratory and involves PSG data collection during five 20-min nap opportunities spaced 2 h apart throughout the day. The sleep onset latency is averaged over the course of the 5 naps to determine sleepiness. If someone falls asleep within 10 min or less, the person is regarded as objectively sleepy. In addition to the assessment of sleep and medical history, it is important to assess for psychiatric factors as well.

Assessment of Anxiety

Those who work with people with sleep disorders assess for a range of psychopathology in addition to the sleep disorder. The diagnosis of insomnia requires that another disorder cannot better account for the insomnia symptoms, thus it is important to understand what other conditions could be causing or affecting the insomnia. This information is also important in the treatment of insomnia as specific anxiety-related strategies may need to be added or emphasized in the CBT insomnia treatment package. There are a variety of tools available to assess for general psychopathology, including semi-structured interviews that assess the range of possible Axis I disorders (e.g., the Structured Clinical Interview for DSM Axis I Disorders) (Spitzer, Williams, Gibbons, & First, 1996) and Axis II disorders (e.g., the Structured Clinical Interview for DSM Axis II Disorders) (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). There are also self-report questionnaires to assess for specific symptoms such as the Beck Depression Inventory to assess for depression symptoms (Beck, Steer, & Brown, 1996). Given the breadth of the area, we focus solely on anxiety disorder-specific tools.

Structured Interviews for Anxiety

Clinician-Administered Interviews for Anxiety

The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV) (Brown, Black, & Uhde, 1994) is a commonly used semi-structured interview for DSM-IV anxiety disorders. Within each disorder, users can collect information on specific symptoms, the intensity of fear and avoidance, the age of onset, and possible causes of the disorder. There are versions that can be used to collect information on lifetime anxiety disorders – ADIS-IV-L (Di Nardo, Brown, & Barlow, 1994), children (ADIS-IV-C) and parental report of their child's anxiety issues (Silverman & Albano, 1996). The ADIS-IV is considered a reliable and valid measure for anxiety disorder assessment (Brown, Di Nardo, Lehman, & Campbell, 2001) with the possible exception of reliability estimates for GAD. Lowered reliability for GAD may reflect nosologic issues with the diagnosis itself (i.e., there is high symptom overlap with a number of other disorders) (Brown et al., 2001). Although the ADIS-IV is a very useful tool in assessing anxiety and related disorders it can take a few hours to administer.

Clinician-Administered Interviews for Specific Anxiety Disorders

OCD: The Yale-Brown Obsessive Compulsive Scale (Goodman et al., 1989) is a 10-item, clinician-administered interview. For a variety of reasons, including strong psychometric performance (Goodman et al., 1989), the Y-BOCS is one of the most widely used rating scales for OCD. The Y-BOCS is used primary to assess symptom severity across five domains: (1) time spent or occupied; (2) interference with functioning or relationships; (3) degree of distress; resistance; and control. The Y-BOCS has been used in those with delayed sleep phase syndrome (Turner et al., 2007), and was used in an investigation of sleep disturbances among those with OCD (Kluge, Schussler, Dresler, Yassouridis, & Steiger, 2007).

PTSD: The Clinician-Administered PTSD Scale (CAPS) (Blake et al., 1990) is a widely used structured interview for the assessment of PTSD. The CAPS assesses for the presence of DSM-IV-TR criteria for PTSD. Each symptom is rated on the basis of frequency and intensity. There is one insomnia item in the interview that was used to evaluate residual insomnia after the completion of CBT in those who had PTSD (Zayfert & DeViva, 2004). Zayfert and colleagues employed a >30 min sleep difficulty cutoff for insomnia and a >90 min cutoff for severe insomnia.

Panic Disorder: The Panic Disorder Severity Scale (PDSS) (Shear et al., 1992) is a brief, clinician-administered interview for the assessment of panic attack and associated avoidance frequency, severity, and distress. It is not a diagnostic measure, but provides a quick assessment of panic disorder symptoms consistent with DSM-IV criteria. There are good demonstrated psychometric properties in those with anxiety

disorders (Shear et al., 1992); although we are unaware of any use in those with insomnia.

Self-Report Measures for Anxiety Symptoms

Sleep centers may be most likely to use a general psychiatric symptom instrument like the Profile of Mood States (POMS) (McNair, Lorr, & Droppleman, 1971), Brief Symptom Inventory (BSI) (Derogatis & Melisaratos, 1983), or the Symptom Checklist – SCL-90 (Derogatis, 1992). Such instruments contain a variety of symptoms, including anxiety symptom scales. However, there are specific measures that can be used for anxiety as well as specialized scales to assess specific anxiety problems.

General Measures for Self-Reported Anxiety

The Beck Anxiety Inventory (BAI) (Beck, Epstein, Brown, & Steer, 1988) is a 21-item screening test designed to distinguish anxiety symptoms from depressive symptoms based on symptoms experienced during the past week. Although the BAI may be helpful in assessing anxiety symptoms, it has been criticized for its overlap with panic attack symptoms (Cox, Cohen, Direnfeld, & Swinson, 1996). Because many individuals with GAD do not experience the range or severity of autonomic symptoms associated with panic attacks, the BAI may be less appropriate as a measure of anxiety symptomatology in individuals with GAD. People with breathing related disorders tend to score in the moderate range on the BAI, which appears to reflect the increased prevalence of anxiety disorders in those with sleep disordered breathing (Sharafkhaneh, Giray, Richardson, Young, & Hirshkowitz, 2005) rather than psychometric shortcomings of the instrument (Sanford, Bush, Stone, Lichstein, & Aguillard, 2008). The State-Trait Anxiety Inventory (STAI) (Spielberger, Gorsuch, & Lushene, 1970) is a widely used measure to assess general levels of anxiety. The STAI has been used across many insomnia studies. However, we are not aware of any specific psychometric evaluations of the properties of the STAI in those with insomnia or other sleep disorders.

The Anxiety Sensitivity Index (ASI) (Peterson & Reiss, 1993) is a 16-item scale measuring fear of anxiety-related symptoms. Endorsement of each item is rated on a 5-point scale ranging from 0 (very little) to 4 (very much) indicating the strength of one's beliefs about the consequences of anxiety, such as fear of embarrassment, illness, and loss of control. Anxiety sensitivity has been identified as an important construct in the onset and exacerbation of anxiety disorders (Peterson & Reiss, 1993; Schmidt, Zvolensky, & Maner, 2006) and has recently been identified as a predictor of sleep-related impairment, but not actual sleep disturbance, in those with insomnia (Vincent & Walker, 2001).

Anxiety Disorder-Specific Self-Report Questionnaires

GAD: The Penn State Worry Questionnaire (PSWQ) (Meyer, Miller, Metzger, & Borkovec, 1990) is one of the most common tools used to assess pathological worry. It is a 16-item inventory intended to measure the generality, excessiveness, and uncontrollability of pathological worry. The PSWQ focuses on the more cognitive concept of worry, as opposed to the BAI, and does not explicitly address sleep symptoms. Total scores range from 0 to 90. The PSWQ has good internal consistency and test–retest reliability over 8–10 weeks (Meyer et al., 1990). Behar, Alcaine, Zuellig, and Borkovec (2003) report that ROC cut-off of 45 gives sensitivity of 99% and specificity of 98% in separating GAD from non anxious controls (the diagnosis was made by self-report). The same authors looked at a sample of undergrads and derived a cut-off of 62 (86% sensitivity and 75% specificity). Fresco, Mennin, Heimberg, and Turk (2003) looked at social phobia vs. GAD and found a cut-off of 65 to be optimal. The PSWQ has been used in sleep populations, including primary insomnia (Buysse et al., 2008; Harvey & Greenall, 2003); however, no psychometric properties of the PSWQ among individuals with sleep disorders were found.

OCD: The Obsessive Compulsive Inventory-Revised (OCI-R) (Foa et al., 2002) is an 18-item self-report questionnaire that assesses the degree of distress related to OCD symptoms in the past month. The OCI-R discriminates OCD from other anxiety disorders (Abramowitz & Deacon, 2006) and has sound psychometric properties (Foa et al., 2002), but its properties in sleep disordered groups are unknown.

PTSD: The Impact of Events Scale (IES) (Horowitz, Wilner, & Alvarez, 1979) is a widely used 22-item questionnaire to assess responses to traumatic events. A Likert rating scale is used to assess the degree of distress produced by each symptom. The PTSD Diagnostic Scale – PDS (Foa, Cashman, Jaycox, & Perry, 1997) is used to inquire about the presence and severity of the DSM-IV PTSD symptoms. The measure asks about the frequency of each of the 49 items in the past month, which are rated on a 4-point scale. The PSQI Addendum for PTSD – PSQI-A (Germain, Hall, Krakow, Shear, & Buysse, 2005) is a 7-item scale measuring the frequency of disruptive nocturnal behaviors such as: hot flashes, general nervousness, memories or nightmares of traumatic experiences, severe anxiety or panic not related to traumatic memories, bad dreams not related to traumatic memories, episodes of terror of screaming, and episodes or acting out dreams. There are also frequency and timing ratings of nocturnal anxiety and anger and timing of these events. Preliminary studies have suggested that this addendum has good internal consistency ($\alpha = 0.85$), good convergent validity with measures of PTSD and the PSQI, and good sensitivity and specificity for distinguishing those with PTSD from those without.

Social Phobia: The Social Phobia Inventory (SPIN) (Connor et al., 2000) is a 17-item self-report measure that assesses multiple facets of social anxiety including the following: (1) avoidance of feared social situations, (2) feelings of embarrassment, (3) physiological changes (e.g., blushing), and (4) fear of being the center of attention. The SPIN has good reported psychometric properties and may be useful

as a brief screen for Social Phobia (Connor et al., 2000). The Social Phobia Scale (SPS) (Mattick & Clarke, 1998) is a 20-item questionnaire to assess the fear of scrutiny/evaluation in performances situations. Internal consistency reliability for the SPS has been shown to be high, with alpha values of 0.94. The Social Interaction Anxiety Scale (SIAS) (Mattick & Clarke, 1998) is a 19-item questionnaire to assess fears of specific social interaction situations (e.g., dating and attending parties). As with the SPS, the internal consistency reliability for the SIAS is excellent ($\alpha = 0.94$). Internal consistency was also high in a study of social anxiety, depression, and insomnia (Buckner, Bernert, Cromer, Joiner, & Schmidt, 2008). This study showed that 18.2% of socially anxious participants had elevations on the ISI suggestive of clinically significant insomnia.

The Social Phobia and Anxiety Inventory (SPAI) (Turner, Beidel, Dancu, & Stanley, 1989) is a 45-item self-report questionnaire of the frequency of social phobia or agoraphobia experiences across a range of social contexts. There is good internal consistency reliability (Turner et al., 1989) and validity (Peters, 2000). A literature search using the PSYCInfo database did not yield any results for the searches "Social Phobia and Anxiety Inventory AND sleep disorder," "SPAI AND sleep disorder," "Social Phobia and Anxiety Inventory AND insomnia," or "SPAI AND insomnia."

Panic Disorder and Agoraphobia: The Agoraphobic Cognitions Questionnaire (ACG) (Chambless, Caputo, Bright, & Gallagher, 1984) is a 15-item measure of "fear of fear" in those with panic disorder or agoraphobia. The frequency of specific catastrophic thoughts about the consequences of experiencing anxiety is rated on a 5-point scale (1 = the thought never occurs, and 5 = the thought always occurs). The ACQ has demonstrated adequate psychometric properties for the full scale, as well as two subscales reflecting the loss of control and the consequences of physical symptoms, and is able to discriminate anxiety disordered from non-clinical samples (Chambless & Gracely, 1989). Although some analyses suggest that the two factor structure lacks validity, the ACQ remains one of the most widely used instruments in research and clinical practice for patients with agoraphobia. To date, no research has reviewed the ACQ for use with sleep disordered population; however, evidence indicates that the catastrophic cognitions measured by the ACQ are specific to the experience of diurnal panic attacks and relatively unrelated to nocturnal panic attacks (O'Mahony & Ward, 2003). The ACG is most typically administered along with the Body Sensations Questionnaire (BSQ) (Chambless et al., 1984); a measure of the intensity of fear of the physical sensations of anxious arousal. Each of the 17 physical symptom/panic sensation items are rated on a scale of 1–5 that corresponds to the degree to which the sensation is frightening. Although the BSQ has not been examined in the context of insomnia, there appears to be no relationship between the fear of interoceptive cues as measured on the BSQ and sleep disturbances related to nocturnal panic attacks relative to those whose panic attacks occur exclusively during daytime (Craske, Lang, Tsao, Mystkowski, & Rowe, 2001).

The Panic Attack Symptoms Questionnaire (PASQ) and Panic Attack Cognitions Questionnaire (PACQ) (Clum, Broyles, Borden, & Watkins, 1990) can be used to

assess the severity of panic attacks and the degree to which patients are preoccupied with catastrophic cognitions respectively, during a panic episode. Preliminary data on the PACQ and PASQ suggest good internal consistency and utility in discriminating those with panic disorder from those with other anxiety disorders not associated with panic attacks (Clum et al., 1990). There are no known studies using these scales in those with insomnia or nocturnal panic attacks.

The Mobility Inventory for Agoraphobia – MI (Chambless, Caputo, Jasin, Gracely, & Williams, 1985) is a 27-item inventory of agoraphobic avoidance and panic attack frequency. For each of the listed situations commonly avoided by people with agoraphobia, the degree of avoidance when alone versus when accompanied by another person are rated on 5-point scales (1 = never avoid; 5 = always avoid). There are demonstrated sound psychometric properties and utility in discriminating between clinical and nonclinical samples (Chambless et al., 1985). We were not able to find psychometric evaluations of the MI in sleep-disordered population.

Regardless of the measures employed, it is important to generate a formulation of the problem and a plan of action for treatment. Below are two abbreviated examples of assessment in those with insomnia and their related case formulations.

Case Example 1: Generalized Anxiety Disorder and Insomnia

Ms. H is a 36-year-old female with a complaint of sleep onset and maintenance insomnia. She is unsure what caused the insomnia, but believes that her problem is currently maintained by her anxiety about sleep. Based on a questionnaire of insomnia symptom severity, her insomnia is in the moderately severe range (Insomnia Severity Index = 21). She reports that it is taking several hours for her to fall asleep, as well as 3–12 awakenings per night. A review of her sleep logs revealed that she tends to go to bed around 11 p.m. and rise around 7:30 a.m. (mean total time in bed = 8.7 h). Her average sleep onset latency is 172 min; her average time being awake after sleep onset is 66 min. Her estimated mean sleep efficiency for 2 weeks of sleep diaries is very poor (54%). She denied napping. She denied daytime sleepiness (Epworth Sleepiness Score = 4) but reported significant daytime fatigue (Fatigue Severity Score = 5.6).

On a scale assessing maladaptive sleep behaviors, she reported reading in bed each night and remaining in bed when she cannot sleep. She denied any other poor sleep habits, and denied regular use of caffeine, alcohol, or tobacco products. She stated that before bed she can "barely keep her eyes open," but when she gets into bed she feels "instantaneously awake and irritated." She begins to have thoughts such as, "I can't sleep." She acknowledged loud snoring, but denied observed apneas, or symptoms of cataplexy, hypnogogic hallucinations, restless legs, or periodic leg movements during sleep. A previous overnight sleep study conducted 4 months ago was unremarkable. She reported past diagnoses of Post-Traumatic Stress Disorder and Generalized Anxiety Disorder. She was in psychotherapy for about 1 year for PTSD, and denies it is a problem any longer. She regarded the

psychotherapy as very helpful. Based on her report it appeared to be an exposure-based psychotherapy. She denied symptoms of re-experiencing the trauma via flashbacks, intrusive thought or nightmares, and no longer avoids her family home (the site of the trauma). She denied any numbing of responsiveness. She currently takes Celexa (40 mg per day) for anxiety. She reported that her anxiety is much better currently; however, her responses on a measure of clinically significant worry would suggest that the current level of worry is in the clinical range (Penn State Worry Questionnaire = 64). She acknowledged that she is currently worrying quite a lot about her sleep problem, and when pressed, she acknowledged worries in other domains, including being late for appointments, work, family, finances, and global affairs. She also reported having difficulties with depression in the past, but denied current symptoms. Her responses on a depression symptom measure would corroborate her report of no depression (Beck Depression Inventory = 10).

Formulation: Ms. H appears to have developed a psychophysiologic insomnia. She also meets the criteria for GAD. She has good insight into her sleep problem, but is less willing to acknowledge a more pervasive worry problem. Ms. H may benefit from augmenting her pharmacologic treatment of anxiety with psychotherapy to address worry. It is unknown as to whether her medication is contributing to her sleep problem, but the sleep problem predated the medication and did not appear to worsen when she began taking the medication. Sleep focused treatment should target the belief that she cannot cope with her sleep problem and the conditioned hyperarousal (i.e., the abrupt switch into alertness when she gets into her bed). Going to bed only when she is sleepy and getting out of bed during prolonged awakenings (stimulus control) should reduce the conditioned arousal she is currently experiencing. Ms. H would also likely benefit from a relaxation practice and worry control training.

Case Example 2: Sleep-Specific Worry

Ms. T is a 28-year-old woman attending graduate school with a complaint of sleep maintenance insomnia. For the past 2 months, Ms. T has been waking after about 4–5 h of sleep and is unable to return to sleep. Occasionally, during these awakenings, she reports that her heart is beating fast and she feels anxious. Her family physician apparently told Ms. T that she was depressed and prescribed Prozac. She stated that she did not "feel" depressed (i.e., she did not have sad mood or depressive thoughts), but the Prozac was helpful in eliminating her "blah mood" and her social avoidance. She indicated that the Prozac was not helpful in reducing her awakenings or daytime fatigue.

Her score on a measure of insomnia symptom severity (Insomnia Severity Index = 17) would suggest that the insomnia is of moderate severity. A review of her sleep logs revealed several nights of excessive time in bed (up to 9 h). She appeared to obtain 6–8 h of sleep. On a measure of sleep-interfering behaviors, she denied using alcohol, caffeine, or any form of tobacco products. She reported that she read

in bed 7 nights per week for 10 min – she denied any other sleep disruptive behavior. Her responses to a questionnaire would not suggest significant daytime sleepiness (Epworth Sleepiness Scale = 6), but her score on the Fatigue Severity Scale would corroborate her report of significant fatigue (Fatigue Severity Scale = 6.1). During the clinical interview, she denied loud snoring, restless legs, observed apneas, periodic leg movements during sleep, cataplexy, sleep paralysis, hypnogogic hallucinations, nightmares, or any form of parasomnia.

Her report of daytime worry about her sleep and the possible consequences it has on her health and her performance at school was corroborated by a high score on a scale assessing unhelpful beliefs about sleep, including sleep worries (Dysfunctional Beliefs and Attitudes about Sleep Scale = 4.9). Her score on a measure of general worry (Penn State Worry Questionnaire = 39) was suggestive of a tendency toward worry and anxiety, although this score was well below the clinical cutoff for pathological worry or GAD. Her responses on a questionnaire that assesses depression symptoms was below the suggested cutoff for moderate, clinically significant depression (Beck Depression Inventory score = 11). During the clinical interview, she denied depressed mood or anhedonia, but acknowledged fatigue, difficulty concentrating, and insomnia.

Formulation: Although a mood episode may have precipitated the insomnia complaint, the mood episode appears to have resolved, and the insomnia remains. It is clear that she has considerable worry about her ability to sleep and the possible consequences that the insomnia will have on her health. She endorsed some unrealistic expectations and beliefs about sleep. It appears that the anxiety generated by her unrealistic beliefs, as well as some excessive time in bed in the morning may be currently maintaining her insomnia. She has been taking the Prozac for only 4 weeks, thus it remains a possibility that her awakenings will resolve after some more time on the antidepressant. In the meantime, I have instructed her to: (1) limit her time in bed to 6.5 h; (2) get out of bed each morning by 7 a.m.; (3) eliminate "resting" periods in the morning and throughout the day, so that she will avoid the possibility of an unintended nap; (4) focus on ways to cope with fatigue symptoms (e.g., engage in activating activities, take breaks during mundane tasks and fresh air); (5) complete Thought Records so that we can challenge her catastrophic thinking about sleep loss; and (6) if she awakens in the morning and cannot return to sleep within 20 min, she is to leave the bedroom and start her day.

References

AASM (2008) http://www.sleepeducation.com/pdf/sleepdiary.pdf

Abramowitz, J. S., & Deacon, B. J. (2006). Psychometric properties and construct validity of the obsessive-compulsive inventory–revised: Replication and extension with a clinical sample. *Journal of Anxiety Disorders, 20*(8), 1016–1035.

American Sleep Disorders Association. (1995). Practice parameters for the use of polysomnography in the evaluation of insomnia. *Sleep, 18*(1), 55–57.

Bastien, C. H., Vallières, A., & Morin, C. M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine, 2*(4), 297–307.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56*, 893–897.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck depression inventory, 2nd edition (BDI-II)*. San Antonio, TX: The Psychological Association.

Behar, E., Alcaine, O., Zuellig, A. R., & Borkovec, T. D. (2003). Screening for generalized anxiety disorder using the Penn State Worry Questionnaire: A receiver operating characteristic analysis. *Journal of Behavior Therapy and Experimental Psychiatry, 34*(1), 25–43.

Blake, D. D., Weathers, F., Nagy, L. M., Kaloupek, D. G., Klauminzer, G., Charney, D. S., et al. (1990). A clinician rating scale for assessing current and lifetime PTSD: The CAPS-1. *The Behavior Therapist, 13*, 187–188.

Broomfield, N. M., & Espie, C. A. (2005). Towards a valid, reliable measure of sleep effort. *Journal of Sleep Research, 14*(4), 401–407.

Brown, T., Black, B., & Uhde, T. (1994). The sleep architecture of social phobia. *Biological Psychiatry, 35*(6), 420–421.

Brown, T. A., Di Nardo, P. A., Lehman, C. L., & Campbell, L. A. (2001). Reliability of DSM-IV anxiety and mood disorders: Implications for classification of emotional disorders. *Journal of Abnormal Psychology, 110*, 49–58.

Buckner, J. D., Bernert, R. A., Cromer, K. R., Joiner, T. E. J., & Schmidt, N. B. (2008). Social anxiety and insomnia: The mediating role of depressive symptoms. *Depression and Anxiety, 25*, 124–130.

Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Lichstein, K. L., & Morin, C. M. (2006). Recommendations for a standard research assessment of insomnia. *Sleep, 29*(9), 1155–1173.

Buysse, D. J., Germain, A., Hall, M. L., Moul, D. E., Nofzinger, E. A., Begley, A., et al. (2008). EEG spectral analysis in primary insomnia: NREM period effects and sex differences. *Sleep, 31*(12), 1673–1682.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213.

Carney, C. E., Edinger, J. D., Krystal, A. D., Stepanski, E. J., &Kirby, A. (2006). The contribution of general anxiety to sleep quality ratings in insomnia subtypes. *Sleep, 29*(Suppl.), A233.

Chambless, D. L., Caputo, G. C., Bright, P., & Gallagher, P. (1984). Assessment of "fear of fear" in agoraphobics: The body sensations questionnaire and the agoraphobic cognitions questionnaire. *Journal of Consulting and Clinical Psychology, 52*, 1090–1097.

Chambless, D. L., Caputo, G. C., Jasin, S. E., Gracely, E. J., & Williams, C. (1985). The mobility inventory for agoraphobia. *Behaviour Research and Therapy, 23*, 35–44.

Chambless, D. L., & Gracely, E. J. (1989). Fear of fear and the anxiety disorders. *Cognitive Therapy and Research, 13*, 9–20.

Clum, G. A., Broyles, S., Borden, J., & Watkins, P. L. (1990). Validity and reliability of the panic attack symptoms and cognitions questionnaires. *Journal of Psychopathology and Behavioral Assessment, 12*, 233–245.

Coates, T. J., & Thoresen, C. E. (1979). Treating arousals during sleep using behavioural self-management. *Journal of Consulting and Clinical Psychology, 47*, 603–605.

Connor, K. M., Davidson, J. R., Churchill, L. E., Sherwood, A., Foa, E. B., & Weisler, R. H. (2000). Psychometric properties of the social phobia inventory (SPIN): A new self-rating scale. *British Journal of Psychiatry, 176*, 379–386.

Cox, B. J., Cohen, E., Direnfeld, D. M., & Swinson, R. P. (1996). Does the beck anxiety inventory measure anything beyond panic attack symptoms? *Behaviour Research and Therapy, 34*, 949–954.

Craske, M. G., Lang, A. J., Tsao, J. C. I., Mystkowski, J. L., & Rowe, M. K. (2001). Reactivity to interoceptive cues in nocturnal panic, *Journal of Behavior Therapy and Experimental Psychiatry*, *32*, 173–190

Derogatis, L. (1992). *Symptom checklist-90-revised: Administration, scoring, and procedures manual II*. Towson, MD: Clinical Psychometrics Research.

Derogatis, L. R., & Melisaratos, N. (1983). The brief symptom inventory: An introductory report. *Psychological Medicine, 13*, 595–605.

Di Nardo, P. A., Brown, T. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV (lifetime version)*. San Antonio, TX: Psychological Corporation.

Edinger, J. D., & Carney, C. E. (2008). *Overcoming insomnia: A cognitive-behavioral therapy approach-therapist guide*. New York: Oxford University Press, Inc.

Edinger, J. D., Olsen, M. K., Stechuchak, K. M., Means, M. K., Lineberger, M. K., Kirby, A., et al. (2009). Cognitive behavioral therapy with primary and comorbid insomnia: A randomized clinical trial. *Sleep, 32*(4), 499–510.

Espie, C. A. (2000). Assessment and differential diagnosis. In K. L. Lichstein & C. M. Morin (Eds.), *Treatment of late-life insomnia* (pp. 81–108). Thousand Oaks, CA: Sage Publications, Inc.

Espie, C., Broomfield, N., MacMahon, K., Macphee, L., & Taylor, L. M. (2006). The attention-intention-effort pathway in the development of psychophysiologic insomnia: A theoretical review. *Sleep Medicine Reviews, 10*(4), 215–245.

First, M., Gibbon, M., Spitzer, R., Williams, J. B. W., & Benjamin, L. S. (1997). *Users Guide for the structured clinical interview for DSM IV personality disorders (SCID-II)*. Washington, DC: American Psychiatric Press.

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The posttraumatic diagnostic scale. *Psychological Assessment, 9*, 445–451.

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., & Hajcak, G. (2002). The obsessive-compulsive inventory: Development and validation of a short version. *Psychological Assessment, 14*(4), 485–496.

Freedman, R. R. (1986). EEG power spectra in sleep-onset insomnia. *Electroencephalography and Clinical Neurophysiology, 63*(5), 408–413.

Fresco, D. M., Mennin, D. S., Heimberg, R. G., & Turk, C. L. (2003). Using the Penn State Worry Questionnaire to identify individuals with generalized anxiety disorder: A receiver operating characteristic analysis. *Journal of Behavior Therapy and Experimental Psychiatry, 34*(3–4), 283–291.

Germain, A., Hall, M., Krakow, B., Shear, M. K., & Buysse, D. J. (2005). A brief sleep scale for posttraumatic stress disorder: Pittsburgh sleep quality index addendum for PTSD. *Journal of Anxiety Disorders, 19*, 233–244.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., et al. (1989). The Yale-Brown obsessive compulsive scale. I. Development, use, and reliability. *Archives of General Psychiatry, 46*(11), 1006–1011.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40*, 869–893.

Harvey, A. G., & Greenall, E. (2003). Catastrophic worry in primary insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 34*(1), 11–23.

Horowitz, M., Wilner, N., & Alvarez, W. (1979). Impact of events scale: A measure of subjective stress. *Psychosomatic Medicine, 41*, 209–218.

Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. *Sleep, 14*, 540–545.

Kluge, M., Schussler, P., Dresler, M., Yassouridis, A., & Steiger, A. (2007). Sleep onset REM periods in obsessive compulsive disorder. *Psychiatry Research, 152*(1), 29–35.

Krupp, L. B., Jandorf, L., Coyle, P. K., & Mendelson, W. (1993). Sleep disturbance in chronic fatigue syndrome. *Journal of Psychosomatic Research, 37*, 325–331.

Krupp, L. B., LaRocca, N. G., Muir-Nash, J., & Sternberg, A. D. (1989). The Fatigue Severity Scale: Application to patients with multiple sclerosis and systemic lupus erythemasis. *Archives of Neurology, 46*, 1121–1123.

Lacks, P. (1987). *Behavioral treatment for persistent insomnia*. New York: Pergamon Press.

Lamarche, C. H., & Ogilvie, R. D. (1997). Electrophysiological changes during the sleep onset period of psychophysiological insomniacs, psychiatric insomniacs and normal sleepers. *Sleep, 20*, 724–733.

Lichstein, K. L., Means, M. K., Noeb, S. L., & Aguillard, R. N. (1997). Fatigue and sleep disorders. *Behaviour Research and Therapy, 35*(8), 733–740.

Mastin, D. F., Bryson, J., & Corwyn, R. (2006). Assessment of sleep hygiene using the sleep hygiene index. *Journal of Behavioral Medicine, 29*(3), 223–227.

Mattick, R. P., & Clarke, J. C. (1998). Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behaviour Research and Therapy, 36*, 455–470.

McNair, D. M., Lorr, M., & Droppleman, L. F. (1971). *Manual for the profile of mood states*. San Diego, CA: EDITS.

Means, M. K., Edinger, J. D., Glenn, D. M., & Fins, A. I. (2003). Accuracy of sleep perceptions among insomnia sufferers and normal sleepers. *Sleep Medicine, 4*, 285–296.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28*(6), 487–495.

Monk, T. H., Reynolds, C. F. R., Kupfer, D. J., Buysse, D. J., Coble, P. A., Hayes, A. J., et al. (1994). The Pittsburgh sleep diary. *Journal of Sleep Research, 3*, 111–120.

Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York: Guilford Press.

Morin, C. M., Vallières, A., & Ivers, H. (2007). Dysfunctional beliefs and attitudes about sleep (DBAS): Validation of a brief version (DBAS-16). *Sleep, 30*(11), 1547–1554.

Nofzinger, E. A., Nowell, P. D., Buysse, D. J., Vasco, M. E., Thase, F. E., Kupfer, D. J., et al. (1999). Towards a neurobiology of sleep disturbance in primary insomnia and depression: A comparison of subjective, visually scored, period amplitude and power spectral density sleep measures. *Sleep, 22*(Suppl 1), S99.

NSF (2007) http://www.sleepfoundation.org

O'Mahony, J. F., & Ward, B. G. (2003). Differences between those who panic by day and those who also panic by night. *Journal of Behavior Therapy and Experimental Psychiatry, 34*, 239–249.

Perlis, M. L., Smith, M. T., Andrew, P. J., Orff, H., & Giles, D. E. (2001). Beta/gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep, 24*, 110–117.

Peters, L. (2000). Discriminant validity of the Social Phobia and Anxiety Inventory (SPAI), the Social Phobia Scale (SPS) and the Social Interaction Anxiety Scale (SIAS). *Behaviour Research and Therapy, 38*(9), 943–950.

Peterson, R. A., & Reiss, S. (1993). *Anxiety sensitivity index revised test manual*. Worthington, OH: IDS Publishing Corporation.

Rechtshaffen, A., & Kales, A. (1968). *A manual of standardized terminology, techniques, and scoring systems of sleep stages of human subjects*. Los Angeles: UCLA Brain Information Service/Brain Research Institute.

Ree, M. J., & Harvey, A. G. (2004). Insomnia: The development of the sleep-related behaviours questionnaire (SRBQ). *Behaviour Change, 21*(1), 26–36.

Sanford, S. D., Bush, A. J., Stone, K. C., Lichstein, K. L., & Aguillard, N. (2008). Psychometric evaluation of the beck anxiety inventory: A sample with sleep-disordered breathing. *Behavioral Sleep Medicine, 6*(3), 193–205.

Sateia, M. J. (2002). Epidemiology, consequences, and evaluation of insomnia. In T. L. Lee-Chiong, M. J. Sateia, & M. A. Carskadon (Eds.), *Sleep medicine* (pp. 151–160). Philadelphia: Hanley and Belfus.

Savard, J., & Morin, C. M. (2002). Insomnia. In M. Antony & D. B. Barlow (Eds.), *Handbook of assessment and treatment planning for psychological disorders* (pp. 523–555). New York: Guilford.

Schmidt, N. B., Zvolensky, M. J., & Maner, J. K. (2006). Anxiety sensitivity: Prospective prediction of panic attacks and Axis I pathology. *Journal of Psychiatric Research, 40*, 691–699.

Sharafkhaneh, A., Giray, N., Richardson, P., Young, T., & Hirshkowitz, M. (2005). Association of psychiatric disorders and sleep apnea in a large cohort. *Sleep, 28*, 1405–1411.

Shear, M. K., Brown, T. A., Sholomskas, D. E., Barlow, D. H., Gorman, J. M., Woods, S. W., et al. (1992). *Panic disorder severity scale (PDSS)*. Pittsburgh, PA: University of Pittsburgh School of Medicine.

Silverman, W. K., & Albano, A. M. (1996). *Manual for the anxiety disorders schedule for DSM-IV: Child version*. Albany, NY: Graywind Publications.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the state-trait anxiety inventory*. Palo Alto, CA: Consulting Psychologists Press.

Spielman, A., & Anderson, M. (1999). The clinical interview and treatment planning as a guide to understanding the nature of insomnia: The CCNY insomnia interview. In S. Chokroverty (Ed.), *Sleep disorders medicine: Basic science, technical considerations, and clinical aspects* (2nd ed., pp. 385–416). Boston: Butterworth-Heinemann.

Spitzer, R. L., Williams, J. B. W., Gibbons, M., & First, M. B. (1996). *Instruction manual for the structured clinical interview for DSM-IV (SCID-IV). (SCID 1996 Revision)*. New York: Biometrics Research Department, New York Psychiatric Institute.

Turner, J., Drummond, L. M., Mukhopadhyay, S., Ghodse, H., White, S., Pillay, A., et al. (2007). A prospective study of delayed sleep phase syndrome in patients with severe resistant obsessive-compulsive disorder. *World Psychiatry, 6*(2), 108–111.

Turner, S. M., Beidel, D. C., Dancu, C. V., & Stanley, M. A. (1989). An empirically derived inventory to measure social fears and anxiety: The social phobia and anxiety inventory. *Psychological Assessment, 1*, 35–40.

Vincent, N., & Walker, J. (2001). Anxiety sensitivity: Predictor of sleep-related impairment and medication use in chronic insomnia. *Depression and Anxiety, 14*, 238–243.

Wohlgemuth, W. K., & Edinger, J. D. (2000). Sleep restriction therapy. In K. L. Lichstein & C. M. Morin (Eds.), *Treatment of late-life insomnia* (pp. 147–184). Thousand Oaks, CA: Sage.

Zayfert, C., & DeViva, J. C. (2004). Residual insomnia following cognitive behavioral therapy for PTSD. *Journal of Traumatic Stress, 17*(1), 69–73.

Chapter 3
Anxiety Disorders and Accompanying Insomnia

Abstract In this chapter, we discuss the different manifestations (both subjective and objective) of sleep disturbance within anxiety disorders. We discuss the associated features of the anxiety disorder and focus especially on those anxiety conditions presumed to have higher comorbidity with insomnia. We also consider whether there are any disorder-specific special insomnia treatment considerations within each diagnostic category. We argue that there appears to be no real impediment to treating the insomnia of those with anxiety disorders, although there may be some special treatment considerations for panic disorder (i.e., allowing for a less conservative time-in-bed restriction) and PTSD (i.e., addressing nightmares). We review the evidence that treating the comorbid anxiety disorder only (and thus ignoring the insomnia) may limit the degree of anxiety disorder treatment response and/or result in residual insomnia, and the evidence for a combined approach (i.e., treating the insomnia and anxiety problem concurrently).

Now that we have discussed the ways in which insomnia and anxiety disorders are assessed, we can discuss the different manifestations (both subjective and objective) of sleep disturbance within anxiety disorders. We discuss the associated features of the anxiety disorder and focus especially on those anxiety conditions presumed to have higher comorbidity with insomnia. We also consider whether there are any disorder-specific special insomnia treatment considerations within each diagnostic category.

Panic Disorder

Panic Disorder (PD) is a disorder characterized by discrete episodes of intense fear or terror (American Psychiatric Association, 1997). Symptoms in a panic attack include autonomic symptoms of dizziness, choking, palpitations, trembling, chest discomfort, parathesias, chills, hot flashes, stomach upset, and sweating. Panic attacks often include cognitive–emotional symptoms such as fear of dying, losing control, or going "crazy," as well as derealization (e.g., a sense that things are

unreal) or depersonalization (e.g., a sense of detachment from oneself). The first set of DSM-IV-TR diagnostic criteria for Panic Disorder requires the occurrence of (1) recurrent unexpected panic attacks and one of the following: (a) persistent fear of having more attacks, (b) persistent fear of the implications of the attacks (e.g., "I may go crazy" or "I may die"), and/or (c) a significant change in behavior related to the attacks (e.g., avoidance of situations wherein panic attacks may occur) (American Psychiatric Association, 1997). Lastly, the panic attacks should not be better accounted for by the physiological effects of a medical condition or substance, or another mental disorder (e.g., such as the panic attacks that can occur in Social Phobia). Panic Disorder can occur with or without agoraphobia.

In addition to insomnia, there are many comorbidities associated with PD including depression, agoraphobia, and alcohol, sedative or hypnotic abuse (American Psychiatric Association, 1997). The overlap with other disorders has clouded polysomnographic investigations of sleep in those with PD, as some of the reported sleep problems may relate to comorbid depression rather than PD (Stein, Enns, & Kryger, 1993). Generally, PSG has verified the subjective report of poor sleep in PD sufferers. For example, PSG has revealed a decreased sleep efficiency (i.e., the percentage of time spent asleep while in bed) and the total minutes spent asleep (Mellman & Uhde, 1989), and increased movement time in those with PD (Brown & Uhde, 2003). Interestingly, movement time is decreased on nights in which there is a nocturnal panic (NP) attack (Brown & Uhde, 2003). This has led to the speculation that NP might occur in reaction to greater relaxation (as evidenced by the perception of decreased movement) in those prone to relaxation-induced anxiety. Those with PD are often characterized by relaxation-induced anxiety (Craske, Lang, Tsao, Mystkowski, and Rowe, 2001), thus the perception of less movement and increased relaxation may be a possible trigger for some with NP.

Nocturnal panic occurs in up to 70% of those with panic disorder, although only about half of these cases experience nocturnal panic on a regular basis (Craske & Barlow, 1989; Mellman & Uhde, 1989). The CBT model of nocturnal panic is essentially the same as it is for PD. That is, panic attacks are postulated to occur in response to physiologic changes in an individual fearful of particular bodily symptoms. Normal physiologic changes in breathing, heart rate, or muscle activity during sleep are perceived as a possible problem producing an arousal and subsequent panic attack. Those with nocturnal panic appear to engage in catastrophic thinking about bodily sensations, a process hypothesized to rouse them out of sleep (Craske, Lang, & Rowe, 2002). While it is intriguing to think that those with NP represent a more severe form of PD, there is little evidence to support this contention (Craske, Lang, Mystkowski et al., 2002).

There are limited data regarding the efficacy of nocturnal panic treatments. Some data support the use of antidepressant medications (Mellman & Uhde, 1990) or single-agent anxiety medications such as alprazolam (Cameron & Thyer, 1985). Cognitive Behavioral Therapy (Craske, Lang, Aikins, & Mystkowski, 2005) for nocturnal panic appears to be an effective treatment, although randomized controlled clinical trials are needed to validate these preliminary efficacy findings.

There are no currently published trials of CBT for treating the insomnia in PD sufferers. Specific treatment of the sleep disturbance may be needed, since Cervena et al. reported that conventional therapy of PD in 20 subjects was not sufficient to treat the coexisting insomnia (Cervena, Matousek, Prasko, Brunovsky, & Paskova, 2005). Smith and colleagues raise an interesting concern with cognitive behavioral insomnia therapy in this population (Smith, Huang, & Manber, 2005). They argue that the sleep restriction component could potentially precipitate daytime PD, and thus, this component should be used with caution in patients suffering from this condition. There are a few studies supporting the concern that partial sleep deprivation lowers PD thresholds (Mellman & Uhde, 1989; Roy-Byrne, Uhde, & Post, 1986), so clinicians may want to restrict the time spent in bed to a lesser extent in those with frequent NP. An abbreviated treatment protocol for NP is more thoroughly presented in Chap. 8.

Generalized Anxiety Disorder

Generalized Anxiety Disorder (GAD) is a disorder characterized by excessive anxiety and pervasive worry. These symptoms are reflected in the DSM-IV-TR criterion A, which specifies excessive anxiety and worry about a number of matters (American Psychiatric Association, 1997). The worry and anxiety must be frequent (i.e., on more days than not for at least 6 months). Criterion B indicates that the experience of the worry is difficult to control. criterion C stipulates the cooccurrence of at least three related symptoms, including insomnia, restlessness, fatigue, impaired concentration, irritability, and muscle tension. GAD and insomnia are intricately tied in the literature perhaps because of the apparent frequency of worry present in insomnia (Borkovec, 1982; Harvey & Greenall, 2003) and the frequency of insomnia in GAD (Culpepper, 2002). In addition, the list of symptoms in criterion C (e.g., restlessness; easily fatigued; difficulty concentrating; irritability; muscle tension; insomnia) overlap considerably with the day and nighttime symptoms of insomnia (Edinger et al., 2004). One key way to distinguish GAD from an insomnia sufferer who is worried about his/her sleep and daytime functioning is that criterion D, which state that "the focus of the anxiety and worry, is not confined to features of an Axis I disorder" (p. 436); thus, if the worry is confined to insomnia-specific topics such as sleep and daytime functioning, an insomnia diagnosis may be more appropriate.

Since sleep disturbance is a symptom in the diagnostic criteria of GAD (American Psychiatric Association, 1997), it is no surprise that people with GAD often have subjective sleep complaints. These complaints have also been found on objective sleep measures. When compared with healthy controls, polysomnographic investigations in those with GAD have generally found evidence of markedly disrupted and fragmented sleep (Monti & Monti, 2000). For example, there is evidence of increased sleep onset latency, reduced sleep efficiency, increased amounts of "light" stages of sleep (i.e., 1 and 2 NREM sleep), reduced "deep" sleep

(i.e., SWS/delta sleep), and increased frequency and duration of awakenings (Papadimitriou, Kerkhofs, Kempenaers, & Mendlewicz, 1988; Reynolds III, Shaw, Newton, Coble, & Kupfer, 1983; Rosa, Bonnet, & Kramer, 1983). In addition, there is a correlation between ratings of anxiety and polysomnographic indices of sleep disruption including the number of awakenings, the latency to stage 1 sleep, and the percentage of stage 2 sleep (Rosa et al., 1983). As noted by Fuller and colleagues, polysomnographic investigations of those with anxiety disorders sometimes can be confounded by factors such as comorbidity and the duration of their anxiety problems (Fuller, Waters, Binks, & Anderson, 1997). Studies designed to tease apart these potential confounds have compared those with high worry and no Axis I disorder to those with low worry and no Axis I disorder. Results of this research has shown that those with high levels of trait worry evidenced longer sleep onset latencies, decreased slow wave sleep, and more frequent transitions into 1 NREM sleep than did those with low levels of worry. These results suggest that the polysomnographic findings of delayed sleep onset and decreased sleep continuity/depth reported in those with GAD are not attributable to comorbid problems or adjustments to longstanding psychopathology. These results in concert with other similar findings (Gross & Borkovec, 1982) suggest that increased worry can disrupt sleep and sleep disruption can increase worry.

There are no published trials that specifically test CBT for insomnia in those with GAD. Perhaps, such studies have not been conducted since one study found that residual insomnia may be less of an issue in those successfully treated with Cognitive Behavior Therapy for GAD (Belanger, Morin, Langlois, & Ladouceur, 2004). If GAD treatment successfully addresses insomnia, it suggests that insomnia treatment would be unnecessary. However, one caveat to this study is that the sample included in this previous trial was relatively mild with respect to insomnia severity, as the mean Insomnia Severity Index scores were below the suggested clinical cutoff for a probable insomnia diagnosis. Another caveat is that there was no follow-up period to assess whether sleep improvements were maintained after treatment. Interestingly, this study found that the severity of the GAD symptoms was not significantly related to the severity of insomnia symptoms suggesting at least some independence of the two syndromes. This picture becomes more complicated by adding results from a large scale combined medication trial for GAD that suggested treating insomnia concurrently with GAD (escitalopram plus eszopiclone) produces greater (and faster) anxiety reduction than treating GAD alone (escitalopram only) (Pollack et al., 2008). In the dual therapy group, the improvement (i.e., change scores) on sleep onset latency and wakefulness after sleep onset were approximately double that of the monotherapy (treating GAD alone). The number of patients with a clinically significant treatment response (i.e., a 50% or greater decline in anxiety scores at posttreatment) was 10% higher in the combined therapy compared to mono-therapy that targeted GAD alone. Although, anxiety improvements were maintained into the follow-up period, sleep improvements were not. Most participants (75%) had insomnia severity index scores that were above the clinical range for insomnia. Hence, the study sample for this trial was a more severely sleep disturbed group than the sample included in the Belanger et al.

([2004](#)) trial. Unfortunately, these two trials are the only published treatment studies concerning insomnia GAD. It would appear that dual therapy that targets both sleep and anxiety would be the treatment of choice in those with clinically significant insomnia (Monti & Monti, [2000](#)). In mild insomnia cases, a CBT for GAD intervention may be sufficient, although it is unknown if sleep improvements are maintained after treatment. CBT for insomnia has more durable treatment effects than insomnia-focused pharmacologic interventions (Morin et al., [2006](#)), so it would be interesting to test a treatment for GAD combined with CBT for insomnia against a GAD mono-therapy.

Posttraumatic Stress Disorder: PTSD

Posttraumatic Stress Disorder (PTSD) is a serious and unfortunately all too common disorder that arises in individuals exposed to unexpected and traumatic life events. PTSD may be precipitated by personal exposure to an event that involves serious injury to self or others, a threat of death to oneself, serious injury or death of others, or threats to the physical integrity and safety of oneself or others. This condition may also arise from "learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member of close associate" (American Psychiatric Association, [1997](#)). Events most commonly leading to PTSD include experiences such as the holocaust, military combat, personal assault (e.g., rape, physical attack), being taken hostage, torture, terrorist attacks, severe automobile or other accidents, man-made or natural disasters, or being diagnosed with a serious illness. Witnessing others exposed to such events as well as seeing dead bodies or body parts may also lead to this condition. Those who develop PTSD respond to such events with intense fear, horror, and/or helplessness and subsequently continue to reexperience the event through intrusive memories, nightmares, or even dissociative "flashbacks" during which the trauma event is relived. Additionally, the PTSD sufferer shows a persistent avoidance of all reminders of the traumatic event and manifests generalized "numbing" of responsiveness and chronic symptoms of heightened arousal.

Current evidence suggests that PTSD is both a relatively prevalent and often debilitating form of mental disorder. Lifetime prevalence estimates for PTSD suggest this condition may affect as many as 1–9% of the general population and 6–45% of all trauma victims (Hoge et al., [2004](#); Kessler et al., [2001](#); Pillar, Harder, & Malhotra, [2006](#); Stein & Mellman, [2005](#)). Factors related to the risk for developing PTSD include genetics, the nature/severity of the trauma, family/developmental history, preexisting mental disorders, and the nature of the recovery environment (American Psychiatric Association, [1997](#); Pillar et al., [2006](#)). Those who develop PTSD experience considerable morbidity in that they are at greater risk for academic and vocational underachievement, marital instability, unemployment, substance abuse, the development of comorbid mental disorders, and poor physical health (Kessler et al., [2001](#)). They are also up to six times more likely to attempt suicide

than are age-matched controls without PTSD symptoms (Kessler et al., 2001). They also incur greater healthcare costs since they use more outpatient medical and mental health services than do age-matched controls (Calhoun, Bosworth, Grambow, Dudley, & Beckham, 2002). To put these costs into perspective, it is estimated that the economic burden of PTSD in the US may be comparable to that of major depression (Walker et al., 2003). Although as many as one half of all PTSD sufferers show remission within 3 months of their trauma exposure, a substantial proportion suffer from PTSD symptoms for decades after the onset of their condition.

For many, if not most, PTSD sufferers, sleep disturbance plays a prominent role in the onset and maintenance of their symptoms. Unlike several of the other anxiety disorders discussed herein, PTSD is characterized by sleep-specific complaints. Indeed, the DSM-IV-TR diagnostic criteria include such complaints as difficulty falling or staying asleep as well as recurrent distressing dreams of the precipitating traumatic event (American Psychiatric Association, 1997). Although such complaints are not mandatory for assigning a PTSD diagnosis, survey studies have demonstrated that insomnia complaints and reports of distressing, sleep-disruptive nightmares are present in the majority of those with this condition (Kuch & Cox, 1992; Neylan et al., 1998). Furthermore, such individuals appear more likely to report excessive body movements and night terrors (i.e., screaming or shaking) during their sleep than do matched controls without PTSD symptoms (Mellman, Kulick-Bell, Ashlock, & Nolan, 1995). Interestingly, one study showed that the presence of subjective sleep complaints one month after experiencing their trauma was predictive of the eventual development of PTSD in a large group of traffic accident victims (Thase et al., 2002).

In contrast to these studies, results of objective, polysomnographic sleep monitoring have provided somewhat equivocal evidence for the presence of sleep pathology in PTSD sufferers. Despite the chronic insomnia complaints common among PTSD patients, PSG studies have been inconsistent in showing marked sleep continuity differences between age and gender matched groups with and without this condition (Dow, Kelsoe, & Gillin, 1996; Fisk et al., 1994; Hurwitz, Mahowald, Kuskowski, & Engdahl, 1998; Mellman, Kulick-Bell et al., 1995; Mellman, Nolan, Hebding, & Kulick-Bell, 1997; Woodward, Friedman, & Bliwise, 1996). Collectively considered, these studies indicate that sleep is slightly, albeit often not significantly, more disturbed than the sleep of matched controls by objective measures. In a more recent large study (Breslau et al., 2004) of a large representative community sample, no polysomnographic sleep differences were noted between those with and without PTSD, other than increased REM-related arousals in the PTSD group. Findings such as these contrast markedly with the subjective complaints of most PTSD sufferers. This apparent subjective/objective discrepancy has led to the notion that PTSD patients may have a form of sleep state misperception, leading them to over-report their sleep difficulties (Hurwitz et al., 1998; Pillar et al., 2006). However, other studies have shown such findings as greater REM density (i.e., more rapid eye movements during REM sleep), tonic and phasic REM activity, and body/leg movements and EMG activity during sleep in PTSD sufferers among normal sleepers (Lavie & Hertz, 1979; Ross, Ball, Sullivan, & Caroff, 1989).

Additionally, one study showed that PTSD sufferers evidenced greater levels of arousal during sleep as measured by heart rate and sleep EEG spectral measures than did matched controls (Woodward, Murburg, & Bliwise, 2000). There also has been speculation that the arousal mechanism in PTSD leads to increased risk for sleep-disordered breathing in patients with this condition (Krakow et al., 2000; Pillar et al., 2006). Unfortunately, many of the PSG studies of PTSD have been confounded by the inclusion of medicated patients, those with comorbid mental conditions and patients at various stages (acute vs. chronic) of their conditions. It should also be noted that the most recent and comprehensive meta-analysis of the literature (Kobayashi, Boarts, & Delahanty, 2007) highlights limitations in this literature and convincingly documents objective sleep difficulties in those who suffer from PTSD syndromes. Thus, PTSD sufferers do appear to suffer from the objective sleep problems of which they complain.

Given the prominence of sleep-related complaints in this group, there has been significant interest in the role or meaning of sleep difficulties in the development and maintenance of this condition. Data suggests that PTSD sufferers do not show a normal drop in noradrenergic production during the nighttime (Mellman, Kuman, Kulick-Bell, Kuman, & Nolan, 1995). This finding in turn supports the speculation of the central role of "hyerarousal" in disruptive nighttime sleep and enhancing daytime startle and hypervigilence in PTSD sufferers. In contrast, other studies showing that PTSD sufferers have elevated awakening thresholds from sleep, suggest that a pathological "sleep deepening" may occur as a compensatory mechanism to suppress trauma related material that emerges during sleep (Dagan, Lavie, & Bleich, 1991; Lavie, Katz, Pillar, & Zinger, 1998). It has also been speculated that chronic hyperarousal leads to insomnia and partial sleep deprivation that gives way to heightened arousal thresholds during recovery sleep (Pillar et al., 2006). Others have focused on the aberrations of REM sleep and the presence of recurrent, repetitive nightmares in PTSD and have postulated that an abnormal REM mechanism is central to this disorder (Fisk et al., 1994). Whereas each of these theories has some appeal, additional studies that carefully control the effects of comorbidities, concurrent medication use, and the specific nature of the trauma are needed to determine which current theory, if any, is best supported by the data.

Current treatment strategies for PTSD include pharmacotherapy with various compounds (e.g., selective serotonin re-uptake inhibitors, tricyclic antidepessants, momamine oxidase inhibitors) and psychological treatments such as cognitive-behavior therapy specifically tailored for this condition. However, as noted recently, clinically significant residual insomnia may persist in almost one half of those patients who achieve PTSD remission with treatment (Zayfert & DeViva, 2004). In such cases with long-standing sleep complaints, insomnia may have developed some independence over time and represent a separate and clinically significant comorbid disorder. Hence, sleep targeted therapies such as the cognitive-behavior insomnia therapy described in Chap. 3 may be particularly appropriate for PTSD sufferers who present insomnia complaints in the context of their PTSD syndrome and also as residual symptoms. Distressing and recurrent nightmares also are a hallmark and often treatment-resistant symptom of this disorder and merit specific

treatment attention. As discussed in more detail in Chap. 7, imagery rehearsal therapy (IRT) involving nightmare rescripting and subsequent imagery rehearsal of the rescripted nightmare has proven particularly effective for management of PTSD-related nightmares. Indeed, this treatment has shown efficacy in groups such as combat veterans and female rape victims (Forbes, Phelps, & McHugh, 2001; Krakow et al., 2001). In many cases, patterns of nighttime sleep avoidance to minimize nightmare exposure and daytime compensatory napping develop as PTSD coping strategies. Those who present with this pattern have a complicated insomnia problem and may benefit from both CBT for insomnia and IRT to manage nightmares. Along with these treatment targets, some may develop excessive and elaborate safety behaviors such as checking door locks or the perimeter around their homes that delay their bedtime, whereas others may self-medicate with alcohol to aid their sleep. Such problem behaviors should be identified and addressed therapeutically to maximize sleep outcomes in PTSD sufferers. Finally, due to the high rate of comorbid mental conditions and particularly depression, appropriate treatment for such conditions is often needed to maximize sleep outcomes among those who suffer from this condition.

Obsessive–Compulsive Disorder

Obsessive–compulsive disorder is a rather debilitating condition characterized by spontaneous, recurring, and upsetting thoughts (obsessions) coupled with repetitive or ritualistic behaviors or mental acts performed as a means of reducing or controlling the anxiety precipitated by the associated obsessions (American Psychiatric Association, 1997). The content of obsessions may vary but often centers on thoughts of germ exposure/contamination, unwelcome sexual urges, doubts about having performed some necessary task (e.g., locking the doors before going to bed), personal or religious failings, or loss of control of aggressive impulses. Compulsions arising from and accompanying such obsessions may include hand washing, repetitive counting or praying, repeated checking, or ritualistic arranging or ordering of personal items. Typically, the person feels driven to engage in the compulsive act in order to control the obsessional thought and/or to prevent some feared and undesired event from occurring. For example, an individual with obsessional fears focused on germ exposure and consequent illness may repeatedly engage in hand washing after contact with what is perceived as a contaminated surface or item so as to reduce chances of developing a serious illness. At some point during the course of this disorder, the affected individual recognizes the obsessions and associated compulsions are irrational, but these symptoms persist over time despite this insight.

Compared to other anxiety disorders, obsessive–compulsive disorder is relatively rare, albeit often very persistent and debilitating. Most epidemiological surveys show a stable prevalence of obsessive–compulsive disorder with approximately 2% of the general population of Western societies meeting criteria for this condition. Those with obsessive–compulsive disorder are not particularly prone to present in

primary care settings (Fireman, Koran, Leventhal, & Jacobson, 2001), but as many as 9.2 % of those who seek psychiatric treatment suffer from this condition (Hantouche, Bouhassira, Lancrenon, Ravily, & Bourgeois, 1995). These findings are, perhaps, not surprising inasmuch as many as two thirds of those with obsessive–compulsive disorder present with comorbid mental disorders, particularly mood or other anxiety disorders (Torres et al., 2006; Tukel, Meteris, Koyuncu, Tecer, & Yazici, 2006). Obsessive–compulsive disorder tends to be chronic in as many as 60% of affected individuals (Angst et al., 2004), and almost one half of those who eventually achieve full symptomatic remission later suffer relapse (Eisen et al., 1999). Whereas some persons with obsessive–compulsive disorder may suffer surprisingly little social and vocational impairment, this condition often results in considerable impairment of social and occupational functioning. In more protracted cases, this disorder may contribute to reduced quality of life, impairment of family and social relationships, reduced productivity and heightened absenteeism from work, chronic disability, and a markedly increased risk for suicide (American Psychiatric Association, 1997; Kamath, Reddy, & Kandavel, 2007; Stengler-Wenzke, Krolla, Matschingera, & Angermeyera, 2006; Torres et al., 2006).

Insomnia and other forms of sleep disturbance are not considered core symptoms or primary associated features of obsessive–compulsive disorder (Stein & Mellman, 2005). However, one recent study (Voderholzer et al., 2007) indicated that those with obsessive–compulsive disorder show relative disturbances of sleep continuity (i.e., more fragmented sleep) compared with well-matched noncomplaining normal sleepers. Another recent study (Kluge, Schüssler, Künzel et al., 2007) showed that obsessive–compulsive disorder sufferers displayed higher plasma concentration levels of ACTH and cortisol during their sleep than did normal controls. Alterations in REM and slow wave sleep architecture have also been noted in some (Insel et al., 1982; Kluge, Schüssler, Dresler, Yassouridis, & Steiger, 2007) but not all obsessive–compulsive sufferers (Hohagen et al., 1994; Robinson, Walsleben, Pollack, & Lerner, 1998; Voderholzer et al., 2007). Considered collectively, these findings suggest obsessive–compulsive disorder patients may have a relative propensity for disrupted nocturnal sleep perhaps mediated by over-activity of the HPA axis. Of course, insomnia may develop independently because of other factors and exist as a comorbid condition, as is the case with other anxiety disorders.

As noted by Smith and colleagues (Smith et al., 2005), compulsive behaviors may sometimes play a role in insomnia complaints. For example, compulsive checking that doors are locked or repetitive praying before retiring for the night may interfere with the act of falling asleep and markedly delay sleep onset. Also, given the recent findings implicating possible over-activity of the HPA axis in obsessive–compulsive patients, excessive arousal during the nighttime may complicate the sleep of some such patients. Furthermore, it is noteworthy that unhelpful beliefs are thought to perpetuate the symptoms of at least some obsessive–compulsive patients (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006). Hence, it seems reasonable to speculate that such patients could have propensity for developing the previously mentioned (see Chap. 2) types of unhelpful beliefs thought to contribute and sustain insomnia. Given these possibilities, a thorough assessment

of factors such as the patient's level of arousal at bedtime, presence of sleep-disruptive compulsions, and unhelpful sleep-related beliefs may be particularly important when evaluating the insomnia complaints of obsessive–compulsive disorder patients. Cognitive and behavioral therapies that reduce bedtime arousal, alter unhelpful beliefs and effectively manage sleep-disruptive rituals may all be useful in managing the insomnia complaints of these patients.

Social Phobia

Social phobia is a fairly prevalent and, frequently debilitating condition characterized by a markedly persistent fear and avoidance of one or more social situations involving exposure to unfamiliar people and/or evaluative scrutiny by others (American Psychiatric Association, 1997). When those with social phobia encounter a situation wherein they expect scrutiny and possible evaluation by others, they experience extreme anxiety. In some cases, this anxiety may culminate in panic characterized by extreme discomfort, palpitations, tremulousness, blushing sweating, and pronounced fears of social rejection or negative evaluation by others. However, unlike the unpredictable, spontaneous panic attacks that characterize panic disorder, those with social phobia recognize that their panic symptoms are situation-specific and derive from their concerns about scrutiny and negative appraisals (Stein & Mellman, 2005). Whereas those with social phobia realize that their fears and beliefs about social scrutiny/ evaluation are unhelpful and often disproportionate to their actual social experiences, they nonetheless remain symptomatic and attempt to avoid or minimize contact with social situations that provoke their physiologic and cognitive phobic symptoms.

Epidemiological studies suggest that between 3% and 13% of the general population suffer from social phobia at some time during their lives (American Psychiatric Association; Cairney et al., 2007; Grant et al., 2005). In clinical samples, prevalence rates are higher with reported rates ranging between 10 and 20% (American Psychiatric Association, 1997) among outpatients with anxiety disorders and up to 26% (Todaro, Shen, Raffa, Tilkemeier, & Niaura, 2007) among inpatients with selected comorbid medical conditions. Individuals with social phobia most often fear speaking in public or interacting with strangers. Less common are fears of performing such activities as eating, drinking or writing in public. In a subset of those with social phobia, social fears and avoidance pervade most routine social situations and, in such cases, the term, generalized social phobia is typically applied. Over time, social phobia places individuals at risk for considerable morbidity including a reduced number and quality of social relationships, a reduced likelihood of marriage, academic and vocational underachievement, disability, and eventual onset of depression and other serious psychiatric conditions (American Psychiatric Association, 1997; Beesdo et al., 2007; Stein & Mellman, 2005). Self-medication with alcohol or other substances may give way to substance abuse/

dependence in a subset of those with this condition, particularly those with general social phobia.

There is mixed evidence that social phobia confers some risk for the development of insomnia. Stein, Kroft and Walker (1993), for example, compared the sleep appraisals of patients with generalized social phobia and a matched group of healthy controls using the Pittsburgh Sleep Quality Index, a measure with high sensitivity and specificity for insomnia (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Comparisons showed that those with social phobia reported significantly poorer sleep quality, longer latencies to sleep onset, more frequent nights with sleep disturbance, and more pronounced daytime dysfunction that did the controls. In contrast, PSG comparisons (Brown, Black, & Uhde, 1994; Papadimitriou & Linkowski, 2005) have shown no differences between those with social phobia and healthy controls on standard sleep measures of sleep onset latency, sleep efficiency, REM latency, REM distribution, REM density, or other measures of sleep architecture. Nonetheless, studies have shown that PSG is less prone to discriminate normal sleepers from insomnia sufferers than are subjective measures such as self-report questionnaires or data derived from subjective sleep diaries (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006; Lineberger, Carney, Edinger, & Means, 2006). Hence, the subjective sleep complaints of those with social phobia should not be underestimated.

As noted by Weissberg, (Weissberg, 2006) social phobia typically involves a form of performance anxiety. It is noteworthy that when faced with the challenge of sleeping, performance anxiety is thought to perpetuate psychophysiological insomnia. Hence, it is possible that this inherent form of anxiety in the social phobic enhances risk for sleep difficulties and should be considered a potential treatment target, at least in some people. In other cases, sleep disturbance may be traced to comorbid depression that evolves as a consequent of the phobic condition. Of course, secondary sleep difficulties may emerge in those who abuse alcohol to cope with social phobia. Given these possibilities, a thorough assessment of factors such as sleep-related performance anxiety, comorbid mood disturbance, and substance use patterns should be included in the evaluation of insomnia complaints in patients who also suffer from social phobias. In turn, cognitive and behavioral therapies that target performance anxiety and mood disturbance as well as specialized substance abuse treatment programs may all be of some value in the management of social phobia in those who present with insomnia complaints.

Specific Phobias

Specific phobia is a condition characterized by marked fear and avoidance of an object or situation (American Psychiatric Association, 1997). For example, someone with a fear of flying may be able to avoid flying by taking ground transportation. If a situation necessitated air travel (e.g., a mandatory business trip to an island), the Specific Phobia sufferer might be able to fly but only with intense anxiety

and fear. The criteria for this disorder also stipulate that: (1) exposure to the feared stimulus results in an immediate anxiety response; (2) the person realizes that the fear is excessive/unreasonable; (3) the phobic situation/stimulus is avoided or endured with intense anxiety/distress; (4) the phobia produces marked distress or functional impairment; (5) and the anxiety/avoidance is not better accounted for by another disorder (American Psychiatric Association, 1997). In adults, the duration criterion is at least 6 months. As outlined by the DSM-IV-TR (American Psychiatric Association, 1997), various types of specific phobia types exist including: (1) Animal type (fear of animals or insects); (2) Natural Environment type (storms, heights, water); (3) Blood-Injection-Injury type (fear of seeing or receiving an injection, medical procedures etc.); (4) Situational type (fear of situations such as riding in an elevator, enclosed spaces, etc.); and (5) Other (phobias that do not fall into the aforementioned types). Prevalence rates for these Specific Phobias are about 10% and approximately 12% lifetime (Kessler et al., 2005). Although not all Specific Phobias have been the subject of treatment efficacy trials, available data suggests that the most commonly occurring Specific Phobias are effectively treated with Cognitive Behavior Therapy consisting of exposure and some form of cognitive restructuring (Antony & Barlow, 2002).

Currently, studies concerning the relation between specific phobias and insomnia or other forms of sleep disturbance are generally lacking. However, it is noteworthy that claustrophobia has been shown to affect adherence to Continuous Positive Airway Pressure treatment of sleep apnea (Edinger & Radtke, 1993) although admittedly this difficulty is not specifically linked to insomnia. Nonetheless, these cases of claustrophobic responses to CPAP treatment of sleep apnea are effectively treated with the behavioral intervention, in-vivo exposure (Edinger & Radtke, 1993; Means & Edinger, 2007). See Chap. 9 for a description of this treatment protocol.

Conceivably, some specific phobias could disrupt sleep. For example, a severe case of arachnophobia could disrupt sleep if there was concern that spiders were in the sleeping environment. Although we could not locate scientific accounts of adult fear of the dark (scotophobia) and insomnia, it is not entirely uncommon to see this problem clinically. In addition, there are some self-help interventions available on the Internet (i.e., guided self-hypnosis) to help adults with a fear of the dark. One reason for the relative absence of scientific accounts may be a stigma attached to acknowledging a persisting fear typically associated with childhood. Turning off the lights has been shown to increase arousal and fear, and facilitate an exaggerated startle response in adults (Grillon, Pellowski, Merikangas, & Davis, 1997), and such reactivity appears to relate to the common fear of the dark seen in children (Grillon et al., 1997). It would be interesting to investigate whether turning off the lights is a mechanism for hyperarousal in insomnia. If a stimulus such as darkness elicits heightened anxiety, this would be expected to interfere with normal sleep onset.

Somniphobia, the fear of sleep, is one specific phobia sometimes associated with general sleep difficulty or insomnia. There are several sleep disorders that could lead to a fear of sleep. The frightening sensorimotor experiences characteristic of sleep paralysis or hypnagogic hallucinations could cause a fear of sleep to develop. Although published papers on "fear of sleep" as a phobia are currently lacking, this

construct is often invoked to explain sleep-avoidant behaviors. PTSD sufferers will often show a fear of sleep and sleep avoidance in reaction to their chronic disturbing nightmares. Likewise, psychophysiologic insomnia sufferers may also manifest "fear of sleep" or emotional reactivity to their impending bedtime. Indeed, one criterion for the diagnosis of psychophysiologic insomnia in the International Classification of Sleep Disorders, Diagnostic and Coding Manual is heightened anxiety about sleep (American Academy of Sleep Medicine, 2005). Some insomnia sufferers begin to "dread" sleep because of the repeated negative emotional experience of not being able to sleep. This conditioning process is seen as a key etiologic factor for many insomnia sufferers. CBT strategies such as stimulus control and cognitive therapy (detailed in Chaps. 7 and 8) target conditioned arousal and fears about sleep and, thus, may be effective in such cases.

Summary

Insomnia as a symptom is a feature of many anxiety disorders. Insomnia can also be an important comorbid condition in anxiety disorders. There can be subjective complaints and EEG-verified sleep disturbances across the range of Anxiety Disorders. Sleep problems may be most prevalent in those with GAD and PTSD. There is no real impediment to treating the insomnia of those with anxiety disorders, although there may be some special treatment considerations for panic disorder (i.e., allowing for a less conservative time-in-bed restriction) and PTSD (i.e., addressing nightmares). There is some evidence that treating the comorbid anxiety disorder only (and thus ignoring the insomnia) may limit the degree of anxiety disorder treatment response and/or result in residual insomnia. A combined approach requires more investigation, but preliminary studies have been promising.

References

American Academy of Sleep Medicine. (2005). *International classification of sleep disorders-second edition (ICSD-2). International Classification of Sleep Disorders Steering Committee, P. J. Hauri (Chairman)*. Chicago.

American Psychiatric Association. (1997). *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition,Text Revision (DSM-IV-TR)* (4th ed.). Washington: DC: American Psychiatric Association.

Angst, J., Gamma, A., Endrass, J., Goodwin, R., Ajdacic, V., Eich, D., et al. (2004). *European Archives of Psychiatry and Clinical Neuroscience, 254*(3), 156–164.

Antony, M., & Barlow, D. H. (2002). Specific phobias. In D. H. Barlow (Ed.), *Anxiety and Its Disorders* (Vol. 2, pp. 380–417). New York: The Guilford Press.

Beesdo, K., Bittner, A., Pine, D., Stein, M., Höfler, M., Lieb, R., et al. (2007). Incidence of social anxiety disorder and the consistent risk for secondary depression in the first three decades of life. *Archives of General Psychiatry, 64*(8), 903–912.

Belanger, L., Morin, C. M., Langlois, F., & Ladouceur, R. (2004). Insomnia and generalized anxiety disorder: Effects of cognitive behavior therapy for GAD on insomnia symptoms. *Journal of Anxiety Disorders, 18*, 561–571.

Borkovec, T. D. (1982). Insomnia. *Journal of Consulting and Clinical Psychology, 50*, 880–895.

Breslau, N., Roth, T., Burduvali, E., Kapke, A., Schultz, L., & Roehrs, T. (2004). Sleep in lifetime posttraumatic stress disorder: a community-based polysomnographic study. *Archives of General Psychiatry, 61*, 508–516.

Brown, T., Black, B., & Uhde, T. (1994). The sleep architecture of social phobia. *Biological Psychiatry, 35*(6), 420–421.

Brown, T. M., & Uhde, T. W. (2003). Sleep panic attacks: a micro-movement analysis. *Depression and Anxiety, 18*, 214–220.

Buysse, D., Ancoli-Israel, S., Edinger, J., Lichstein, K., & Morin, C. (2006). Recommendations for a standard research assessment of insomnia. *Sleep, 29*(9), 1155–1173.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213.

Cairney, J., McCabe, L., Veldhuizen, S., Corna, L., Streiner, D., & Herrmann, N. (2007). Epidemiology of social phobia in later life. *American Journal of Psychiatry, 15*(3), 224–233.

Calhoun, P., Bosworth, H., Grambow, S., Dudley, T., & Beckham, J. (2002). Medical service utilization by veterans seeking help for posttraumatic stress disorder. *American Journal of Psychiatry, 159*, 2081–2086.

Cameron, O. G., & Thyer, B. A. (1985). Treatment of pavor nocturnus with alprazolam. *Journnal of Clinical Psychology, 46*, 405.

Cervena, K., Matousek, M., Prasko, J., Brunovsky, M., & Paskova, B. (2005). Sleep disturbances in patients treated for panic disorder. *Sleep Medicine Reviews, 6*, 149–153.

Craske, M. G., & Barlow, D. H. (1989). Nocturnal panic. *Journal of Nervous and Mental Disease, 177*, 160–167.

Craske, M. G., Lang, A. J., Aikins, D., & Mystkowski, J. L. (2005). *Behavior Therapy, 36*, 43–54.

Craske, M. G., Lang, A. J., Mystkowski, J. L., Zucker, B. G., Bystritsky, A., & Yan-Go, F. (2002). Does nocturnal panic represent a more severe form of panic disorder? *Journal of Nervous and Mental Disease, 190*, 611–618.

Craske, M. G., Lang, A. J., & Rowe, M. (2002). Pre-sleep attributions about arousal during sleep. *Journal of Abnormal Psychology, 111*, 53–62.

Craske, M. G., Lang, A. J., Tsao, J. C. I., Mystkowski, J. L., & Rowe, M. K. (2001). Reactivity to interoceptive cues in nocturnal panic. *Journal of Behavior Therapy and Experimental Psychiatry, 32*, 173–190.

Culpepper, L. (2002). Generalized anxiety disorder in primary care: emerging issues in management and treatment. *The Journal of Clinical Psychiatry, 63*(Suppl. 8), 35–42.

Dagan, Y., Lavie, P., & Bleich, A. (1991). Elevated awakening thresholds in sleep stage 3–4 in war-related post-traumatic stress disorder. *Biological Psychiatry, 30*, 618–622.

Dow, B. M., Kelsoe, J., & Gillin, J. (1996). Sleep and dreams in Vietnam PTSD and depression. *Biological Psychiatry, 39*, 42–50.

Edinger, J. D., Bonnet, M., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., et al. (2004). Derivation of research diagnostic criteria for insomnia: Report on an American Academy of Sleep Medicine work group. *Sleep, 27*, 1567–1596.

Edinger, J. D., & Radtke, R. A. (1993). Use of in vivo desensitization to treat a patient's claustrophobic response to nasal CPAP. *Sleep, 16*(7), 678–680.

Eisen, J., Goodman, W., Keller, M., Warshaw, M., DeMarco, L., Luce, D., et al. (1999). Patterns of remission and relapse in obsessive-compulsive disorder: a 2-year prospective study. *Journal of Clinical Psychiatry, 60*(5), 346–351.

Espie, C., Broomfield, N., MacMahon, K., Macphee, L., & Taylor, L. M. (2006). The attention-intention-effort pathway in the development of psychophysiologic insomnia: a theoretical review. *Sleep Medicine Reviews, 10*(4), 215–245.

Fireman, B., Koran, L., Leventhal, J., & Jacobson, A. (2001). The prevalence of clinically recognized obsessive-compulsive disorder in a large health maintenance organization. *American Journal of Psychiatry, 158*(11), 1904–1910.

Fisk, J. D., Ritvo, P. G., Ross, L., Haase, D. A., Marrie, T. J., & Schlech, W. F. (1994). Measuring the functional impact of fatigue: Initial Validation of the Fatigue Impact Scale. *Clinical Infectious Diseases, 18*(Suppl 1), S79–S83.

Forbes, D., Phelps, A., & McHugh, T. (2001). Treatment of combat-related nightmares using imagery rehearsal therapy: a pilot study. *Journal of Traumatic Stress, 14*(2), 433–442.

Fuller, K. H., Waters, W. F., Binks, P. G., & Anderson, T. (1997). Generalized anxiety and sleep architecture: a polysomnographic investigation. *Sleep, 20*, 370–376.

Grant, B., Hasin, D., Blanco, C., Stinson, F., Chou, S., Goldstein, R., et al. (2005). The epidemiology of social anxiety disorder in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry, 66*(11), 1351.

Grillon, C., Pellowski, M., Merikangas, K. R., & Davis, M. (1997). Darkness facilitates the acoustic startle reflex in humans. *Biological Psychiatry, 42*(6), 453–460.

Gross, R., & Borkovec, T. (1982). Effects of cognitive intrusion manipulation on the sleep onset latency of good sleepers. *Behaviour Therapy, 13*, 112–116.

Hantouche, E., Bouhassira, M., Lancrenon, S., Ravily, V., & Bourgeois, M. (1995). Prevalence of obsessive-compulsive disorders in a large French patient population in psychiatric consultation. *Encephale, 21*(5), 571–580.

Harvey, A. G., & Greenall, E. (2003). Catastrophic worry in primary insomnia. *Journal of Behavior Therapy Experimental Psychiatry, 34*(1), 11–23.

Hoge, C. W., Castro, C., Messer, S., McGurk, D., Cotting, D., & Koffman, R. (2004). Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *New England Journal of Medicine, 351*, 13–22.

Hohagen, F., Lis, S., Krieger, S., Winkelmann, G., Riemann, D., Fritsch-Montero, R., et al. (1994). Sleep EEG of patients with obsessive-compulsive disorder. *European Archives of Psychiatry and Clinical Neuroscience, 243*(5), 273–278.

Hurwitz, T., Mahowald, M., Kuskowski, M., & Engdahl, B. (1998). Polysomnographic sleep is not clinicall impaired in Vietnam combat verterans with chronic posttraumatic stress disorder. *Biological Psychiatry, 44*, 1066–1073.

Insel, T., Gillin, J., Moore, A., Mendelson, W., Loewenstein, R., & Murphy, D. (1982). The sleep of patients with obsessive-compulsive disorder. *Archives of General Psychiatry, 39*(12), 1372–1377.

Kamath, P., Reddy, Y. C., & Kandavel, T. (2007). Suicidal behavior in obsessive-compulsive disorder. *Journal of Clinical Psychiatry, 68*(11), 1741–1750.

Kessler, R., Berglund, P., Bruce, M., Koch, J., Laska, E., Leaf, P., et al. (2001). The prevalence and correlates of untreated serious mental illness. *Health Services Research, 36*, 987–1007.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 593–602.

Kluge, M., Schüssler, P., Dresler, M., Yassouridis, A., & Steiger, A. (2007). Sleep onset REM periods in obsessive compulsive disorder. *Psychiatry Research, 152*(1), 29–35.

Kluge, M., Schüssler, P., Künzel, H., Dresler, M., Yassouridis, A., & Steiger, A. (2007). Increased nocturnal secretion of ACTH and cortisol in obsessive compulsive disorder. *Journal of Psychiatry Research, 41*(11), 928–933.

Kobayashi, I., Boarts, J. M., & Delahanty, D. L. (2007). Polysomnographically measured sleep abnormalities in PTSD: A meta-analytic review. *Psychophysiology, 44*(4), 660–669.

Krakow, B., Hollifield, M., Schrader, R., Koss, M., Tandberg, D., & Lauriello, J. (2000). A controlled study of imargery rehearsal for chronic nightmares in sexual assault survivors with PTSD: a preliminary report. *Journal of Traumatic Stress, 13*(4), 589–609.

Krakow, B., Johnston, L., Melendrez, D., Hollifield, M., Warner, T. D., Chavez-Kennedy, D., et al. (2001). An open-label trial of evidence-based cognitive behavior therapy for nightmares and insomnia in crime victims with PTSD. *American Journal of Psychiatry, 158*(12), 2043–2047.

Kuch, K., & Cox, B. (1992). Symptoms of PTSD in 124 survivors of the Holocaust. *American Journal of Psychiatry, 149*(3), 337–340.

Lavie, P., & Hertz, G. (1979). Increased sleep motility and respiration rates in combat neurotic patients. *Biological Psychiatry, 14*, 983–987.

Lavie, P., Katz, N., Pillar, G., & Zinger, Y. (1998). Elevated awakening thresholds during sleep: characteristics of chronic war-related posttraumatic stress disorder patients. *Biological Psychiatry, 44*, 1060–1065.

Lineberger, M., Carney, C., Edinger, J., & Means, M. (2006). Defining Insomnia: Quantitative criteria for insomnia severity and frequency. *Sleep, 29*, 479–485.

Means, M. K., & Edinger, J. D. (2007). Graded exposure therapy for addressing claustrophobic reactions to continuous positive airway pressure: a case series report. *Behavioral Sleep Medicine, 5*(2), 105–116.

Mellman, T., Kulick-Bell, R., Ashlock, L., & Nolan, B. (1995). Sleep events among veteran with posttraumatic stress disorder. *American Journal of Psychiatry, 152*, 110–115.

Mellman, T., Nolan, B., Hebding, J., & Kulick-Bell, R. (1997). A polysomnographic comparison of veterans with combat-related PTSD, depressed mean and non-ill controls. *Sleep, 20*, 46–51.

Mellman, T. A., Kuman, A., Kulick-Bell, R., Kuman, M., & Nolan, B. (1995). Nocturnal/daytime urine noradrenergic measures and sleep in combat-related PTSD. *Biological Psychiatry, 38*, 174–179.

Mellman, T. A., & Uhde, T. W. (1989). Sleep panic attacks: New clinical findings and theoretical implications. *American Journal of Psychiatry, 146*, 1024–1027.

Mellman, T. A., & Uhde, T. W. (1990). Patients with frequent sleep panic: Clinical findings and response to medication treatment. *Journal of Clinical Psychiatry, 51*(12), 513–516.

Monti, J. M., & Monti, D. (2000). Sleep disturbance in generalized anxiety disorder and its treatment. *Sleep Medicine Reviews, 4*(3), 263–276.

Morin, C., Bootzin, R., Buysse, D., Edinger, J., Espie, C., & Lichstein, K. (2006). Psychological and behavioral treatment of insomnia: update of the recent evidence (1998–2004). *Sleep, 29*(11), 1398–1414.

Neylan, T., Marmar, C., Metzler, T., Weiss, D., Zatzick, D., & Delucchi, K. (1998). Sleep disturbances in the Vietnam generation: Findings from a nationally representative sample of male Vietnam Veterans. *American Journal of Psychiatry, 155*, 929–933.

Papadimitriou, G., & Linkowski, P. (2005). Sleep disturbance in anxiety disorders. *International Review of Psychiatry, 17*(4), 229–236.

Papadimitriou, G. N., Kerkhofs, M., Kempenaers, C., & Mendlewicz, J. (1988). EEG sleep studies in patients with generalized anxiety disorder. *Psychiatry Research, 26*, 183–190.

Pillar, G., Harder, L., & Malhotra, A. (2006). Trauma and post-traumatic stress disorder. In T. Lee-Chiong (Ed.), *Sleep: A comprehensive handbook* (pp. 857–866). Hoboken, NJ: John Wiley & Sons.

Pollack, M. H., Kinrys, G., Krystal, A. D., McCall, V. W., Roth, T., Schaefer, K., et al. (2008). Eszopiclone coadministered with escitalopram in patients with insomnia and comorbid generalized anxiety disorder. *Archives of General Psychiatry, 65*(5), 551–562.

Reynolds, C. F., III, Shaw, D. H., Newton, T. F., Coble, P. A., & Kupfer, D. J. (1983). EEG sleep in outpatients with generalized anxiety: a preliminary comparison with depressed outpatients. *Psychiatry Research, 8*, 81–89.

Robinson, D., Walsleben, J., Pollack, S., & Lerner, G. (1998). Nocturnal polysomnography in obsessive-compulsive disorder. *Psychiatry Reseach, 80*(3), 257–263.

Rosa, R. R., Bonnet, M. H., & Kramer, M. (1983). The relationship of sleep and anxiety in anxious subjects. *Biological Psychology, 16*, 119–126.

Ross, R., Ball, W., Sullivan, K., & Caroff, S. (1989). Sleep disturbance as a hallmark of posttraumatic stress disorder. *American Journal of Psychiatry, 146*, 697–707.

Roy-Byrne, P. P., Uhde, T. W., & Post, R. M. (1986). Effects of one night's sleep deprivation on mood and behavior in panic disorder: Patients with panic disorder compared with depressed patients and normal controls. *Archives of General Psychiatry, 43*(9), 895–899.

Smith, M. T., Huang, M. I., & Manber, R. (2005). Cognitive Behavior Therapy for chronic insomnia occuring within the context of medical and psychiatric disorders. *Clinical Psychology Review, 25*, 559–592.

Stein, M., Kroft, C., & Walker, J. (1993). Sleep impairment in patients with social phobia. *Psychiatry Research, 49*(3), 251–256.

Stein, M., & Mellman, T. (2005). Anxiety Disorders. In M. Kyrger, T. Roth, & W. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 1297–1310). Philadelphia: Elsevier Saunders.

Stein, M. B., Enns, M. W., & Kryger, M. H. (1993). Sleep in nondepressed patients with panic disorder II: Polysomnographic assessment of sleep architecture and sleep continuity. *Journal of Affective Disorders, 28*, 1–6.

Stengler-Wenzke, K., Krolla, M., Matschingera, H., & Angermeyera, M. C. (2006). Quality of life of relatives of patients with obsessive-compulsive disorder. *Comprehensive Psychiatry, 47*(6), 523–527.

Thase, M. E., Rush, A. J., Manber, R., Kornstein, S. G., Klein, D., & Markowitz, J. C. (2002). Effects of nefazodone and cognitive behavioral analysis system of psychotherapy, singly and in combination, on insomnia associated with chronic depression. *Journal of Clinical Psychiatry, 63*(6), 493–500.

Todaro, J., Shen, B., Raffa, S., Tilkemeier, P., & Niaura, R. (2007). Prevalence of anxiety disorders in men and women with established coronary heart disease. *Journal of Cardiopulmonary Rehabilitation and Prevention, 27*(2), 86–91.

Torres, A. R., Prince, M. J., Bebbington, P. E., Bhugra, D., Brugha, T. S., Farrell, M., et al. (2006). Obsessive-compulsive disorder: Prevalence, comorbidity, impact, and help-seeking in the British National Psychiatric Morbidity Survey of 2000. *The American Journal of Psychiatry, 163*, 1978–1985.

Tukel, R., Meteris, H., Koyuncu, A., Tecer, A., & Yazici, O. (2006). The clinical impact of mood disorder comorbidity on obsessive-compulsive disorder. *European Archives of Psychiatry and Clinical Neuroscience, 256*, 240–245.

Voderholzer, U., Riemann, D., Huwig-Poppe, C., Kuelz, A., Kordon, A., Bruestle, K., et al. (2007). Sleep in obsessive compulsive disorder: polysomnographic studies under baseline conditions and after experimentally induced serotonin deficiency. *European Archives of Psychiatry and Clinical Neuroscience, 257*(3), 173–182.

Walker, E. A., Katon, W., Russo, J., Ciechanowski, P., Newman, E., & Wagner, A. (2003). Health care costs associated with Posttraumatic Stress Disorder symptoms in women. *Archives of General Psychiatry, 60*, 369–374.

Weissberg, M. (2006). Anxiety disorders and sleep. In T. Lee-Chiong (Ed.), *Sleep: A comprehensive handbook* (pp. 845–855). Hoboken, NJ: John Wiley & Sons.

Woodward, S., Friedman, M., & Bliwise, D. (1996). Sleep and depression in combat-related PTSD inpatients. *Biological Psychiatry, 39*, 182–192.

Woodward, S., Murburg, M., & Bliwise, D. (2000). PTSD-related hyperarousal assessed during sleep. *Physiology and Behavior, 70*(1–2), 197–203.

Zayfert, C., & DeViva, J. C. (2004). Residual insomnia following cognitive behavioral therapy for PTSD. *Journal of Traumatic Stress, 17*(1), 69–73.

Chapter 4
Cognitive Behavior Therapy for Insomnia: Treatment Considerations

Abstract Sleep is a process that is heavily influenced by a number of psychological factors including daytime activities and overall schedules. While it may seem intuitively obvious that engaging in such routine practices as allowing oneself to wind down or relax prior to bed or following a routine sleep schedule should benefit sleep, the systematic use of such psychological strategies for insomnia management did not emerge until the latter 1950s. We discuss the rationale and theoretical basis of early strategies (e.g., relaxation training and stimulus control therapy) originally developed for other psychological and behavioral disorders but adapted for use in insomnia. We also describe the rationale, theoretical basis, and treatment components of strategies that evolved out of the field of sleep disorders medicine (e.g., sleep hygiene and sleep restriction therapy). We discuss the development of more omnibus insomnia therapies (e.g., multi-component Cognitive-Behavior Therapy (CBT) for insomnia) that emerged in the mid-1980s and early 1990s. Finally, we review the evidence for CBT as a well-established front-line therapy for management of chronic insomnia in adults (Sleep 29:1398–1414, 2006; Sleep 28:1049–1057, 2005).

> *There is a time for many words, and there is also a time for sleep……(Ecclesiastes 3:7)*
>
> *A well-spent day brings happy sleep…(Leonardo Da Vinci, circa 1500)*
>
> *Early to bed and early to rise makes a man healthy, wealthy, and wise…(Benjamin Franklin, circa 1735)*

What is Cognitive Behavior Therapy and Who Can Benefit from It?

As implied by these few quotations, we have long recognized that our sleep is a process that is heavily influenced by a number of psychological/behavioral factors, including our daytime activities and overall schedules. When our sleep becomes problematic and unpredictable, it then seems reasonable to question whether there

are psychological and behavioral strategies that can be employed to "right the ship" again and reestablish a normal and reliable sleep pattern. While it may seem intuitively obvious that engaging in such routine practices as allowing oneself to wind down or relax prior to bed or following a routine sleep schedule should benefit sleep, the systematic use of such psychological and behavioral strategies for insomnia management did not emerge until the latter 1950s. Between that time and the mid-1980s, a number of psychological and behavioral therapies, described briefly in Table 4.1, were proposed for insomnia management. Some of these, such as relaxation training and stimulus control therapy, were therapies originally developed for other psychological and behavioral disorders, but adapted for the use in insomnia. Others, such as sleep hygiene and sleep restriction therapy, evolved in the then emerging field of sleep disorders medicine and were specifically developed for insomnia management. As indicated in Table 4.1, each of these therapies is

Table 4.1 Common behavioral therapies

Type of treatment	Treatment description
Sleep hygiene	Education about healthy sleep behaviors and sleep-conducive environmental conditions. Treatment focuses on: (1) encouraging daily exercise, (2) reducing/eliminating caffeine, alcohol, and nicotine, (3) eating a light bedtime snack at bedtime, and (4) ensuring that the bedroom is quiet, dark, and comfortable.
Relaxation therapy	Various techniques including progressive muscle relaxation, autogenic training, biofeedback, imagery training, meditation, and hypnosis. This treatment targets sleep-disruptive physiological (e.g., muscle tension) and/or cognitive (e.g., racing thoughts) arousal.
Stimulus control therapy	Based on the assumption that both the timing (bedtime) and sleep setting (bed/bedroom) become conditioned cues for arousal that perpetuate insomnia as a result of their repeated association with unsuccessful sleep attempts. The goal of this treatment is that of re-associating the bed and bedroom with successful sleep attempts.
	Therapy instructions include: (a) go to bed only when sleepy; (b) establish a standard wake-up time; (c) get out of bed whenever awake for long periods; (d) avoid reading, watching TV, eating, worrying and other sleep-incompatible behaviors in the bed/bedroom; and (e) refrain from daytime napping.
Sleep restriction therapy	Sleep restriction therapy reduces nocturnal sleep disturbance primarily by restricting the time allotted for sleep each night so that the time spent in bed closely matches the individual's presumed sleep requirement.
	This treatment typically begins by calculating the individual's average total sleep time (ATST) from a sleep log that is kept for 1–2 weeks.
	An initial time-in-bed (TIB) prescription may either be set at the ATST or at a value equal to the ATST plus an amount of time that is deemed to represent normal nocturnal wakefulness (e.g., ATST + 30 min). The initial TIB prescription is seldom set below 5 h per night.
	On subsequent visits TIB may be adjusted up or down in 15–30 min increments dependent upon sleep performance and waking function.

fashioned to target and address a specific and somewhat distinctive subset of psychological or behavioral factors that is thought to be important in perpetuating insomnia. This observation, in turn, implies that none of these therapies are likely to address all of the psychological and behavioral factors presumed to contribute to chronic insomnia problems.

Given this realization, interest in the development of more omnibus insomnia therapies emerged in the mid-1980s and in the early 1990s. To address the multitude of psychological and behavioral factors presumed to sustain insomnia, a multicomponent Cognitive-Behavior Therapy (CBT) for insomnia emerged. The original renditions of this treatment (Edinger, Hoelscher, Marsh, Lipper, & Ionescu-Pioggia, 1992; Hoelscher & Edinger, 1988; Morin, 1993; Morin, Kowatch, Barry, & Walton, 1993) included a combination of the above-mentioned first generation treatments, including sleep hygiene education, stimulus control instructions, and sleep restriction therapy. These early protocols also acknowledged the sleep-disruptive role of cognitive factors (i.e. unhelpful sleep-related beliefs) by incorporating belief-targeted corrective sleep education or traditional cognitive therapy strategies, such as cognitive restructuring (Beck, Rush, Shaw, & Emery, 1979; Morin, 1993) to address these cognitions. Since the emergence of these early protocols, the combination of stimulus control, sleep restriction therapy, and some form of cognitive therapy have persisted as the core of current-day CBT insomnia treatment. As discussed in detail below, this treatment has been widely tested and shown efficacy for those with chronic insomnia. In fact, CBT is now regarded as a well-established front-line therapy for the management of chronic insomnia in adults (Morin et al., 2006; National Institutes of Health State of the Science Conference Statement, 2005). Evidence supporting the efficacy of this treatment is discussed in some detail later in this chapter. However, before considering the support for such treatments, we first discuss the rationale and theoretical basis for this treatment approach in the ensuing discussion.

Cognitive Behavior Therapy Model

Most individuals experience a night of poor sleep now and then, but chronic or persistent insomnia develops in 10–15% of those in the general population who are the most vulnerable and who are subject to the proper set of sleep-disruptive circumstances. Perhaps the most popular and useful heuristic for understanding the evolution of chronic insomnia is the theoretical model proposed by Spielman et al. (1987). According to the model, the evolution of chronic insomnia is dependent upon the interplay of predisposing factors, precipitating events, and perpetuating mechanisms. Some individuals are presumed at relative risk for developing insomnia due to predisposing vulnerabilities, such as a weakened or highly sensitive biological sleep system or a special propensity to sleep poorly when confronted with stress. Such vulnerabilities alone, however, do not place most such individuals over an insomnia threshold.

Typically, such individuals only experience the onset of insomnia when confronted with precipitating events such as a stressful life event, sudden alteration in their normal sleep-wake schedule, or a major illness. Whereas, insomnia may arise as a transient condition in some of these individuals, others may manifest psychological and behavioral characteristics and reactions to their sleep difficulties that ultimately serve to perpetuate it over time. Thus, although predisposing and precipitating factors lead to the onset of insomnia, the psychological and behavioral perpetuating factors that sustain it serve as the treatment targets for insomnia therapy.

The cognitive behavior model of insomnia posits that an array of sleep-disruptive cognitive factors and behavioral practices act as the key perpetuating mechanisms for sustaining the sleep difficulties of insomnia patients. Figure 4.1 provides a schematic representation of the role and interplay of these factors in perpetuating sleep disturbance. Setting the stage for persistent sleep problems is a thinking style that can include: misattributions about the causes of insomnia, attentional bias for sleep-related threats, worry and/or rumination about the consequences of poor sleep, and unhelpful beliefs about sleep promoting practices (Carney & Edinger, 2006; Carney, Edinger, Manber, Garson, & Segal, 2007; Edinger & Carney, 2008; Espie, 2002; Harvey, 2002; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). These cognitions, in turn, promote sleep-disruptive habits and conditioned emotional
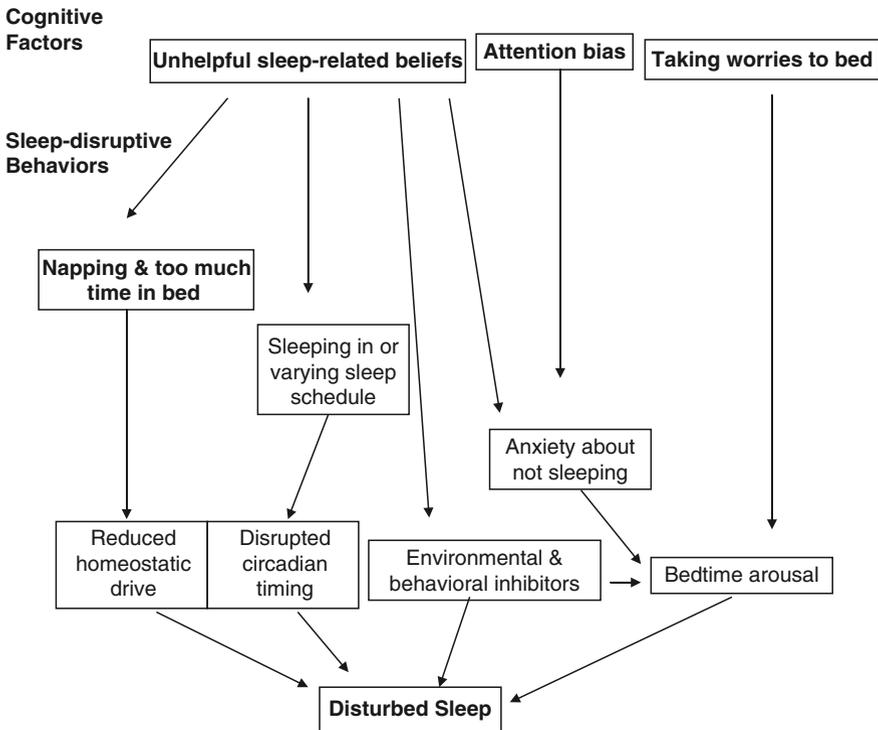

Fig. 4.1 Cognitive behavioral model of factors that perpetuate insomnia

responses that may alter normal sleep drive, interfere with circadian timing mechanisms, or serve as environmental/ behavioral inhibitors to sleep (Bootzin, 1977; Morin, 1993; Spielman, Caruso, & Glovinsky, 1987; Webb, 1988). For example, daytime napping or spending extra time in bed to compensate for a poor night's sleep interferes with homeostatic mechanism of the body that operates automatically to increase sleep drive in response to increasing periods of wakefulness (i.e., sleep debt). Alternately, the habit of remaining in bed well beyond the normal rising time following a poor night's sleep disrupts the body's circadian or "clock" mechanism that controls the timing of sleep and wakefulness within a 24-h period. Additionally, the repeated association of the bed and bedroom with unsuccessful sleep attempts may eventually result in sleep-disruptive conditioned arousal in the home sleeping environment. Finally, failure to discontinue mentally demanding work and allot sufficient "wind-down" time before bed may raise bedtime arousal and serve as a significant sleep inhibitor during the subsequent sleep period. In sum, all of these factors may contribute to and perpetuate PI (Bootzin & Epstein, 2000; Edinger & Wohlgemuth, 1999; Hauri, 2000; Morin, Savard, & Blais, 2000). CBT for insomnia was designed to modify the range of cognitive and behavior factors that ostensibly sustain or add to sleep problems.

The Evidence Supporting CBT for Insomnia

Currently, there is ample evidence supporting the efficacy, effectiveness, and applicability of CBT for a range of insomnia. A variety of studies have shown that CBT is superior to no treatment (wait list conditions) (Mimeault & Morin, 1999; Morin, Kowatch et al., 1993; Rybarczyk, Lopez, Benson, Alsten, & Stepanski, 2002) or such stand-alone therapies such as relaxation training (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001a; Edinger et al., 1992; Rybarczyk et al., 2002), sleep hygiene education (Edinger & Sampson, 2003; Leger, Guilleminault, Bader, Levy, & Paillard, 2002), and a credible sham (placebo) psychological treatment (Edinger et al., 2001a) for the management of uncomplicated, primary forms of insomnia. In addition, several randomized trials (Jacobs, Pace-Schott, Stickgold, & Otto, 2004; Morin, Colecchi, Stone, Sood, & Brink, 1999; Sivertsen et al., 2006; Wu, Jinfeng, Chungai, & Chunling, 2006) collectively have shown that CBT and pharmacotherapy with common prescription hypnotics (e.g., zolpidem, zolpiclone, temazepam) yield similar sleep improvements during active treatment. However, improvements obtained with medications tend to disappear after medications are withdrawn, whereas the improvements obtained during CBT therapy endure long after active therapy is discontinued. Furthermore, large clinical effectiveness studies (Espie et al., 2007; Espie, Inglis, Tessier, & Harvey, 2001) as well as clinic-based case series studies (Morin, Stone, McDonald, & Jones, 1994; Perlis, Sharpe, Smith, Greenblatt, & Giles, 2001) have shown that multimodal CBT is an effective treatment for the management of those with primary insomnia.

More recent studies have provided growing evidence that CBT can be effective for those with more complex forms of insomnia. A number of studies have shown

that CBT leads to sleep improvements in those suffering from insomnia arising from or associated with chronic peripheral pain syndromes (Currie, Wilson, Pontefract, & deLaplante, 2000), breast cancer (Savard, Simard, Ivers, & Morin, 2005), fibromyalgia (Edinger, Wohlgemuth, Krystal, & Rice, 2005), mixed medical conditions (Rybarczyk et al., 2002), and chronic alcohol abuse (Greeff & Conradie, 1998). In addition, case series or clinic-based investigations support the usefulness of CBT among patients with mixed mental and medical conditions (Kuo, Manber, & Loewy, 2001; Morawetz, 2003; Morin, Stone et al., 1994; Perlis et al., 2001). Some of these reports also suggest that CBT may lead to improvements in mood status or other disease-specific symptoms (Edinger et al., 2005; Kuo et al., 2001; Manber, Edinger, San Pedro, & Kuo, 2007; Morawetz, 2003; Savard et al., 2005). These findings coupled with those derived from results obtained in those with primary insomnia indicate that CBT can be regarded as a well-established or front-line therapy for ameliorating sleep disturbance in both uncomplicated and complex forms of persistent insomnia (Morin et al., 2006; National Institutes of Health State of the Science Conference Statement, 2005).

In addition to the data supporting the efficacy/effectiveness of CBT, there are some results of mechanistic studies that suggest that this therapy addresses the cognitive and behavioral factors presumed to perpetuate insomnia. Studies designed to assess therapy effects on cognitive mechanisms have shown that CBT reduces sleep-interfering beliefs (Carney & Edinger, 2006; Espie, Inglis & Harvey, 2001; Morin, Blais, & Savard, 2002), enhances sleep-related self-efficacy (e.g., confidence in one's ability to produce sleep) (Currie et al., 2000; Edinger et al., 2001a; Edinger & Sampson, 2003), and lowers bedtime cognitive arousal (Mitchell, 1978; Mitchell & White, 1977; Sanavio, 1988). Likewise, CBT seemingly reduces sleep-disruptive behaviors such as spending excessive time in bed (Spielman, Saskin, & Thorpy, 1987), or maintaining erratic sleep/wake schedules (Bootzin, 1972; Edinger et al., 2001a; Edinger et al., 1992; Edinger & Sampson, 2003; Espie, Inglis, Tessier, et al., 2001; Monk, Petrie, Hayes, & Kupfer, 1994; Monk, Reynolds III, Buysse, DeGrazia, & Kupfer, 2003; Morin et al., 1999; Morin, Kowatch et al., 1993). Furthermore, mechanistic studies have shown that changes in selected CBT-targeted cognitive (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001b; Morin et al., 2002), and behavioral (Edinger et al., 2001a; Vincent & Lionberg, 2001; Vincent & Hameed, 2003) insomnia perpetuating mechanisms mediate improvements in sleep and global insomnia symptoms. Thus, the improvements achieved with CBT result, at least in part, from the fact that this therapy effectively addresses the cognitive and behavioral mechanisms critical to sustaining insomnia problems.

Treatment Delivery Issues

CBT originally evolved as a treatment that is delivered via individual therapy using four to eight therapy sessions. Moreover, a review of the available efficacy studies showed that this has been the most popular form of treatment delivery used

in these investigations over the past two decades. However, to improve the cost effectiveness of CBT and accessibility, a number of alternative delivery methods have been developed. Not surprisingly, the most common alternative CBT delivery format that has been used thus far is group therapy. Although one meta-analytic review (Morin, Culbert, & Schwartz, 1994) suggested a slight superiority of individually-administered CBT over a group delivery format, several studies have shown that group CBT models involving six to eight sessions produce significant improvements in sleep and global insomnia symptoms (Backhaus, Hohagen, Voderholzer, & Riemann, 2001; Espie et al., 2007; Espie, Inglis, Tessier, et al., 2001; Morin et al., 1999; Morin, Kowatch et al., 1993). Yet, studies directly comparing the relative benefits of individual versus group formats have been extremely limited. Nonetheless, one recent study (Bastien, Morin, Ouellet, Blias, & Bouchard, 2004) did show similar outcomes for insomnia patients assigned to either group or individualized CBT therapy, so this finding in conjunction with the others mentioned suggest that group CBT can and should be considered a viable treatment approach.

Inasmuch as CBT is a psychological treatment, it was originally intended for use by trained and licensed psychologists. However, the number of such individuals who are experts in CBT for insomnia is currently very limited. Moreover, the majority of treatment seeking insomnia patients present in primary care settings (Richardson, 2000) where direct access to psychologist providers is usually limited. Hence, in such settings, the use of nontraditional therapists to deliver this insomnia intervention might be considered. Several studies utilizing nontraditional therapists have found that family physicians (Baillargeon, Demers, & Ladouceur, 1998) and nurses (Epstein & Dirksen, 2007; Espie et al., 2007; Espie, Inglis, Tessier, et al., 2001), rural mental health clinic staff, such as mental health counselors and social workers (McCrae, McGovern, Lukefabr, & Stripling, 2007), and primary care counselors (Morgan, 2003) can effectively administer treatments such as stimulus control and multicomponent CBT in general practice settings. In several of these trials, the therapists received training/supervision from a clinician experienced with CBT for insomnia, so the nontraditional therapists could be considered specialist provider "extenders." This model in which the CBT specialist assumes a trainer/consultant role may represent a reasonable alternative for optimal dissemination when access to the CBT specialist may be limited.

Of course, many people with insomnia may wish to initiate treatment on their own, thus we need to consider the effectiveness of home-based self-help renditions of CBT. In an effort to address this question, Mimeault and Morin (Mimeault & Morin, 1999) tested a booklet of self-help CBT instructions, used independently or with the assisted phone consultations with a therapist. Compared to the wait-list control condition, those treated with the self-help therapy showed greater sleep improvements, and these improvements persisted at a 3-month follow up. Telephone consultations with a therapist conferred some short-term advantage over the self-help booklet, but these benefits disappeared by follow up. Similarly, Currie et al. (Currie, Clark, Hodgins, & El-Guebaly, 2004) compared individual

CBT, treatment with a self-help manual, and a waiting list condition for ameliorating insomnia in those recovering from alcohol addiction. Results showed treated patients achieved significantly greater improvements than controls, but no significant differences were noted between the in-person therapy and home-administered self-help program. It should also be noted that Bastien et al. (Bastien et al., 2004) found comparable effectiveness of CBT provided in individual, group, and telephone formats. Finally, Rybarczyk et al. (Rybarczyk et al., 2005) found a home-based video CBT program superior to no treatment (wait-list condition), but less effective than CBT delivered face-to-face in a classroom setting among older adults with insomnia with comorbid medical conditions. Considered collectively, these findings suggest that self-administered behavioral insomnia treatments are promising although some form of contact with a therapist may be needed to obtain the most optimal results.

There have been a few published attempts to deliver CBT via mass media dissemination. In perhaps the largest and most unique study to date, Oosterhuis and Klip tested an insomnia therapy provided via a series of 8, 15-min educational programs broadcast on radio and television in the Netherlands (Oosterhuis & Klip, 1997). Over 23,000 people ordered the accompanying course material, and data from a random subset of these showed sleep improvements and reductions in hypnotic use, medical visits, and physical complaints were achieved program participants. Unfortunately, the single group nature of their design makes it difficult to discern how these results compare to more conventional treatment. More recently, investigators (Ritterband et al., 2009; Strom, Pettersson, & Andersson, 2004) have begun testing self-help interactive CBT programs delivered via the Internet. The initial venture (Strom et al., 2004) found that those who were treated achieved significantly greater reductions in maladaptive sleep-related beliefs than did those assigned to a wait-list condition, but treatment and control groups otherwise did not differ on study outcome measures, including measures of sleep changes. However, in a later study, Ritterband et al. (Ritterband et al., 2009) found that those who received an internet-delivered CBT appreciated significantly greater improvements in sleep diary measures and in their global insomnia symptoms than did those in a wait-list condition. Although these studies provide a mixed view of internet-based CBT interventions, the promising findings in the latter trial encourage more tests of this form of treatment delivery.

It should be understood that the best-tested and well-established forms of CBT delivery are the individual and group therapy protocols directed by those with extensive training both psychotherapy in general and CBT insomnia therapy specifically. Treatment delivery approaches that diverge from that paradigm through their use of alternative providers or self-help vehicles currently have less empirical support. This does not mean that they are not effective for some insomnia sufferers. However, the bulk of the data supporting CBT efficacy/effectiveness comes from the studies of traditional individual and group delivery methods employed by well-trained CBT therapists. Thus, this form of treatment delivery would seem preferred and recommended when feasible.

References

Backhaus, J., Hohagen, F., Voderholzer, U., & Riemann, D. (2001). Long-term effectiveness of a short-term cognitive-behavioral group treatment for primary insomnia. *European Archives of Psychiatry and Clinical Neuroscience, 251*, 35–41.

Baillargeon, L., Demers, M., & Ladouceur, R. (1998). Stimulus-control: Nonpharmacologic treatment for insomnia. *Canadian Family Physician, 44*, 73–79.

Bastien, C., Morin, C., Ouellet, M., Blias, F., & Bouchard, S. (2004). Cognitive-behavioral therapy for insomnia: comparison of individual therapy, group therapy, and telephone consultations. *Journal of Consulting and Clinical Psychology, 72*(4), 653–659.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression: A treatment manual*. New York: Plenum Press.

Bootzin, R.R. (1972). *Stimulus control treatment for insomnia. Proceedings of the 80th Annual Meeting of the American Psychological Association, 7*, 395–396.

Bootzin, R. (1977). Effects of self-control procedures for insomnia. In R. B. Stuart (Ed.), *Behavioral self-management* (pp. 176–195). New York: Brunner/Mazel.

Bootzin, R. R., & Epstein, D. R. (2000). Stimulus control. In K. L. Lichstein & C. M. Morin (Eds.), *Treatment of late-life insomnia*. Thousand Oaks, CA: Sage.

Carney, C. E., & Edinger, J. D. (2006). Identifying critical dysfunctional beliefs about sleep in primary insomnia. *Sleep, 29*, 325–333.

Carney, C. E., Edinger, J. D., Manber, R., Garson, C. S., & Segal, Z. V. (2007). Beliefs about sleep in disorders characterized by sleep and mood disturbance. *Journal of Psychosomatic Research, 62*(2), 179–188.

Currie, S. R., Clark, S., Hodgins, D. C., & El-Guebaly, N. (2004). Randomized controlled trial of brief cognitive-behavioural interventions for insomnia in recovering alcoholics. *Addiction, 99*(9), 1121–1132.

Currie, S. R., Wilson, K. G., Pontefract, A. J., & deLaplante, L. (2000). Cognitive-behavioral treatment of insomnia secondary to chronic pain. *Journal of Consulting and Clinical Psychology, 68*, 407–416.

Edinger, J. D., & Carney, C. E. (2008). *Overcoming insomnia: A cognitive-behavioral therapy approach-therapist guide*. New York: Oxford University Press, Inc.

Edinger, J. D., Hoelscher, T. J., Marsh, G. R., Lipper, S., & Ionescu-Pioggia, M. (1992). A cognitive-behavioral therapy for sleep-maintenance insomnia in older adults. *Psychology and Aging, 7*, 282–289.

Edinger, J. D., & Sampson, W. S. (2003). A primary care "friendly" cognitive behavioral insomnia therapy. *Sleep, 26*, 177–182.

Edinger, J. D., & Wohlgemuth, W. K. (1999). The significance and management of persistent primary insomnia. *Sleep Medicine Reviews, 3*, 101–118.

Edinger, J. D., Wohlgemuth, W. K., Krystal, A. D., & Rice, J. R. (2005). Behavioral insomnia therapy for Fibromyalgia patients: A randomized clinical trial. *Archives of Internal Medicine, 165*, 2527–2535.

Edinger, J., Wohlgemuth, W., Radtke, R., Marsh, G., & Quillian, R. (2001a). Cognitive behavioral therapy for treatment of chronic primary insomnia: A randomized controlled trial. *The Journal of the American Medical Association, 285*, 1856–1864.

Edinger, J., Wohlgemuth, W., Radtke, R., Marsh, G., & Quillian, R. (2001b). Does cognitive-behavioral therapy alter dysfunctional beliefs about sleep? *Sleep, 24*, 591–599.

Epstein, D. R., & Dirksen, S. R. (2007). Randomized trial of a cognitive-behavioral intervention for insomnia in breast cancer survivors. *Oncology Nursing Forum, 34*(5), E51–E59.

Espie, C. A. (2002). Insomnia: Conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annual Review of Psychology, 53*, 215–243.

Espie, C. A., Inglis, S. J., & Harvey, L. (2001). Predicting clinically significant response to cognitive behavior therapy for chronic insomnia in general medical practice: analysis of outcome data at 12 months posttreatment. *Journal of Consulting and Clinical Psychology, 69*, 58–66.

Espie, C. A., Inglis, S. J., Tessier, S., & Harvey, L. (2001). The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: Implementation and evaluation of a sleep clinic in general medical practice. *Behaviour Research and Therapy, 39*, 45–60.

Espie, C., MacMahon, K., Kelly, H., Broomfield, N., Douglas, N., Engleman, H., et al. (2007). Randomized clinical effectiveness trial of nurse-administered small-group cognitive behavior therapy for persistent insomnia in general practice. *Sleep, 30*(5), 574–584.

Greeff, A., & Conradie, W. (1998). Use of progressive relaxation training for chronic alcoholics with insomnia. *Psychological Reports, 82*(2), 407–412.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40*, 869–893.

Hauri, P. (2000). Primary insomnia. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (3rd ed., pp. 633–639). Philadelphia: W.B. Saunders.

Hoelscher, T., & Edinger, J. D. (1988). Treatment of sleep-maintenance insomnia in older adults: Sleep period reduction, sleep education and modified stimulus control. *Psychology and Aging, 3*, 258–263.

Jacobs, G. D., Pace-Schott, E. F., Stickgold, R., & Otto, M. W. (2004). Cognitive behavior therapy and pharmacotherapy for insomnia: A randomized controlled trial and direct comparison. *Archives of Internal Medicine, 164*, 1888–1896.

Kuo, T., Manber, R., & Loewy, D. (2001). Insomniacs with comorbid depression achieved comparable improvement in a cognitive-behavioral group treatment program as insomniacs without comorbid depression. *Sleep, 14*, A62.

Leger, D., Guilleminault, C., Bader, G., Levy, E., & Paillard, M. (2002). Medical and socio-professional impact of insomnia. *Sleep, 25*, 621–625.

Manber, R., Edinger, J. D., San Pedro, M., & Kuo, T. (2007). Combining escitalopram oxalate (ESCIT) and individual cognitive behavioral therapy for insomnia (CBTI) to improve depression outcome. *Sleep, 20*(Suppl.), A232.

McCrae, C. S., MCGovern, R., Lukefabr, R., & Stripling, A. M. (2007). Research evaluating brief behavioral sleep treatments for rural elderly (RESTORE): A preliminary examination of effectiveness. *American Journal of Geriatric Psychiatry, 15*(11), 9797–9982.

Mimeault, V., & Morin, C. M. (1999). Self-help treatment for insomnia: Bibliotherapy with and without professional guidance. *Journal of Consulting and Clinical Psychology, 67*(4), 511–519.

Mitchell, K. R. (1978). Behavioral treatment of presleep tension and intrusive cognitions in patients with severe predormital insomnia. *Journal of Behavioral Medicine, 2*, 57–69.

Mitchell, K. R., & White, R. G. (1977). Self-management of severe predormital insomnia. *Journal of Behavior Therapy and Experimental Psychology, 8*, 57–63.

Monk, T. H., Petrie, S. R., Hayes, A. J., & Kupfer, D. J. (1994). Regularity of daily life in relation to personality, age, gender, sleep quality and circadian rhythms. *Journal of Sleep Research, 3*, 196–205.

Monk, T. H., Reynolds, C. F., III, Buysse, D. J., DeGrazia, J. M., & Kupfer, D. J. (2003). The relationship between lifestyle regularity and subjective sleep quality. *Chronobiology International, 20*(1), 97–107.

Morawetz, D. (2003). Depression and insomnia: What comes first? *Australian Journal of Counselling Psychology, 3*, 19–24.

Morgan, K. (2003). Daytime activity and risk factors for late life insomnia. *Journal of Sleep Research, 12*, 231.

Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York: Guilford Press.

Morin, C. M., Blais, F., & Savard, J. (2002). Are changes in beliefs and attitudes related to sleep improvements in the treatment of insomnia? *Behaviour Research and Therapy, 40*, 741–752.

Morin, C., Bootzin, R., Buysse, D., Edinger, J., Espie, C., & Lichstein, K. (2006). Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998–2004). *Sleep, 29*(11), 1398–1414.

Morin, C. M., Colecchi, C., Stone, J., Sood, R., & Brink, D. (1999). Behavioral and pharmacological therapies for late-life insomnia: A randomized controlled trial. *The Journal of the American Medical Association, 281*, 991–999.

Morin, C. M., Culbert, J. P., & Schwartz, S. M. (1994). Nonpharmacological interventions for insomnia: A meta-analysis of treatment efficacy. *American Journal of Psychiatry, 151*, 1172–1180.

Morin, C. M., Kowatch, R. A., Barry, T., & Walton, E. (1993). Cognitive-behavior therapy for late-life insomnia. *Journal of Consulting and Clinical Psychology, 61*, 137–147.

Morin, C. M., Savard, J., & Blais, F. C. (2000). Cognitive therapy. In K. L. Lichstein & C. M. Morin (Eds.), *Treatment of late-life insomnia* (pp. 207–230). Thousand Oaks, CA: Sage.

Morin, C. M., Stone, J., McDonald, K., & Jones, S. (1994). Psychological management of insomnia: A clinical replication series with 100 patients. *Behavioral Therapy, 25*, 291–309.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J., & Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychology and Aging, 8*, 463–467.

National Institutes of Health State of the Science Conference Statement. (2005). Manifestations and management of chronic insomnia in adults. *Sleep, 28*, 1049–1057.

Oosterhuis, A., & Klip, E. C. (1997). The treatment of insomnia through mass media, the results of a televised behavioral training programme. *Social Science and Medcine, 45*(8), 1223–1229.

Perlis, M. L., Sharpe, M. C., Smith, M. T., Greenblatt, D. W., & Giles, D. E. (2001). Behavioral treatment of insomnia: Treatment outcome and the relevance of medical and psychiatric morbidity. *Journal of Behavioral Medicine, 24*, 281–296.

Richardson, G. S. (2000). Managing insomnia in the primary care setting: Raising issues. *Sleep, 23*(Suppl. 1), S9–S12.

Ritterband, L. M., Thorndike, F. P., Gonder-Frederick, L. A., Magee, J. C., Bailey, E. T., & Saylor, D. K. (2009). Efficacy of Internet-based behavioral intervention for adults with insomnia. *Archives of General Psychiatry, 66*(7), 692–698.

Rybarczyk, B., Lopez, M., Benson, R., Alsten, C., & Stepanski, E. (2002). Efficacy of two behavioral treatment programs for comorbid geriatric insomnia. *Psychology and Aging, 17*, 288–298.

Rybarczyk, B., Stepanski, E., Fogg, L., Lopez, M., Barry, P., & Davis, A. (2005). A placebo-controlled test of cognitive-behavioral therapy for comorbid insomnia in older adults. *Journal of Consulting and Clinical Psychology, 73*(6), 1164–1174.

Sanavio, E. (1988). Pre-sleep cognitive intrusions and treatment of onset-insomnia. *Behaviour Research and Therapy, 26*(6), 451–459.

Savard, J., Simard, S., Ivers, H., & Morin, C. (2005). Randomized study on the efficacy of cognitive-behavioral therapy for insomnia secondary to breast cancer, part I sleep and psychological effects. *Archives of Internal Medicine, 23*, 6083–6096.

Sivertsen, B., Omvik, S., Palleson, S., Bjorvatn, B., Havik, O. E., Kvale, G., et al. (2006). Cognitive behavioral therapy vs. Zopiclone for treatment of chronic primary insomnia in older adults; a randomized controlled trial. *The Journal of the American Medical Association, 295*, 2851–2858.

Spielman, A. J., Caruso, L. S., & Glovinsky, P. B. (1987). A behavioral perspective on insomnia treatment. *Psychiatric Clinics of North America, 10*, 541–553.

Spielman, A. J., Saskin, P., & Thorpy, M. J. (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep, 10*, 45–55.

Strom, L., Pettersson, R., & Andersson, G. (2004). Internet-based treatment for insomnia: A controlled evaluation. *Journal of Consulting and Clinical Psychology, 72*, 113–120.

Vincent, N. K., & Hameed, H. (2003). Relation between adherence and outcome in the group treatment of insomnia. *Behavioral Sleep Medicine, 1*(3), 125–139.

Vincent, N., & Lionberg, C. (2001). Treatment preference and patient satisfaction in chronic insomnia. *Sleep, 24*(4), 411–417.

Webb, W. (1988). An objective behavioral model of sleep. *Sleep, 11*, 488–496.

Wu, R., Jinfeng, B., Chungai, Z., & Chunling, L. (2006). Comparison of sleep condition and sleep-related psychological activity after cognitive behavior and pharmacological therapy for chronic insomnia. *Psychotherapy and Psychosomatics, 75*, 220–228.

Chapter 5
Medication Considerations

Abstract Despite the general efficacy of CBT for insomnia, this form of therapy is generally not the first intervention chosen for the majority of treatment seeking insomnia sufferers. Most are seen in primary care settings where they usually are treated with some form of hypnotic medication. Many of those who eventually present for a trial of CBT do so while continuing hypnotic medications prescribed for their sleep difficulties. Additionally, many people with insomnia may continue using medications to manage their sleep problems for long periods of time despite less than optimal benefits and their ongoing desires to become medication free. Also, many insomnia sufferers use combinations of sedating anxiolytics and prescription hypnotics in an effort to reduce their sleep-related anxiety and sleep difficulties in general. These observations spawn a number of important questions in regard to the use of CBT with such individuals. First, it is important to consider whether these people derive similar benefits from CBT, as do those who enter treatment medication free. It also seems useful to question if there is an optimal treatment protocol for people who wish to combine CBT with pharmacotherapy for insomnia. Finally, it seems useful to ascertain if CBT and other psychological techniques are useful to those who exhibit hypnotic-dependence and ultimately wish to discontinue their sleep medication use.

Will Cognitive Behavior Insomnia Therapy Work with Those Who Use Sleep Medications?

Despite the general efficacy of CBT for insomnia, this form of therapy is generally not the first intervention chosen for the majority of treatment seeking insomnia sufferers. Most are seen in primary care settings where they usually are treated with some form of hypnotic medication (Richardson, 2000; Walsh & Schweitzer, 1998). In fact, nearly 50% of those consulting for insomnia in medical practice are prescribed a medication for sleep, and the majority of those will continue using sleep medications almost nightly for periods of a year or longer (Ohayon, 1996; Schramm et al., 1993). Moreover, experience indicates that a substantial proportion of those who eventually present for a trial of CBT do so while continuing

hypnotic medications prescribed for their sleep difficulties. The following case vignette exemplifies the common clinical features and treatment challenges presented by such patients:

> Mr. P was a 52-year-old married man complaining of insomnia. He reported a history of sleep difficulties dating back about 14 years to a time when he was having major medical problems. At that time, he was experiencing considerable pain, immobility, and general distress subsequent to undergoing surgery on one of his legs. Shortly after developing his sleep problem, he obtained a prescription for clonazepam to treat his sleep difficulty and general distress. He found this medication helpful and continued using that medication almost nightly since that time. He also subsequently received a prescription for zolpidem, 10 mg, to help his sleep. Thus, when he presented for CBT treatment, he was taking 10 mg of zolpidem along with 1 mg of clonazepam on a nightly basis to help his sleep. Whereas he indicated a desire to discontinue these medications, he noted that he became very anxious and unable to sleep without them. In fact, his previous efforts to stop these medications resulted in experiencing elevated anxiety about sleep and pronounced wakefulness during the night. With his medications, he indicated that he was able to function in the daytime without significant impairment. However, he did indicate that his sleep still was not ideal and he experienced a significant level of fatigue many days each week despite his nightly use medicinal sleep aids. A sleep diary, which he maintained prior to his initial clinic visit, showed difficulty initiating sleep on two nights, and relatively poor quality on several nights. This diary also showed he varied his sleep schedule significantly from day-to-day reportedly in his efforts to compensate for intermittent poor night's sleep.

As suggested by this case, many people with insomnia may continue using medications to manage their sleep problems for long periods of time despite less than optimal benefits and their ongoing desires to become medication free. Also, as shown by this case, many insomnia sufferers use combinations of sedating anxiolytics and prescription hypnotics in an effort to reduce their sleep-related anxiety and sleep difficulties in general. These observations spawn a number of important questions in regard to the use of CBT with such individuals. First, it is important to consider whether these people derive similar benefits from CBT, as do those who enter treatment medication free. It also seems useful to question if there is an optimal treatment protocol for people who wish to combine CBT with pharmacotherapy for insomnia. Finally, it seems useful to ascertain if CBT and other psychological techniques are useful to those who exhibit hypnotic-dependence who ultimately wish to discontinue their sleep medication use.

These questions are of critical importance to the CBT therapist, and for this reason, a substantial proportion of this chapter is devoted to thoroughly addressing them. However, before embarking on this discussion, it seems necessary to provide some background information about prescription hypnotic (sleep) medications as well as about the nonprescription products that are commonly used as sleep aids. Given the focus of this text, it also seems important to consider how various agents commonly prescribed for anxiety disorders might affect the sleep of patients who suffer from insomnia and such comorbid conditions. Hence, the ensuing three sections provide discussions of these topics. This information should lead to a better understanding of CBT implementation with medicated patients.

Prescription Hypnotics: Advantages and Disadvantages

It is estimated that insomnia sufferers spend well over $285 million per year on medications prescribed to aid their sleep (Walsh & Engelhardt, 1999). The most frequently prescribed hypnotic medications are benzodiazepine receptor agonists (BzRAs) (Wagner, Wagner, & Hening, 1998; Walsh & Engelhardt, 1999). These include several benzodiazepines (e.g., temazepam, triazolam, estazolam, quazepam, flurazepam) and newer nonbenzodiazepine agents (e.g., zolpidem, eszopiclone, zaleplon) that act at the same binding site on the GABA-A receptor complex. In addition to these medications, sedating antidepressant drugs such as trazodone (TRZ) and several sedating tricyclic antidepressants (e.g., amitriptyline, doxepin) as well as newer generation antipsychotics (e.g., olanzapine, quetiapine) have been used widely for insomnia treatment (Walsh & Engelhardt, 1999) despite varying evidence for efficacy. Finally, the melatonin agonist ramelteon recently has been approved for insomnia management.

Of the various prescription medications used for insomnia treatment, BzRAs have the greatest amount of efficacy and safety data. Both traditional and newer BzRAs have undergone rigorous premarket safety/efficacy trials and are FDA approved for insomnia management. A meta-analysis (Nowell et al., 1997) of 22 placebo-controlled trials involving traditional BzRAs and zolpidem in patients with primary insomnia showed that these agents produce reliable short-term (median treatment duration = 7 days; range = 4–35 days) improvements of sleep-onset latency, number of awakenings, total sleep time, and sleep quality. Furthermore, the newer BzRAs such as eszopiclone may have continued efficacy and safety for periods of 3–12 months of nightly use (Krystal et al., 2003; Roth, Stubbs, & Walsh, 2005). Like the BzRAs, ramelteon is FDA-approved for treatment of insomnia, but published efficacy and safety data for this medication are currently more limited. Trazodone and most sedating tricyclic antidepressants have very limited empirical support for insomnia management, and they lack FDA approval for such use. Curiously, these agents remain relatively popular and are used widely "off-label" for insomnia treatment (Walsh & Engelhardt, 1999).

Prescription hypnotics and particularly the BzRAs have a number of advantages that support their continued use in primary care and other medical settings. BzRAs are widely available, easy to administer, and they are generally well tolerated by patients. Furthermore, they typically produce rapid sleep improvements usually on the first night they are taken. Given this latter feature, such prescription hypnotics would seem to be the treatments of choice for treating cases of transient or short-term sleep problems. For example, they seem ideal for helping a person sleep in response to stressful life circumstances (death of a loved one), or following an abrupt change in one's sleep–wake schedule as occurs in jet lag. Likewise, they are useful for those who intermittently have difficulty sleeping due to episodic, albeit recurrent, stressful circumstances, such as special assignments at work or before giving a speech or presentation to the public. In fact, sleep medications may be favored over a psychological/insomnia therapy for the management of these various forms of transient insomnia.

However, there are a number disadvantages incumbent in the range of sleep medications that may make them less desirable for long-term insomnia management. Long-acting BzRAs (e.g., flurazepam) may have "hang-over" effects, leading to more motor and cognitive impairment, especially among older age groups (Roehrs, Kribbs, Zorick, & Roth, 1986; Roth & Roehrs, 1991). Long-acting BzRAs also have been blamed for an increased rate of motor vehicle accidents (Hemmelgarn, Suissa, Huang, Boivin, & Pinard, 1997) and hip fractures (Ray, 1992) among older age groups. The shorter acting agents, with rapid onset and offset of effects, are less prone to these sorts of problems, although residual daytime effects may occur with these in some individuals, particularly the elderly, when higher doses are prescribed. More common among the shorter acting agents are such problems as anterograde amnesia (Jonas, Coleman, Sheridan, & Kalinske, 1992; Wysowski & Barash, 1991), which in some individuals may lead to episodes of sleep-related cooking or driving a motor vehicle (US Food and Drug Administration, 2007). Also common to the shorter acting agents are such problems as drug tolerance and the phenomenon of rebound insomnia (Greenblatt, 1992), a dramatic worsening of sleep occurring when the agent is abruptly discontinued or withdrawn. And, as will be discussed in more detail later, all sleep aids are associated with the risk of dependence, particularly psychological dependence that results in persistent difficulty sleeping and increased sleep-related anxiety upon their discontinuation.

Given the noted disadvantage of sleep medications, many healthcare providers have traditionally had some reluctance to prescribe these agents on a long-term basis for insomnia management. This reluctance has been attenuated somewhat by recent studies (Krystal et al., 2003; Roth, Walsh, Krystal, Wessel, & Roehrs, 2005) showing continued safety and efficacy of some of the newer generation hypnotics over extended periods of continuous use. Nonetheless, it should be noted that there are currently no data to demonstrate that people are able to maintain the sleep improvements they obtained with any of the available prescription hypnotics after such medications are discontinued. Moreover, at least one study (Morin, Gaulier, Barry, & Kowatch, 1992) has shown that those with insomnia expect that psychological therapies will produce more positive results with fewer treatment-related side effects than will pharmacotherapy for insomnia. Given these considerations, CBT may be favored over the prescription sleep aids by many of those with chronic sleep difficulties.

Over-the-Counter Medications, Herbal Remedies, and Alternative Treatments

Many, if not most, of those who desire treatment for their insomnia will initiate treatments on their own without seeking medical advice of consultation. Although various self-help books (Edinger & Carney, 2008; Hauri, 1996; Jacobs, 1999; Morin & Wooten, 1996) are available that describe psychological strategies for managing

insomnia, most self-treating insomnia sufferers resort to some form of nonprescription sleep aid. Included among these are over-the-counter compounds specifically marketed as sleep aids, herbal and dietary supplements that are presumed to have sleep benefits or sleep-inducing qualities, and alcoholic beverages. In general, these compounds have, at best, limited data to support their effectiveness for insomnia management and many may result in undesirable side effects or even adverse reactions. Moreover, as is the case with the prescription sleep aids, people may develop a psychological dependence on such agents with their continued use. A detailed summary of the range of publicly available compounds used as sleep aids is beyond the scope of this text. However, the following provides a brief overview of the general categories of substances commonly used as self-help sleep aids.

Antihistamine-Based Sleep Aids

A variety of over-the-counter agents are manufactured and FDA approved specifically as sleep aids. These agents are sold under numerous brand names but all such products marketed specifically for treating insomnia contain diphenhydramine (i.e., benedryl) or doxylamine as their active, sedating compound. Some such products contain one or the other of these compounds as the sole active ingredient whereas some products combine one of these compounds with an analgesic (e.g., aspirin, acetometaphan) and are targeted for patients who have insomnia in the context of ongoing pain. Both diphenhydramine and doxylamine act on the H-1 hystamine receptor and block the effects of histamine, an alerting neurotransmitter found in the central nervous system. As such, ingestion of these compounds leads to subjective drowsiness and sleepiness, thus leading to their use as insomnia therapies. Clinical studies in which doses of 12.5–50 mg of such compounds were used have shown subjective improvements in various sleep measures (Buysse, Germain, Moul, & Nofzinger, 2005; Morin, Beaulieu-Bonneau, LeBlanc, & Savard, 2005). However, a recent study showed that objective sleep recordings failed to corroborate improvements noted on self-reported sleep measures (Morin et al., 2005). Nonetheless, the subjective benefits of these compounds seem sufficient to lead to their current widespread use by the general public.

Although these products are sold without a prescription, they are not without notable side effects. Daytime sedation or "hangover" and impairments of psychomotor performance are commonly reported (Buysse et al., 2005; Meoli et al., 2005). Other reported side effects include dizziness, nausea, depression, malaise, dry mouth, weakness, headaches, tinnitus, gastrointestinal distress, impotence, and voiding problems (Buysse et al., 2005; Meoli et al., 2005). In a minority of users, paradoxical effects including restlessness, anxiety, and increased alertness seem to occur. Impaired cognition is also noted in a large percentage of older hospitalized adults who are given such compounds as sleep aids (Agostini, Leo-Summers, & Inouye, 2001). In general, these compounds are contraindicated in those with

narrow angle glaucoma. Serious, life-threatening side effects are extremely rare but have been described in selected case reports (Buysse et al., 2005).

Herbal Compounds and Dietary Supplements

A variety of herbal compounds and dietary supplements are sold as sleep aids. Included among these are valerian root, kava kava, hops, St. John's Wort, lemon balm, Jamaican dogwood, California poppy, passion flower, and lavender. With the exception of valerian root, data are lacking concerning the safety and efficacy of most of these compounds for insomnia treatment. In the case of valerian root, the results concerning treatment efficacy have been mixed. A recent study showed some subjective benefits of a valerian root/hops combination in the treatment of primary insomnia. However, concurrent objective sleep recordings did not corroborate these subjective benefits. In other trials, valerian root has produced some subjective and objective sleep benefits (Donath, Quispe, & Diefenbach, 2000; Schulz, Stolz, & Müller, 1994). Thus, despite the mixed findings, it seems valerian preparations may produce sleep benefits for some users.

Currently, there are very limited safety data concerning this class of compounds. Side effects associated with valerian generally have been mild and include morning sleepiness, lightheadedness, weakness, and headache (Buysse et al., 2005). In rare cases, hepatoxicity has been associated with the use of valerian and kava kava, whereas there is one report of heart failure and delirium upon the abrupt withdrawal of valerian (Meoli et al., 2005). Currently, data are lacking concerning the safety of most of the herbal compounds and supplements mentioned here so their use as sleep aids cannot be recommended.

Melatonin

Unlike the other compounds mentioned, melatonin is a hormone that is produced by the pineal gland and, thus, occurs naturally in the human body. Melatonin is synthesized from serotonin and mostly is secreted at night. Typically, melatonin concentration levels in the blood begin to rise around dusk, reach a peak during the middle of the night, and then decrease around dawn. Melatonin seems to have influences on the endogenous circadian system that regulates the timing of sleep in the 24-h day. For this reason, melatonin has been used to alter the timing of the sleep–wake schedule under such conditions as jet lag, or to reset the biological clock in those who have marked endogenous delay in the timing of their sleep onset each night. In applications to insomnia, some studies have shown that melatonin results in self-reported improvements in sleep onset latency and general sleep quality, but its effects on other self-reported sleep measures is more equivocal (Buysse et al., 2005). In addition, some studies suggest that melatonin administration leads to objective

improvements in sleep latency (Hughes, Sack, & Lewy, 1998; Zhdanova et al., 2001). However, a recent comprehensive review of the melatonin literature suggested that the research supporting the use of this agent as an insomnia remedy is of questionable quality (Buscemi et al., 2004).

Currently, melatonin sold over-the-counter does not have an FDA-approved indication for insomnia and, therefore, it is not regulated by the FDA. As such, formulations sold to the general public are not standardized in their compositions. However, the side effects associated with melatonin use appear to be minimal. The most common side effect is headache. In rare cases, other side effects including disorientation, nausea, seizures, and shortness of breath have been reported (Buysse et al., 2005). Nonetheless, for most individuals, short-term use to address insomnia is safe, but little data currently exist concerning its long-term effects.

Alcohol

Alcoholic beverages are widely used as a common home remedy for sleep difficulties. In fact, it is estimated that as many as 30% of all chronic insomnia sufferers use alcohol as a routine sleep aid (Ancoli-Israel & Roth, 1999). Alcohol is a CNS depressant and, as such, has a relaxing and sleep-inducing effect, particularly on anxious individuals. It tends to reduce sleep onset time and increase the amount of nonrapid eye movement sleep (NREM) while reducing rapid eye movement sleep (REM) during the initial half of the night (Gillin, Drummond, Clark, & Moore, 2005). However, alcohol is metabolized very rapidly, typically at the rate of one glass of wine or about 8 oz of beer for 1 h. After several drinks, alcohol is fully metabolized by the body about halfway through the night resulting in shallow, broken sleep with increased REM (dreaming) sleep in the latter portion of the night (Gillin et al., 2005). In some individuals, sleep may be disrupted by stomach irritation, a full bladder, rebound wakefulness, sweating, or nightmares. Thus, whereas alcohol often makes it easier for the insomnia sufferer to fall asleep, sleep maintenance and overall sleep quality are usually disrupted resulting in an overall compromise of the total sleep period.

Alcohol has a number of side effects and risks associated with both its short- and longer-term use. In the short run, alcohol tends to increase the likelihood of snoring and apneic (i.e., breathing pauses) episodes even in those without any history of sleep apnea (Dawson, Lehr, Bigby, & Mitler, 1993). If alcohol is used routinely, tolerance usually develops resulting in the need for dose escalation to obtain constant subjective effects. In addition, alcohol is associated with considerable risk for dependence. If dependence does develop, the alcohol user typically reports difficulty sleeping without a drink. With prolonged use of alcohol, daytime hypersomnolence and cognitive dysfunction may be observed. Individuals who develop alcohol dependence often show marked sleep disruption upon becoming abstinent. Moreover, heavy and long-term users often show continued disruption of sleep even 1–2 years after becoming abstinent (Brower, Aldrich, Robinson, Zucker, & Greden,

2001; Gillin et al., 2005). Thus, despite its popularity as a sleep aid, alcohol cannot be recommended to address insomnia.

Can Common Anxiety Medications Cause Insomnia Symptoms?

Many individuals with anxiety disorders receive some form of pharmacotherapy as treatment for their anxiety symptoms. Currently, the two classes of prescription medications that are commonly used to treat anxiety disorders are the serotonin reuptake inhibitors and the benzodiazepines. Included in the former group of compounds are selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine, paroxetine, sertraline, fluvoxamine, citalopram, and escitalopram. Also included in this broad class of medications are the serotonin and norepinephrine reuptake inhibitors (SNRIs) such as venlafaxine. Included in the latter class of medications are such substances as diazepam, lorazepam, and clonazepam. As both of these types of compounds can have effects on sleep or wakefulness, it is useful to consider those effects here.

Whereas SSRIs and SNRIs are often considered the first line pharmacologic agents prescribed for managing anxiety symptoms, these medications can have negative effects on sleep and wakefulness. All of these medications may induce insomnia. In fact, the rate of treatment-induced insomnia ranges from a low of about 5% with escitalopram to as high as 22% with fluoxetine (Schweitzer, 2005). Likewise, these medications may result in daytime sedation with such symptoms occurring in 0–5% of all patients treated with escitalopram and slightly over 20% of those treated with fluoxetine or paroxetine (Schweitzer, 2005). Thus, with patients receiving an SSRI or SNRI report insomnia and/or over sedation in the daytime, the possibility that such symptoms relate to their antianxiety agents should be considered, particularly when such symptoms emerge after treatment initiation.

Benzodiazepines such as diazepam, lorazepam, and clonazepam are commonly prescribed for the management of anxiety disorders. These medications may be administered to manage daytime anxiety symptoms and/or at bedtime if insomnia accompanies the anxiety symptoms. The benzodiazepines mentioned all have such properties as tolerance and dependence with continued use (Greenblatt, 1992). Generally, insomnia does not develop during periods of active use but may develop upon abrupt withdrawal, particularly if such medications are typically taken at bedtime to manage insomnia symptoms. This withdrawal effect is likely to be most pronounced with lorazepam, given its short half-life, but insomnia may develop upon withdrawal of the other medications mentioned as well (Greenblatt, 1991; Sironi, Miserocchi, & De Rui, 1984). Daytime sedation/drowsiness may result from these medications with such effects being more likely and pronounced with the longer acting agents (e.g., clonazepam) as opposed to the shorter acting ones (e.g., lorazepam). Other side effects common to these agents include impaired motor coordination, dizziness, impaired learning, anterograde amnesia, agitation, and depression.

Combining Sleep Medications with CBT for Insomnia

Sleep medications are so widely available and well publicized that many who seek out cognitive behavioral insomnia therapy have either used such medications or at least considered their use. As noted previously, many who begin CBT for their insomnia present on one or a combination of sleep medications, both prescription and over-the-counter preparations. These patients may ultimately want to discontinue their sleep medications entirely but are not ready to do so at the time they enter treatment. Others may not wish to stop using their medications at all but desire additional nonmedicinal intervention for sleep. Usually, a strong psychological dependence on sleep medications has developed and such individuals feel unable to sleep without them (Belleville, Guay, Guay, & Morin, 2007; Lichstein et al., 1999). Such individuals may overvalue the effectiveness of their sleep medications and lack confidence in their ability to sleep without them (Belleville et al., 2007). Moreover, compared to insomnia sufferers without medication dependence, there may be a greater degree of unhelpful beliefs about their insomnia and notable pessimism about their ability to gain control over their sleep (Carney, Edinger, Manber, Garson, & Segal, 2007). Given these observations, it seems reasonable to assume that they could be relatively difficult to treat with strategies such as CBT.

Despite this concern, the data available tend to suggest that such people can and do benefit from CBT for their insomnia problems. In fact, most studies that have examined the relative responses of those who enter treatment on and off hypnotic medications tend to show that hypnotic use does not necessarily dampen CBT treatment response (see Morin et al. 2006 for recent review). Typical of such findings are the data shown in Fig. 5.1. These data derived from a large ($n=127$) case replication series study (Verbeek, Schreuder, & Declerck, 1999) of those treated with CBT showed no difference in the global treatment responses of hypnotic users and nonusers. Similarly, in two large effectiveness studies, Espie (Espie, Inglis, Tessier, & Harvey, 2001; Espie et al., 2007) found that patients who used hypnotics on a chronic basis were no less likely to respond to a nurse-administered CBT insomnia intervention delivered in primary care settings than were those patients who were medication free. Although at least one small ($n=21$) uncontrolled trial (Backhaus, Hohagen, Voderholzer, & Riemann, 2001) suggested that medication-free patients respond better to CBT than do those who are hypnotic dependent, this finding appears contrary to most CBT studies that have conducted post-hoc comparisons of such groups. Thus, given most current evidence, it seems that a history of hypnotic medication use should not be considered as a contraindication or limiting factor when considering people for insomnia treatment with CBT.

A slightly different set of considerations arises when determining whether hypnotic medications should be used in combination with CBT to enhance insomnia treatment outcomes. Rationale for such a treatment combination derives from consideration of the relative advantages and disadvantages of each of these two treatment modalities. Hypnotic medication has the advantage of producing immediate sleep improvements usually on the first night of administration. However, all hypnotic

Subjective Improvement Ratings
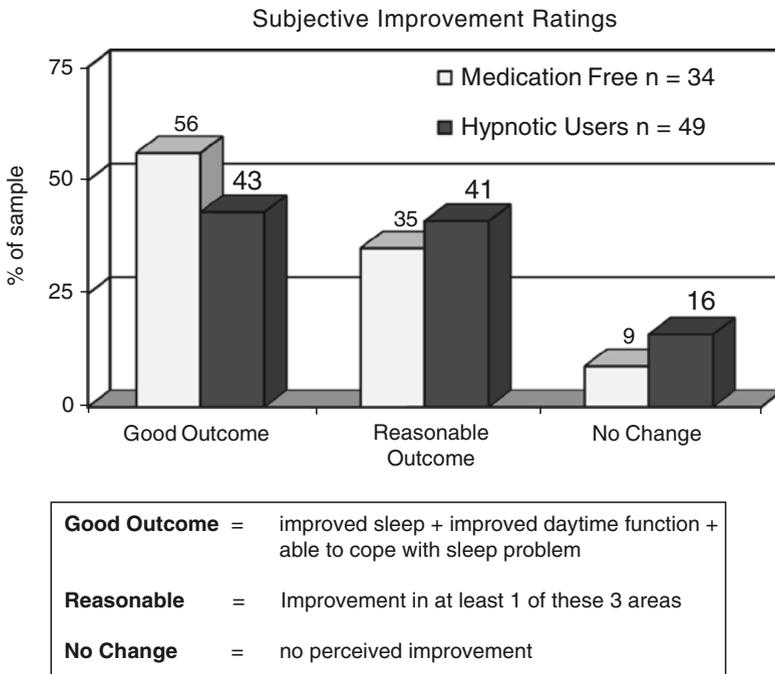
Fig. 5.1 Response of hypnotic users & nonusers to CBT (Verbeek et al., 1999)

agents carry some risk for at least psychological dependence with their long-term use and they have limited data supporting their efficacy either following prolonged hypnotic usage or following treatment discontinuation. In contrast, CBT usually has a slower rate of therapeutic action but has much more durable effects long after active treatment (i.e., therapist contact) is discontinued. Therefore, the combination of hypnotic medication with CBT might result in a more rapid treatment response than seen with CBT alone and more durable treatment effects than when hypnotics are used in isolation. As a consequence, such a treatment combination could prove to be the "ideal" insomnia therapy.

Unfortunately, previous studies pertaining to CBT/hypnotic combination therapy has provided somewhat mixed results. Three studies (Jacobs, Pace-Schott, Stickgold, & Otto, 2004; Morin, Colecchi, Stone, Sood, & Brink, 1999; Wu, Jinfeng, Chungai, & Chunling, 2006) with similar research designs compared treatments consisting of CBT, hypnotic medication, a CBT + hypnotic treatment combination, and a placebo medication. In each of these studies, active treatment was delivered for a fixed period of time (6–8 weeks) and then was discontinued during an extended follow-up period. In each of these studies, those who received CBT alone showed better long-term sleep improvements than did those who received the other treatments including the combined CBT/medication therapy. Such results imply that the presence of medication somehow dampens patients' responses to

CBT perhaps because they rely on the medication effects rather than fully learning and implementing the CBT strategies. However, more recent studies (Morin et al., 2009; Vallieres, Morin, & Guay, 2005) have shown that a sequential treatment protocol, in which patients receive hypnotic medication during the initial stages of an extended CBT protocol, produces better short- and long-term results than does a treatment composed of CBT alone. Seemingly, this treatment combination does not encourage as much dependence on medication and places greater emphasis on CBT for longer-term insomnia improvements. Although more research of this nature is needed, these findings suggest that a time-limited course of hypnotic medication at the outset of CBT therapy may potentiate the treatment effects of this multimodal behavioral intervention.

Sleep Medication Discontinuation Strategies

As noted earlier, many of those with hypnotic-dependence who present for psychological treatment of their insomnia have the immediate or long-term goal of being able to break their dependence on medications for sleep. Those who typically obtain satisfactory sleep on medications wish to maintain such patterns of medications, whereas those sleeping poorly on medications wish to come off of their medications and learn to sleep better without them. Hence, it is important to provide both of these groups with the information and skills included in a psychological insomnia treatment such as CBT so they can establish and maintain a satisfactory sleep pattern free of medications. However, it is also important to provide a medication-tapering program that they can tolerate so as to enhance their chances of achieving a medication-free status. Admittedly, both the psychological and medication tapering aspects of the overall treatment have some features that are unique and specific to those who are hypnotic dependent, so it is useful to consider each of the aspects of treatment separately.

Hypnotic-dependent insomnia sufferers display many of the unhelpful beliefs and sleep-disruptive behaviors (e.g., napping, erratic sleep schedules, too much time in bed, etc.) common to other forms of insomnia. As a result, they benefit from the sleep education, stimulus control, and sleep restriction strategies included in typical CBT protocols. However, these people also present with some specific features that merit special treatment considerations. Clinical observations often show that such individuals have a low sense of self-efficacy in regard to sleep and tend to believe that they simply cannot sleep at night or function in the daytime without their sleep medications. Intermittently, they may try to sleep without their medications to test how they do without medication, but such attempts invariably result in elevated sleep-focused anxiety and arousal that makes sleep more difficult and inadvertently reinforces their unhelpful beliefs about sleep. In working with these individuals, it is useful to discourage such "experiments" at least at the outset of treatment to avoid the undesirable outcomes mentioned. Often, it is also useful to help examine and challenge their beliefs and attitudes about sleep and about their

inability to sleep and cope with poor nights off of medications. For some, this may be achieved through simple discussion, but many people benefit from structured "homework" exercises designed to help them reframe their thoughts about their sleep problems. For example, we (Edinger & Carney, 2008) have suggested using "thought records" (presented more fully in Chap. 8) as a tool towards achieving this end. This instrument helps to identify unhelpful sleep-related thoughts, weigh out evidence for and against these beliefs, and then develop more balanced and constructive modes of thinking to manage their sleep-related distress. Figure 5.2 shows how someone with hypnotic-dependence might complete this instrument to combat unhelpful thinking about discontinuing sleep medication.

Along with these strategies, those exhibiting hypnotic-dependence benefit from structured medication-tapering programs to assist them in striving toward eventual medication abstinence. In implementing any medication-tapering program, it is advisable to enlist the collaboration of a physician to guide the tapering process and to address any adverse effects that may arise. Two approaches that have shown some efficacy when combined with psychological insomnia therapies are those reported by Lichstein et al. (1999) and Morin's group (Belleville et al., 2007; Morin et al., 2004). In the former approach, the patient's usual p.r.n. hypnotic medication dose at the time to treatment entry is first converted into the number of lowest recommended dosage (LRD) as defined by the Physician's Desk Reference (PDR). For example, if the LRD for a particular medication is 5 mg, then the patient would be taking 14 LRDs per week if the nightly dose taken was 10 mg. An individual's sleep medication is then gradually tapered by one nightly dose per week (i.e., 1–2 LRDs per week depending on the starting dose), usually starting with the

Situation	Mood (Intensity 0-100%)	Thoughts	Evidence for the thought	Evidence against the thought	Adaptive/Coping statement	Do you feel any differently?
Sitting in my office after a night without taking sleep medicine	Frustrated (100%)	thinking how sleepy I feel	Last week I fell asleep at my desk several times after night without sleep medicine	I've slept poorly and felt the same way even when I have taken my sleeping pills	I may not be at my best, but the truth is, I end up doing well at work anyway	Frustrated (50%)
		I'm never going to be able to sleep without sleeping pills				
	Worried (80%)					Worried (20%)
		I'm guess I am stuck having to take sleeping pills forever	I'm starting to avoid doing things I used to enjoy	I often feel better once I get myself started and end up enjoying what I choose to do socially	I've noticed there are things I can do to cope with the fatigue, so it is not hopeless	
	Tired (100%)					Tired (85%)
		I can't keep going on like this	I've been taking these pills for two years now	I am learning some new skills that should help me be able to sleep better on my own		
		What's wrong with me?				

Fig. 5.2 Thought Record example for hypnotic-dependence

easiest nights and initially avoiding consecutive nights that are medication free. The approach utilized by Morin's group (Belleville et al., 2007; Morin et al., 2004) includes the following: (a) patients initially establish a medication reduction goal for each week; (b) those using more than one hypnotic first complete a stabilization phase, which requires them to eliminate multiple hypnotics and to use of a single hypnotic only; (c) the initial dosage is reduced by about 25% every 2 weeks until the lowest therapeutic dose is reached; (d) drug-free nights are progressively introduced; and (e) nights with and without hypnotics were planned in advance (i.e., noncontingently).

Previous tests of these approaches have yielded positive results with each. Lichstein et al. (1999) noted that their approach led to an 80% in sleep medication use by the end of the treatment. Patients who underwent this tapering method coupled with relaxation therapy also achieved notable improvements in sleep efficiency and sleep quality as well. In studies by Morin's group (Belleville et al., 2007; Morin et al., 2004), those who received a combined treatment of CBT and the medication-tapering approach described, achieved greater sleep and medication reduction outcomes considered collectively than did comparison groups who received CBT alone or the medication tapering instructions alone. The use of the types of structured medication tapering approaches, described along with a psychological insomnia therapy, appears to be an optimal method for addressing the sleep problems and medication reduction goal of those who are dependent on hypnotic medications.

References

Agostini, J. V., Leo-Summers, L. S., & Inouye, S. K. (2001). Cognitive and other adverse effects of diphenhydramine use in hospitalized older patients. *Archives of Internal Medicine, 161*(17), 2091–2097.

Ancoli-Israel, S., & Roth, T. (1999). Characteristics of insomnia in the United States: Results of the 1991 National Sleep Foundation Survey I. *Sleep, 2*(Suppl.), S347–353.

Backhaus, J., Hohagen, F., Voderholzer, U., & Riemann, D. (2001). Long-term effectiveness of a short-term cognitive-behavioral group treatment for primary insomnia. *European Archives of Psychiatry and Clinical Neuroscience, 251*, 35–41.

Belleville, G., Guay, C., Guay, B., & Morin, C. M. (2007). Hypnotic taper with or without self-help treatment of insomnia: A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 75*(2), 325–335.

Brower, K. J., Aldrich, M., Robinson, E. A. R., Zucker, R. A., & Greden, J. F. (2001). Insomnia, self-medication, and relapse to alcoholism. *The American Journal of Psychiatry, 158*, 399–404.

Buscemi, N., Vandermeer, B., Pandya, R., Hooton, N., Tjosvold, L., Hartling, L., et al. (2004). Melatonin for treatment of sleep disorders. *Evidence Report/Technology Assessment, 108*, 1–7.

Buysse, D. J., Germain, A., Moul, D., & Nofzinger, E. A. (2005). Insomnia. In D. J. Buysse (Ed.), *Sleep disorders and psychiatry* (pp. 29–75). Washington, DC: American Psychiatric Publishing.

Carney, C. E., Edinger, J. D., Manber, R., Garson, C. S., & Segal, Z. V. (2007). Beliefs about sleep in disorders characterized by sleep and mood disturbance. *Journal of Psychosomatic Research, 62*(2), 179–188.

Dawson, A., Lehr, P., Bigby, B. G., & Mitler, M. M. (1993). Effect of bedtime ethanol on total inspiratory resistance and respiratory drive in normal nonsnoring men. *Alcoholism, Clinical and Experimental Research, 17*(2), 256–262.

Donath, F., Quispe, S., & Diefenbach, K. (2000). Critical evaluation of valerian extract on sleep structure and sleep quality. *Pharmacopsychiatry, 33*(2), 47–53.

Edinger, J. D., & Carney, C. E. (2008). *Overcoming insomnia: A cognitive behavior therapy approach therapist guide*. New York: Oxford University Press.

Espie, C. A., Inglis, S. J., Tessier, S., & Harvey, L. (2001). The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: Implementation and evaluation of a sleep clinic in general medical practice. *Behaviour Research and Therapy, 39*, 45–60.

Espie, C. A., MacMahon, K., Kelly, H.-L., Broomfield, N. M., Douglas, N. J., Engleman, H. M., et al. (2007). Randomized clinical effectiveness trial of nurse-administered small-group cognitive behavior therapy for persistent insomnia in general practice. *Sleep, 30*(5), 574–584.

Gillin, J. C., Drummond, S. P. A., Clark, C. P., & Moore, P. (2005). Medication and substance abuse. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 1345–1358). Philadelphia: Elsevier Saunders.

Greenblatt, D. (1991). Benzodiazepine hypnotics: Sorting the pharmacokinetic facts. *Journal of Clinical Psychiatry, 52*(Suppl.), 4–10.

Greenblatt, D. J. (1992). Pharmacology of benzodiazepine hypnotics. *Journal of Clinical Psychiatry, 53*(Suppl.), 7–13.

Hauri, P. (1996). *No more sleepless nights*. Wiley.

Hemmelgarn, B., Suissa, S., Huang, A., Boivin, J. F., & Pinard, G. (1997). Benzodiazepine use and the risk of motor vehicle crash in the elderly. *The Journal of the American Medical Association, 278*, 27–31.

Hughes, R. J., Sack, R. L., & Lewy, A. J. (1998). The role of melatonin and circadian phase in age-related sleep-maintenance insomnia: Assessment in a clinical trial of melatonin replacement. *Sleep, 21*(1), 52–68.

Jacobs, G. D. (1999). *Good night to insomnia*. New York: Owl Books.

Jacobs, G. D., Pace-Schott, E. F., Stickgold, R., & Otto, M. W. (2004). Cognitive behavior therapy and pharmacotherapy for insomnia: A randomized controlled trial and direct comparison. *Archives of Internal Medicine, 164*, 1888–1896.

Jonas, J. M., Coleman, B. S., Sheridan, A. Q., & Kalinske, R. W. (1992). Comparative clinical profiles of triazolam versus other shorter-acting hypnotics. *The Journal of Clinical Psychiatry, 53*, 19–31.

Krystal, A. D., Walsh, J. K., Laska, E., Caron, J., Amato, D. A., Wessel, T. C., et al. (2003). Sustained efficacy of eszopiclone over six months of nightly treatment: Results of a randomized, double-blind, placebo controlled study in adults with chronic insomnia. *Sleep, 26*, 793–799.

Lichstein, K. L., Peterson, B. A., Riedel, B. W., Means, M. K., Epperson, M. T., & Aguillard, R. N. (1999). Relaxation to assist sleep medication withdrawal. *Behavior Modification, 23*, 379–402.

Meoli, A. L., Rosen, C., Kristo, D., Kohrman, M., Gooneratne, N., Aguillard, R. N., et al. (2005). Oral nonprescription treatment for insomnia: An evaluation of products with limited evidence. *Journal of Clinical Sleep Medicine, 1*, 173–187.

Morin, C. M., Bastien, C. H., Guay, B., Radouco-Thomas, M., Leblanc, J., & Vallieres, A. (2004). Randomized clinical trial of supervised tapering and cognitive behavior therapy to facilitate benzodiazepine discontinuation in older adults with chronic insomnia. *American Journal of Psychiatry, 161*, 332–342.

Morin, C. M., Beaulieu-Bonneau, S., LeBlanc, M., & Savard, J. (2005). Self-help treatment for insomnia: A randomized controlled trial. *Sleep, 28*(10), 1319–1327.

Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998–2004). *Sleep, 29*(11), 1398–1414.

Morin, C. M., Colecchi, C., Stone, J., Sood, R., & Brink, D. (1999). Behavioral and pharmacological therapies for late-life insomnia: A randomized controlled trial. *The Journal of the American Medical Association, 281*, 991–999.

Morin, C. M., Gaulier, B., Barry, T., & Kowatch, R. A. (1992). Patients' acceptance of psychological and pharmacological therapies for insomnia. *Sleep, 15*, 302–305.

Morin, C. M., Vallières, A., Guay, B., Ivers, H., Savard, J., & Mérette, C. (2009). Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: A randomized controlled trial. *The Journal of the American Medical Association, 301*(19), 2005–2015.

Morin, C. M., & Wooten, V. (1996). Psychological and pharmacological approaches to treating insomnia: Critical issues in assessing their separate and combined effects. *Clinical Psychology Review, 16*, 521–542.

Nowell, P. D., Mazumdar, S., Buysse, D. J., Dew, M. A., Reynolds, C. F. I., & Kupfer, D. J. (1997). Benzodiazepines and zolpidem for chronic insomnia: A meta-analysis of treatment efficacy. *The Journal of the American Medical Association, 278*, 2170–2177.

Ohayon, M. (1996). Epidemiological study on insomnia in the general population. *Sleep, 3*(Suppl. 19), S7–15.

Ray, W. A. (1992). Psychotropic drugs and injuries among the elderly: A review. *Journal of Clinical Psychopharmacology, 12*(6), 386–396.

Richardson, G. S. (2000). Managing insomnia in the primary care setting: Raising issues. *Sleep, 23*(Suppl. 1), S9–S12.

Roehrs, T., Kribbs, N., Zorick, F., & Roth, T. (1986). Hypnotic residual effects of benzodiazepines with repeated administration. *Sleep, 9*, 309–316.

Roth, T., & Roehrs, T. (1991). A review of the safety profiles of benzodiazepine hypnotics. *Journal of Clinical Psychiatry, 52*, 38–41.

Roth, T., Stubbs, C., & Walsh, J. K. (2005). Ramelteon, a selective MT1/MT2-receptor agonist, reduces latency to persistent sleep in a model of transient insomnia related to a novel sleep environment. *Sleep, 28*, 303–307.

Roth, T., Walsh, J., Krystal, A., Wessel, T., & Roehrs, T. (2005). An evaluation of the efficacy and safety of eszopiclone over 12 months in patients with chronic primary insomnia. *Sleep Medicine, 6*(6), 487–495.

Schramm, E., Hohagen, P., Grasshoff, M., Riemann, D., Hajak, G., Weess, H., et al. (1993). Test-retest reliability and validity of the Structured Interview for Sleep Disorders according to the DSM-III-R. *American Journal of Psychiatry, 150*, 867–872.

Schulz, H., Stolz, C., & Müller, J. (1994). The effect of valerian extract on sleep polygraphy in poor sleepers: A pilot study. *Pharmacopsychiatry, 27*(4), 147–151.

Schweitzer, P. K. (2005). Drugs that disturb sleep and wakefulness. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 499–518). Philadelphia: Elsevier Saunders.

Sironi, V. A., Miserocchi, G., & De Rui, P. L. (1984). Clonazepam withdrawal syndrome. *Acta Neurologica, 6*(2), 134–139.

US Food and Drug Administration. (2007). FDA requests label change for all sleep disorder drug products from http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/2007/ucm108868.htm

Vallieres, A., Morin, C. M., & Guay, B. (2005). Sequential combinations of drug and cognitive behavioral therapy for chronic insomnia: An exploratory study. *Behaviour Research and Therapy, 43*(12), 1611–1630.

Verbeek, I., Schreuder, K., & Declerck, G. (1999). Evaluation of short-term non-pharmacological treatment of insomnia in a clinical setting. *Journal of Psychosomatic Research, 47*, 369–383.

Wagner, J., Wagner, M. L., & Hening, W. A. (1998). Beyond benzodiazepines: Alternative pharmacologic agents for the treatment of insomnia. *The Annals of Pharmacotherapy, 32*(6), 680–691.

Walsh, J. K., & Engelhardt, C. L. (1999). The direct economic costs of insomnia in the United States for 1995. *Sleep, 22*, S386–S393.

Walsh, J. K., & Schweitzer, P. K. (1998). Ten-year trends in the pharmacological treatment of insomnia. *Sleep, 22*, 371–375.

Wu, R., Jinfeng, B., Chungai, Z., & Chunling, L. (2006). Comparison of sleep condition and sleep-related psychological activity after cognitive behavior and pharmacological therapy for chronic insomnia. *Psychotherapy and Psychosomatics, 75*, 220–228.

Wysowski, D., & Barash, D. (1991). Adverse behavioral reactions attributed to triazolam in the Food and Drug Administration's Spontaneous Reporting system. *Archives of Internal Medicine, 151*, 1779–1783.

Zhdanova, I. V., Wurtman, R. J., Regan, M. M., Taylor, J. A., Shi, J. P., & Leclair, O. U. (2001). Melatonin treatment for age-related insomnia. *Journal of Clinical Endocrinology and Metabolism, 86*, 4727–4730.

Chapter 6
Behavioral Strategies for Managing Insomnia

Abstract This chapter is devoted to actual treatment implementation of the core behavioral strategies. In this and subsequent chapters, we provide "nuts and bolts" descriptions to aid the reader in understanding and effectively delivering a number of psychological, evidence-based treatments to those with insomnia and other anxiety-related forms of sleep difficulties. This chapter focuses exclusively on insomnia and provides methods for identifying and correcting common sleep-disruptive habits or practices that are generic characteristics among insomnia sufferers. The discussion begins by demonstrating the use of a sleep diary for identifying the common behavioral treatment targets, namely those common problematic behaviors that serve to perpetuate insomnia. Subsequently, we provide text that can be used to present the treatment rationale in therapy and then consider how to combine and deliver stimulus control and sleep restriction therapy instructions. The chapter concludes by reviewing strategies for addressing other sleep-interfering habits and sleep environments that cause them some sleep disruption. Overall, this chapter is designed to provide step-by-step instructions for implementing effective behavioral insomnia treatment strategies as well as a number of useful paper-and-pencil tools for aiding the clinician in conducting this form of intervention.

Assessing the Sleep Problem Using the Sleep Diary

To demonstrate the value of the sleep diary for insomnia assessment, it is useful to consider some case examples. Figures 6.1–6.3 provide a sampling of the type of information that may be gleaned from sleep diary monitoring. Figure 6.1 shows a common problem among people with insomnia – an erratic sleep schedule. The diary reveals that the time at which these individuals go to bed over the course of a week varies by over 5 h. Additionally, the time at which they get out of bed in the mornings across the week varies over 3 h. When there is an erratic sleep schedule, it can produce symptoms similar to jetlag (e.g., sleep disruption, fatigue, mood disturbance). Remember that 1 h of variability is like traveling one time zone, so 5 h of variability is like traveling across 5 time zones. Those who exhibit these erratic schedules are often motivated by a belief that they should try and obtain the

EXAMPLE

DAY OF THE WEEK	Monday	Mon	Tue	Wed	Thurs	Fri	Sat	Sun
CALENDAR DATE	3/24/08	1/15	1/16	1/17	1/18	1/19	1/20	1/21
1. Yesterday I napped from ___ to ___ (note time of all naps).	2:30-3:15 PM	None	None	None	None	None	None	None
2. Last night I took ___ mg. of ___ or ___ of alcohol as a sleep aid	Ambien 5 mg.	None	None	None	None	None	None	None
3. Last night I got in my bed at ___	11:00 PM	11:00 PM	9:45 PM	10:30 PM	11:30 PM	1:20 AM	11:20 PM	2:45 AM
4. Last night I turned off the lights and attempted to fall asleep at ___	11:30 PM	11:00 PM	10:45 PM	10:30 PM	11:30 PM	12:20AM	11:20 PM	2:45 AM
5. After turning off the lights it took me about ___ minutes to fall sleep.	40 Min.	20 min.	45 min	10 min	65 min	35 min	35 min	10 min
6. I woke from sleep ___ times. (Do not count your final awakening)	2 Times	1	2	2	2	1	1	1
7. My awakenings lasted ___ minutes. (List each awakening separately)	25 Min. 40 Min.	50 min.	25 min 25 min	45 min 90 min	40 min 90 min	55min	55 min	5 min
8. Today I woke up at ___ NOTE this is your final awakening.	6:30 AM	6:05 AM	8:30 AM	8:00 AM	6:40 AM	5:15 AM	5:15 AM	7:25 AM
9. Today I got out of bed for the day at ___.	7:15 AM	6:30 AM	8:40 AM	8:05 AM	7:30 AM	5:20 AM	5:20 AM	7:30 AM
10. I would rate the quality of last night's sleep as: 1 = very poor 4 = good 2 = poor 5 = excellent 3 = fair.	3	3	3	4	3	2	3	2
11. How well rested did you feel upon rising today? 1 = not all rested 4 = rested 2 = slightly rested 5 = well rested 3 = somewhat rested	2	2	2	3	3	2	2	1

Fig. 6.1 Sample sleep diary case 1

EXAMPLE

DAY OF THE WEEK	Monday	Mon	Tue	Wed	Thurs	Fri	Sat	Sun
CALENDAR DATE	3/24/08	3/5	3/6	3/7	3/8	3/9	3/10	3/11
1. Yesterday I napped from ___ to ___ (note time of all naps).	2:30-3:15 PM	None	2:15-3:00 PM	None	3:30 – 3:35 PM	None	None	None
2. Last night I took___ mg. of ___ of alcohol as a sleep aid	Ambien 5 mg.	None	None	None	None	None	None	None
3. Last night I got in my bed at ___	11:00 PM	9:30 PM	10:00 PM	9:00 PM	10:00 PM	9:15 PM	9:00 PM	9:15 PM
4. Last night I turned off the lights and attempted to fall asleep at ___	11:30 PM	11:00 PM	11:15 PM	10:45 PM	11:30 PM	11:00PM	10:50 PM	11:20 PM
5. After turning off the lights it took me about ___ minutes to fall sleep.	40 Min.	30 min.	10 min	30 min	5 min	15 min	15 min	20 min.
6. I woke from sleep ___ times. (Do not count your final awakening)	2 Times	1	2	2	2	2	1	2
7. My awakenings lasted ___ minutes. (List each awakening separately)	25 Min. 40 Min.	30 min	15 min 45 min	15 min 15 min	15 min 10 min	15 min 15 min	15 min	20 min. 20 min
8. Today I woke up at ___. NOTE this is your final awakening.	6:30 AM	6:00 AM	5:45 AM	5:45 AM	6:00 AM	5:45 AM	5:50 AM	6:00 AM
9. Today I got out of bed for the day at ___	7:15 AM	6:30 AM	6:30 AM	6:30 AM	7:00 AM	6:15 AM	6:30 AM	6:30 AM
10. I would rate the quality of last night's sleep as: 1 = very poor 2 = poor 3 = fair. 4 = good 5 = excellent	3	3	2	3	4	3	3	3
11. How well rested did you feel upon rising today? 1 = not all rested 2 = slightly rested 3 = somewhat rested 4 = rested 5 = well rested	2	2	2	2	3	2	2	2

Fig. 6.2 Sample sleep diary case 2

EXAMPLE

DAY OF THE WEEK	Monday	Mon.	Tue.	Wed	Thurs	Fri	Sat	Sun
CALENDAR DATE	3/24/08	4/1	4/2	4/3	4/4	4/5	4/6	4/7
1. Yesterday I napped from ___ to ___ (note time of all naps).	2:30-3:15 PM	None	2:00-4:00 PM	None	None	5:00-6:30 PM	None	None
2. Last night I took___ mg. of ___ of alcohol as a sleep aid	Ambien 5 mg.	None	None	1 glass wine	1 beer	2 beers	None	None
3. Last night I got in my bed at ___	11:00 PM	11:30 PM	12:30 PM	11:00 PM	12:00 PM	11:30PM	2:45 AM	2:30 AM
4. Last night I turned off the lights and attempted to fall asleep at	11:30 PM	11:30 PM	12:30 PM	11:00 PM	12:00 PM	11:30PM	2:45 AM	2:30 AM
5. After turning off the lights it took me about ___ minutes to fall sleep.	40 min.	3 hrs	2 hrs	3.5 hrs	2.5 hrs	3.5 hrs	30 min	25 min
6. I woke from sleep ___ times. (Do not count your final awakening)	2 Times	1	2	1	1	2	1	1
7. My awakenings lasted ___ minutes. (List each awakening separately)	25 min. 40 min.	20 min	25 min 25 min	10 min.	20 min	40 min 30 min	5 min	10 min
8. Today I woke up at ___ NOTE this is your final awakening.	6:30 AM	8:30 AM	9:30 AM	8:05 AM	8:40 AM	9:00 AM	11:25 AM	10:30 AM
9. Today I got out of bed for the day at ___	7:15 AM	8:40 AM	9:40 AM	8:30 AM	8:45 AM	9:05 AM	11:30 AM	10:40 AM
10. I would rate the quality of last night's sleep as: 1 = very poor 4 = good 2 = poor 5 = excellent 3 = fair.	3	2	3	3	1	3	5	4
11. How well rested did you feel upon rising today? 1 = not all rested 4 = rested 2 = slightly rested 5 = well rested 3 = somewhat rested	2	1	3	2	1	3	4	5

Fig. 6.3 Sample sleep diary case 3

maximum amount of sleep possible (for more details on the effect of beliefs on sleep-interfering behaviors, see Chap. 7). This practice of "chasing" sleep, regardless of the timing of sleep, perpetuates the sleep difficulty. As will be described in many places in this book, sleep quality has more to do with timing and sleep stage architecture than quantity. This erratic sleep "schedule" and the beliefs that drive it (e.g., anxiety about getting "enough" sleep) are primary treatment targets for those who present such diary data.

Figure 6.2 shows diary results for an individual who manifests another common practice seen frequently among people with insomnia. These data show a pattern of retiring to bed well in advance of the actual time chosen for attempting to fall asleep. This can be discerned by examining the discrepancy between the time the individual got into bed (item #3 on Fig. 6.2) and the time he/she turned out the lights to attempt to sleep (item #4 on Fig. 6.2). Follow-up queries of a pattern such as this one commonly reveal practices such as watching television or reading in bed for an hour or more before intending to fall asleep. Those who engage in such practices can routinely spend 9 or more hours in bed per night and experience extended awakenings each night as a result. Indeed, it is difficult for most adults to produce 9 h of quality sleep. Early bedtimes, a prolonged amount of time in bed, and accompanying delayed sleep onset latencies require careful follow-up. Those who produce such a pattern often exhibit a tendency to doze off while watching TV or reading in bed before their intended "lights-out" period indicated on the diary. The extra time spent in bed, using the bed for activities other than sleep, occasional napping, and the unrecorded dozing are important behavioral treatment targets uncovered by sleep diary monitoring and related questioning. This pattern is often related to unhelpful and in some cases inaccurate beliefs about sleep needs and what factors promote good quality sleep. Such beliefs will also be targeted during treatment (see Chaps. 7 and 8 for further discussion).

Presented in Fig. 6.3 are some data that demonstrate the importance of the sleep diary during the assessment and treatment planning phase. These data are from a young adult who complained about difficulty falling asleep each night. Throughout the week of monitoring, this person spent 2.5–3.5 h awake before falling asleep. Careful query revealed that unlike the previous case example, this person did not report any dozing during this period. There were several instances wherein alcohol was used as a sleep aid. If you calculate when this person falls asleep (by adding the sleep onset latency to the lights out time), you see that this person tends to fall asleep within the same hour each night (between 2:30 and 3:30 A.M.). Interestingly, on weekends, this person goes to bed during this 2:30–3:30 window. When this person goes to bed close to the time that they normally fall asleep, the sleep onset latency is much shorter and actually falls within normal limits (≤30 min). Also of note is that the weekend rise times are much later than the rise times during the week and the sleep quality and restedness ratings improve on the weekends too. The hypothesis formed at this point may be that the weekend permits a more optimal (i.e., later) sleep schedule for this person, thus allowing them to obtain a full night's sleep. This picture is consistent with delayed sleep phase syndrome, a circadian rhythm disorder in which the person is biologically disposed to fall asleep

later than most people and to rise much later than most people. In such people, the constraints of a work or school schedule can interfere with the natural body clock's schedule; thus, sleep-depriving them during the week. The weekend offers a small respite by matching their schedule to their body clock, but then the person returns to the problematic schedule on Monday. We do not discuss circadian rhythm disorder treatments in this book, so when encountering this type of pattern, it is best to refer to a sleep specialist.

Setting the Stage for Treatment Recommendations: The Role of Psychoeducation

As the preceding discussion demonstrates, those with insomnia engage in many practices that sustain and/or exacerbate their sleep difficulties. As a result, most benefit by making marked changes in their sleep habits and their general approach toward sleep in general. However, it should be recognized that many of the attitudes and behaviors they "bring with them" to treatment are logical and sensible. Moreover, many of their sleep disruptive habits represent reasonable attempts to cope with or compensate for sleep difficulties. Despite the ineffectiveness of their approach to sleep, they may still be reluctant to make the types of changes required of them by behavioral insomnia treatment, particularly in the absence of a convincing rationale supporting the need for such changes. Indeed, it is not reasonable to expect a high degree of adherence to treatment recommendations that call for substantial changes in sleep habits unless they are provided an adequate and convincing treatment rationale. Therefore, presenting such rationale in the form of psychoeducation has become a mainstay in the provision of CBT for insomnia.

The educational information provided prior to introducing the specific treatment recommendations discussed later in this chapter has two primary functions. First, it helps people overcome their unhelpful, anxiety-provoking beliefs about sleep so that they may develop more realistic sleep expectations. Secondly, it enables insomnia sufferers to better understand the rationale for the recommendations in this treatment. This understanding, in turn, increases the likelihood of adherence to treatment recommendations. Generally, we have found it most helpful to consider with clients what their actual sleep needs might be since many of them have poorly conceived notions about their sleep requirements. In addition, most people seem to benefit from some basic education about how the human sleep system works.

We tell people that we will recommend many changes to the way they currently approach sleep in order to improve sleep. To prepare for these changes, we say that it is often helpful to understand why these changes are necessary, and thus we discuss: what their current sleep needs are and what controls the quantity and quality of sleep; in short, we hope that understanding how sleep works will help them make the changes necessary to obtain better sleep. The information below exemplifies the type of information we use in psychoeducation to set the stage for behavioral and cognitive change.

The information can be paraphrased according to the preferences of the therapist and needs of the specific patient.

The burning question for many is, "How much sleep do I need each night?" Many people believe that they already know the answer, but research tells us that these beliefs are often inaccurate (Carney & Edinger, 2006; Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). We like to answer this question with an analogy about shoe size; that is, just as there is no single "correct" shoe size that fits everyone, there is no single amount of sleep that is required of everyone either. Sleep needs vary across individuals and across time and circumstance. On average, most people need between 6 and 9 h of sleep per night, but a proportion of people need less or more. The purpose of this piece of psychoeducation is that it is important for people to set aside preconceived or rigid ideas about how much sleep they need, as their body often has other ideas. We reassure them that we will let their body tell us how much sleep it needs and we will follow its lead; doing so will improve the quality of sleep.

Most people including nonsleep specialists often have little idea about how the body's sleep system works. The sleep system can be easily explained by focusing on three main factors or sets of circumstance that have important and controlling effects on the ability to sleep when making a sleep attempt. The first of these is the amount of time spent awake before the attempt to sleep again. There is a simple principle that should be understood about the effect that staying awake has on sleep. The longer someone is awake between their last sleep episode and the current attempt to sleep, the higher the sleep drive and the more likely it becomes that they will be able to fall asleep quickly and sleep for an extended period. In contrast, if the same person awakens from an extended period of sleep and shortly thereafter tries to sleep again, it is unlikely they will be very successful because they have not built up enough sleep debt to warrant more sleep. Therefore, it is important that people stay awake long enough each day to ensure the build-up of adequate sleep drive to initiate and sustain sleep through the night.

The second regulatory factor in the sleep system is the body clock. Sleep is best attempted during a window in the 24 h day that matches the internal clock. Not only is the circadian clock tied to the sleep-wake system, but it also affects mood, cognition, and a multitude of bodily systems. As the term circadian implies ("about a day"), the body clock operates on a roughly 24-h cycle and produces predictable 24-h variations in such things as digestion, body temperature, and the sleep/wake pattern. Throughout the day, the body clock controls alertness levels so as to keep people awake and functioning effectively. However, during the night, the body clock turns off the alerting signals and allows us to fall asleep and stay asleep through the night. In the morning, the clock again turns on the alerting signal causing us to awaken for the day. It is important to recognize that the body clock works at its best, when adhering to a fairly regular sleep/wake schedule. When schedules (rise times and bedtimes) are erratic, the sleep period fall in-and-out of sync with the body clock and sleep becomes disrupted. In a sense, following a highly variable sleep/wake schedule is like making the body clock "shoot at a moving target," and under such circumstances it works very poorly. Hence, it is important to follow a

fairly routine sleep/wake schedule so that attempts to sleep fall in unison with the body clock's sleep/wake rhythm.

The last factor we discuss is the importance of protecting the nighttime sleep period from physical and psychological intrusions. We tell patients: "If you take your worries to bed or if you do not address physical disturbances such as noise, phone calls at night, or too much light in the bedroom environment, your sleep can be disturbed as well, even if have a regular sleep schedule and you have built up sufficient sleep drive by the time you enter bed." Therefore, some additional measures are necessary to eliminate any psychological or physical causes of sleep difficulties. After providing these explanations of the three factors that regulate sleep, we spend time asking if they understand the information and whether they have any questions or concerns.

Presenting Behavioral Insomnia Treatment Recommendations

As noted previously, people with insomnia usually present with a number of well-established sleep disruptive habits that serve to perpetuate their sleep difficulties. Many such behaviors reflect ineffectual attempts to compensate for ongoing sleep difficulties. For example, daytime napping to make up for poor nights or spending an excessive amount of time in bed in an effort to provide an adequate opportunity to get the sleep they feel they need. Because sleep becomes elusive and unpredictable, many insomnia sufferers assume that they should sleep whenever they are able and, thus, end up with a highly erratic sleep-wake schedule. When finding themselves awake in bed, many insomnia sufferers engage in behaviors such as tossing and turning, watching the clock, or just lying in bed thinking how poorly they will feel the next day because of the inadequate sleep they are getting. For some, sleep difficulty arises due to a practice of taking worries to bed or engaging in stimulating mental (e.g., studying for an exam) or physical (e.g., physical exercise) activity too close to bedtime. Not uncommonly, people with insomnia present with a multitude of these behavioral targets that need to be addressed for effective insomnia management.

Various behavioral insomnia treatment strategies are available for addressing one or more of the practices mentioned, but a combination of stimulus control (Bootzin, 1972) and sleep restriction (Spielman, Saskin, & Thorpy, 1987) instructions provides an omnibus treatment for these problematic behaviors. This combination of treatment recommendations, standardizes the sleep/wake schedule, eliminates sleep incompatible behaviors occurring in the bed and bedroom, and restricts time in bed (TIB) to produce a consolidated sleep pattern. Most recommendations included in this regimen are standard for everyone. However, the TIB prescriptions provided are derived from the sleep diary completed during a 2-week period prior to instituting active treatment. Accordingly, TIB prescriptions may vary across people, so these prescriptions allow for some tailoring of treatment to fit each person's specific sleep needs. Nonetheless, it is important to have people

complete sleep diaries for a period of time, preferably 2 weeks before beginning treatment (Wohlgemuth, 1999), so the diary data can be available to ascertain the TIB prescription.

For most people, we present the combined stimulus control/sleep restriction treatment recommendations in a standard fashion. The following italicized text exemplifies how these recommendations are typically presented. Subsequently, we provide text that may be used verbatim or paraphrased depending on therapists' preferences and patients' needs.

> In order to overcome your insomnia and improve your sleep pattern, you should first select a standard rising time and stick to that time each regardless of how well or poorly you sleep on any specific night. Changes in your sleep-wake schedule can disturb your sleep and contribute to your insomnia problem. When you vary your sleep schedule, you create the type of sleep problem that occurs in jetlag. In a sense, varying your sleep schedule causes you to sleep in and out of sync with your body clock, and this produces a very unreliable and unpredictable sleep pattern. The best way for you and your body clock to work together is for you to stick to a standard rising time each and every day. This routine rising time will send a strong and consistent signal to your body clock and, in turn, you body clock will begin working with you to produce a very strong and consistent sleep-wake rhythm.

> Secondly, you should use the bed solely for sleep and sexual activity. You should not read, watch T.V., eat, study, use the phone, or do other things that require you to be awake while you are in bed. If you routinely use your bed for these sorts of activities, you are unintentionally training yourself to be awake in bed. If you avoid these activities while in bed, your bed will eventually become a place where it is easy to go to sleep and stay asleep. In fact, you may eventually find that you become even sleepier when you retire to bed and consequently fall asleep easily.

> Thirdly, it is important that your bed remains a very pleasant place for you to be. It should not be a place that you dread going or where you agonize about your sleep. To avoid these feelings, you should never stay in bed, either at the beginning of the night or during the middle of the night, for long periods without being asleep. Lying awake in bed for long periods of time usually leads to tossing and turning, becoming frustrated, or worrying about not sleeping. Having these understandable reactions actually make it more difficult to get to asleep. If you cannot fall asleep or return to sleep quickly (within 15–20 minutes), or you do not feel as though you are about to go to sleep, it is preferable to get up and leave the bedroom. While in another room, try and find something relaxing (but not activating) to do for a while. It is best if you do what you do in a seated position and that you avoid activities that are stimulating or that hold your attention. Often activities such as light, recreational reading, watching TV, or listening to soft music are helpful. Only return to bed when you feel sleepy enough to fall asleep quickly.

> Next, it is important that you avoid worrying, contemplating problems, planning future events, or engaging in other thinking while you are in bed. These activities can unintentionally become mental habits, and the best way to break a habit is to interrupt it when it occurs. So, if you cannot turn off your thoughts, it is best to get up and go to another room and stay up until your mind is clear. While you are up, it is a good idea to engage in the types of relaxing activities we just discussed. If you find your disruptive thinking occurs frequently, you may find it helpful to routinely set aside a time early each evening to do the thinking, problem-solving, and planning you need to do. When done on a consistent basis, this practice should result in fewer of these thoughts following you to bed.

It is also important that you eliminate all daytime napping. Naps count toward your 24-hour sleep requirement. If you take a 2-hour afternoon nap, you are likely to sleep much more poorly that following night. Therefore, if you want satisfying sleep at night, you should avoid napping.

Finally, you should go to bed when you feel sleepy but not so early that you find yourself spending much more time in bed each night than you need for sleep. Spending much more time in bed at night is like buying yourself a shoe that is much bigger than you need. If for example you bought a shoe that was two sizes larger than you require, your foot would not grow to fill the shoe but would, rather, flop around inside it. Likewise, if you spend far more time in bed than your body needs for sleep, your sleep needs will not increase to fill the extra time you are spending in bed. Instead, you will find that this practice results in you spending a lot of time awake in bed. If you spend too much time in bed, you may actually make your sleep problem worse. The amount of time you spend in bed each night should "fit" you like your shoe fits. That is, you should be in bed only a little bit longer than you need to satisfy your sleep requirement. We will use your sleep diary to decide the amount of time to spend in bed and what times you should go to bed at night and get out of bed in the morning.

At this juncture, it is necessary to consult the pretreatment sleep diaries to determine the optimal starting time-in-bed allotment. To demonstrate how this is done, the diary data shown for the 1-week period in Fig. 6.3 will be used. As can be determined from simple mathematical calculations, this diary shows an average time in bed slightly over 9 h (i.e., calculate from the time they get into bed in item 3 to the time they got out of bed in item 9), but they only spend an average of 6 h sleeping (calculated by subtracting the time spent awake in bed from the time spent in bed). Different approaches have been suggested for implementing sleep restriction instructions with such data. A common approach (Morin, 1993) is that of restricting time in bed to whatever the average amount of total sleep time is from the pretreatment sleep diary. In this case, that amount of time would be 6 h. However, this approach is somewhat austere and does not take into account the additional amounts of time people normally are awake in bed at the beginning of the night and in the middle of the night. It also should be noted that insomnia sufferers, as a group, tend to underestimate the time they sleep at night (Edinger & Fins, 1995; Means, Edinger, Glenn, & Fins, 2003). Given these considerations, limiting the time spent in bed to the average total amount of sleep produced on the pretherapy sleep diary may actually result in partial sleep deprivation. For these reasons, we (Edinger & Carney, 2008) have suggested setting the initial time in bed (TIB) allotment at the average total sleep time observed from the diary plus an additional 30 min to accommodate normal wake times in bed and the proneness many people have to underestimate their time slept. Thus, using the diary data shown in Fig. 6.3 we would allocate 6.5 h as the initial TIB allotment.

Once the initial TIB prescription is determined, it is necessary to help the person choose a standard wake-up time and earliest bedtime that "fit" with the TIB prescription. In doing so, it is important to encourage people to think about the most acceptable bed and rising times. An individual may initially decide that 8:00 A.M. is a desirable rising time. That choice may initially seem reasonable; however, if the initial TIB prescription is 6 h, this wake-up time would result in an

earliest bedtime of 2:00 A.M. Upon discovering this fact, this person may opt to select an earlier rising time so that the bedtime can also be earlier the preceding night. In addition, it is helpful to select a time that is at least as early as the earliest time they have to rise for work or other responsibilities. For example, if three mornings during the week involve an earlier (7 A.M.) rise time, this person should consider setting the rise time at 7 A.M. to avoid the jetlag-type of problems we discussed earlier. It is important to stress that the bedtime is the *earliest* time at which they can go to bed, but if they are not sleepy, they should still follow the recommendation above which is to stay out of bed until sleepy. This may result in some restriction of sleep in the beginnings of treatment, but this will also increase sleep drive for subsequent nights. Whatever rise times and bed times are chosen, it is essential to work collaboratively to arrive at a schedule. Adherence to the TIB prescription will be enhanced when the person takes an active role in selecting the initial bed and rising times to employ.

Whereas it is useful to discuss the stimulus control and sleep restrictions verbally during the initial therapy visit, a written summary of the treatment recommendations helps with remembering the instructions. The instruction sheet shown in Fig. 6.4 includes the essential points to remember and can be given as a "take-home" reminder of the protocol to follow. This particular instruction sheet lists the combined stimulus control and sleep restriction guidelines to follow, and includes an area where the specific agreed upon bed and rising times can be entered.

Addressing Sleep Hygiene Issues

While the above treatment recommendations form the core of effective treatment, it is important to address any other factors that disrupt sleep and/or impair daytime functioning. The generic name for such a recommendation is sleep hygiene. Sleep hygiene is not an effective standalone treatment (Morin et al., 1999); however, it is commonly included in CBT to address any sleep-interfering behaviors such as caffeine overuse or sleeping environments that are not conducive to sleep. Most people have heard about sleep hygiene and readily accept these recommendations (Vincent & Lionberg, 2001). However, people may also assert that sleep hygiene recommendations have not improved their insomnia in the past, so it may be useful to explain that while instituting sleep hygiene recommendations alone may not be enough to improve their insomnia, changes in this area may be necessary to derive maximal benefit from the treatment. That is, while stimulus control and restricting time in bed may produce considerable improvements, full improvement may be impeded by excessive caffeine use in the latter part of the day. The exact content of sleep hygiene instructions may vary somewhat depending upon the needs of the particular person being treated. However, the following text provides the treatment rationale and instructional set that is useful with most people. This text can be used verbatim or paraphrased depending upon therapists' preferences and patients' needs.

TREATMENT GUIDELINES

1   Select a standard wake-up time. Use it every day regardless of the sleep you obtain on any particular night.

2   Use the bed only for sleeping. Do not read, eat, watch TV, etc. in bed. Sexual activity is the only exception.

3   Get up when you can't sleep. When you are unable to sleep, get up and go to another room until you feel sleepy enough to fall asleep quickly before returning to bed. Get up again if sleep does not come on quickly.

4   Don't worry, plan, etc., in bed. If such mental activities come on automatically in bed, get up and stay up until you can return to bed without these mental activities interfering with your sleep.

5 Avoid daytime napping. Napping, particularly in the late afternoon or early evening may interfere with your night's sleep.

6 Go to bed when you are sleepy, but not before the time suggested. Long periods of time in bed will lead to shallow, fragmented sleep. You should spend only the amount of time in bed that you actually need for sleep.   Adherence to the bedtime and wake time suggested for you below should help you overcome your persistent sleep problem.

Fig. 6.4 Behavioral treatment instructions handout

As a first step, you should limit your intake of caffeine. Foods and beverages that contain caffeine are items such as coffee, tea, soft drinks with added caffeine or chocolates. Caffeine is a powerful stimulant that stays in your system for several hours after you consume it, and it may make it harder for you to sleep well at night. Therefore, you should limit your caffeine to the equivalent of no more than 3 cups of coffee per day and avoid caffeine in the late afternoon or evening hours.

It is also important that you limit your use of alcohol. While alcoholic beverages consumed close to bedtime may make you drowsy and fall asleep more easily initially, as alcohol is broken down in your body, it produces effects that cause sleep to be less continuous and

therefore less restorative. The cost of alcohol consumption at least cancels, and in some cases outweighs the apparent sleep-initiating benefits. As a result, it is better to avoid consuming much alcohol in the evening, and alcohol should not be used as a sleep aid.

Regular exercise can be beneficial to your sleep. Try some regular moderate exercise such as walking, swimming, or bike riding. Moderate exercise or activities occurring in the late afternoon or early evening may contribute to deeper sleep at night. Moreover, improving your fitness level, regardless of when you exercise can improve the quality of your sleep. However, since exercise does provide a temporary boost in alertness, it is not a good idea to exercise right before bed because it may make it harder to get to sleep quickly.

Too much mental or physical stimulation just prior to bedtime is usually disruptive. Most people find it difficult to perform physical or mental work right up to bedtime and then fall asleep quickly. So you should protect the hour or two before your usual bedtime as a time to relax and wind-down before you enter bed. If you make this a regular habit, you will find it is easier to shut off the demands of the day and get to sleep more easily.

Your sleep setting has an important influence on your sleep. Make sure that your bedroom is quiet and dark. Noise and even dim light may interrupt or shorten your sleep. You can block out disturbing noise by wearing earplugs, running a fan, or using a "white noise" machine that is specifically designed to screen sleep-disruptive sounds. Also, when possible, eliminate the use of nightlights and consider using dark shades in your bedroom so that light does not awaken you prematurely in the morning. Make sure that the temperature in your bedroom is comfortable. Generally speaking, excessively warm temperatures (above 75 degrees Fahrenheit) can disrupt sleep. Consider using an air conditioner during hot weather.

As is the case for the combined stimulus control/sleep restriction regimen, it is useful to provide written instructions to reinforce these recommendations. Hence, the list of sleep hygiene recommendations presented in Fig. 6.5 is provided as an additional take-home handout that can be provided to help people remember and adhere to these suggestions.

The Value of Follow-Up Sessions

Usually, the combined stimulus control/sleep restriction regimen and the sleep hygiene instructions can be provided in the context of the first visit. However, a series of follow-up sessions are typically beneficial for a number of reasons. First, such sessions are often needed to assist in making adjustments or refinement in the initial TIB prescriptions. Secondly, follow-up visits provide additional opportunity to encourage and reinforce treatment adherence. These visits also enable the therapist an opportunity to evaluate treatment response and "troubleshoot" problems enacting the behavioral regimen. Lastly, some people present with sleep-interfering cognitions that need to be specifically addressed; thus subsequent sessions may be devoted to cognitive restructuring. We will discuss cognitive restructuring in the next two chapters. The following discussion provides separate consideration of the behavioral aspects of the follow-up sessions.

SLEEP HYGIENE INSTRUCTIONS

1   Limit your use of caffeinated foods and beverages such as coffee, tea, soft drinks with added caffeine or chocolates. Caffeine is a stimulant that may make it harder for you to   sleep well at night.

2   Limit your use of alcohol. Alcoholic beverages may make you drowsy and fall asleep more easily. However, alcohol also usually causes sleep to be much more broken and far less refreshing than normal.

3   Avoid exercise and other physical activity right before bed. Being too active right before bed may make it harder to get to sleep quickly.

4   Protect the hour or two before your usual bedtime as a time to relax and wind-down before you enter bed. If you make this a practice, you will find it is easy to "shut of the day" and get to sleep more easily.

5   Try a light bedtime snack that includes such items as cheese, milk, or peanut butter. These foods contain chemicals that your body uses to produce sleep. As a result, this type of bedtime snack may actually bring on drowsiness.

6   Make sure that your bedroom is quiet and dark. Noise and even dim light may interrupt or shorten your sleep.

7   Make sure the temperature in your bedroom is comfortable. Generally speaking excessively warm temperatures cause unwanted wake-ups from sleep.

Notes:

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Fig. 6.5 Sleep hygiene instructions handout

Assistance with TIB Changes

A minimum of 1 week and preferably two weeks are needed with most people to provide a test of the initial TIB prescription they are given. If upon the first return visit they are sleeping soundly at night and feeling generally alert and functional in

the daytime, no adjustments to the initial TIB prescription are needed. However, if there continues to be an undesirable amount of nighttime wakefulness, it may be necessary to decrease the TIB somewhat. What constitutes an "undesirable amount of wakefulness" can vary somewhat from person to person, but recent studies (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003; Lineberger, Carney, Edinger, & Means, 2006) suggest that sleep onset times or periods of wakefulness in the middle of the night exceeding 30 min are outside the common experience of normal sleepers. Hence, when the average sleep onset time or the average time awake in the middle of the night exceeds this amount of time, it is useful to consider a reduction in TIB to encourage a more consolidated sleep pattern. Unless, an excessively large amount of wake time remains, a 15–30 min downward titration in the initial TIB prescription is usually all that is needed to achieve an optimal sleep pattern.

In contrast, if at the first follow-up visit the sleep diary shows consistently solid sleep, yet there are complaints about ongoing daytime fatigue or sleepiness, then an upward titration of the initial TIB is indicated. One signal that this is needed is that the patients report being woken by their alarm clock most mornings. Arguably, such circumstances suggest they could routinely sleep longer in the morning if the alarm had not sounded. When this is the case, titrating the TIB upward slowly in 15-min increments from visit to visit is typically the best approach. Alternately, the person may find it difficult to stay awake until the prescribed bedtime, fall asleep quickly each night (i.e., within 5–15 min), sleep solidly throughout the night, and complain of daytime sleepiness. In this case, one can consider titrating the 15 min by setting an earlier bedtime. The optimal TIB prescription is reached when the average sleep onset time and wake time after sleep onset are each <30 min, the person reports routinely awakening slightly before the alarm each day, and there is an absence of significant daytime sleepiness and fatigue.

Reviewing/Reinforcing Adherence

It is not difficult to appreciate the central importance of treatment adherence in ensuring the efficacy of the treatment strategies discussed in this chapter. These behavioral changes require considerable commitment, and adherence to them is recognized as one of the most critical factors affecting treatment success (Chambers & Alexander, 1992; Morin & Wooten, 1996). Approximately 15% of research participants fail to follow through and complete behavioral insomnia therapy (Perlis, Aloia, & Millikan, 2000), but some studies suggest that this rate may approach 40% in clinical venues (Ong, Kuo, & Manber, 2008; Perlis et al., 2000). Factors which have been linked to nonadherence and attrition include greater sleep impairment, poorer perceived general health, higher levels of depression, less favorable ratings of behavioral treatment strategies, and a greater tendency to view the therapist as critical and confrontive (Constantino et al., 2008; Morgan, Thompson, Dixon, Tomeny, & Mathers, 2003; Ong et al., 2008; Perlis et al., 2000; Vincent & Walker, 2001). Monitoring adherence and reinforc-

ing proper treatment enactment, thus, is critical to the outcome of these insomnia interventions.

Therapists should nonjudgmentally ask about adherence difficulties and review sleep diary information to assess any variance from stimulus control, sleep restriction, and sleep hygiene recommendations. The therapist should freely compliment attempts at following treatment recommendations. In doing so, however, it is particularly useful to point out the relationship between the enactment of treatment suggestions and improvement noted by sleep diaries and/or self-report. For example, therapist comments like, "You have done a really good job following the recommendations we discussed last time. It seems as though your efforts have been rewarded. Your sleep diaries show that you are now sleeping much better. Great job." Positive reinforcement of the existing adherence and the relation to sleep improvement can also be used to improve remaining areas of nonadherence. For example, "Wow, you have really managed to keep to your scheduled rise time, and it seems to have paid off with improved sleep. I notice that you napped a few times during the week and these seemed to be followed by your worst sleep nights. You are already doing such a good job; do you think eliminating these two naps might make the difference in getting even better sleep?" In providing such comments, it is important to remain genuine and avoid patronizing the patient. Thus, language that is consistent with the therapist's usual interpersonal style should be used in reinforcing adherence.

Trouble-Shooting Problems

Often, an inadequate treatment response results from a misunderstanding of or not enacting treatment recommendations. The most common of these adherence problems include not adhering to a standard rise time, not getting out of bed during the night during extended periods of wakefulness, and engaging in unintentional sleeping during the daytime. Common sleep hygiene violations include consumption of caffeine or alcohol too close to bedtime and failure to allow sufficient "wind-down time" prior to bed. A careful review of sleep diaries can identify deviations from prescribed rising times. Adherence problems such as the occurrence of daytime dozing episodes, problems with alcohol or caffeine use, difficulties setting aside time to relax before bed, and extended periods of wakefulness spent in bed, should be queried with curiosity not judgment. When such problems are identified, the relevant treatment recommendations and their rationale should be reviewed. The therapist should also suggest ways the person might avoid the identified sleep disruptive practices. When there are difficulties enacting recommendations, the therapist should encourage problem-solving of the difficulty and develop plans that will facilitate enactment of that recommendation. The following case examples demonstrate how the therapist may intervene when these problems are identified.

Case 1

Mr. G presented with a complaint of sleep-maintenance insomnia. Initial evaluation suggested that he showed many of the sleep disruptive practices addressed by the behavioral treatment discussed herein, so he was provided a course of behavioral insomnia treatment to address his complaints. After 1 week of this treatment, he reported little improvement. However, his sleep diaries and a follow-up discussion revealed that he did not adhere to the instructed standard rise time. On three of the nights during the first week of treatment, he stayed in bed over 2 h beyond his prescribed wake-up time reportedly to make up for poor nights of sleep. Also, he admitted that he did not get out of bed during extended periods of wakefulness because he hoped that if he would lie in bed long enough, he would eventually go to sleep. Although he denied daytime napping, he acknowledged some unintentional dozing in the evening while reclining on a couch watching TV.

To address this sleep problem, the therapist invited exploration of the possible disruptive effect of the noted adherence difficulties would have on his sleep. Collaboratively, Mr. G and the therapist decide that placing the alarm clock in a location out of reach from the bed might help force him to get out of bed at the agreed upon rising time. They also jointly derive a list of activities he might do instead of lying in bed when he experiences extended nocturnal awakenings. Mr. G was also encouraged to sit upright while watching TV in the evening and to have his wife help avoid his usual dozing during the early evening hours. At a follow-up session 1 week later, he showed markedly improved treatment adherence and an associated reduction in his sleep maintenance difficulty.

Case 2

Ms. Q was a retired 74-year-old woman who also presented with sleep maintenance complaints. There were many treatment targets evident from the initial assessment including unintentional evening napping and spending an excessive amount of time in bed on most nights. Standard behavioral insomnia treatment was initiated which included restricting her time in bed to 7 h and enlisting her husband's help with dozing in the evening. Less than 1 week after her first appointment, she phoned the therapist with concerns about her increased daytime sleepiness. She expressed concerns about driving because of an incident wherein she fell asleep in her car while stopped for a traffic light. Ms. Q adhered to the time in bed restriction diligently and she was sleeping very soundly on most nights. However, she continued to feel sleepy in the daytime and had to constantly fight off naps.

Given the seriousness of the driving accident and Ms. Q's concerns, the therapist suggested her to increase her time in bed by 30 min per night to try to reduce this sleepiness and temporarily ask her husband to assume driving responsibilities. At her next appointment, she reported reduced daytime sleepiness with the

increased time in bed. Her diaries showed that she was sleeping well at night with very few long awakenings. Since she continued to report some mild sleepiness, the therapist suggested her to add another 15 min to her TIB each night. After trying this new TIB prescription, she reported elimination of her daytime sleepiness and a continuation of her improved nighttime sleep.

Case 3

Mr. M was a 52-year-old man with a long history of insomnia and generalized anxiety disorder. To combat his problem, he developed the habit of consuming 1–2 shots of alcohol in the evening shortly before bedtime. Usually he had little difficulty falling asleep, but he often awakened and could not return to sleep easily. During his follow-up appointment, it was evident that his sleep had become worse as he continued to have fragmented sleep but also began to have difficulties falling asleep. When asked about his experience in trying to follow the treatment recommendations, Mr. M acknowledged that he continued to drink alcohol close to bedtime several nights each week. He also reported that the idea of giving up the alcohol and restricting his sleep had made him very anxious. The therapist asked whether a more lenient amount of time in bed would be more achievable and whether Mr. M thought he could adhere to this schedule over the next few weeks. Mr. M said this was less anxiety provoking and he thought that this was something he could try. The therapist and Mr. M reviewed the sleep diary data to explore the association between bedtime alcohol consumption and subsequent poor sleep. To address this problem, the therapist encouraged Mr. M to move his alcohol consumption to an early time such as dinner, so that it did not interfere with his sleep. Subsequently, he was able to follow the sleep schedule and was generally able to refrain from alcohol consumption after his evening meal, and his nighttime awakening problem diminished.

These cases demonstrate some strategies that might be used to address behavioral adherence issues in follow-up sessions. Admittedly, the cases presented do not illustrate all possible problems patients might present in adhering and tolerating treatment. Nonetheless, they do illustrate some commonly encountered problems and provide some demonstration as to how to intervene. In the end, therapy should be guided by the sleep diary data and by the patient's self-appraisal. Sleeping soundly at night and having no daytime symptoms of insomnia (e.g. fatigue, impaired concentration, distress about sleep) should be the ultimate goal for each patient. When this is the case, sleep diaries typically show a regular sleep/wake schedule and little difficulty with sleep initiation or maintenance. Once the person achieves a sound sleep pattern at night and is satisfied with his or her daytime function, therapy termination may be considered.

References

Bootzin, R. R. (1972). Stimulus control treatment for insomnia. *Proceedings of the 80th Annual Meeting of the American Psychological Association, 7*, 395–396.

Carney, C. E., & Edinger, J. D. (2006). Identifying critical dysfunctional beliefs about sleep in primary insomnia. *Sleep, 29*(4), 440–453.

Chambers, M. J., & Alexander, S. D. (1992). Assessment and prediction of outcome for a brief behavioral insomnia treatment program. *Journal of Behavior Therapy and Experimental Psychiatry Annals, 23*, 289–297.

Constantino, M. J., Manber, R., Ong, J., Kuo, T. F., Huang, J. S., & Arnow, B. A. (2008). Patient expectations and therapeutic alliance as predictors of outcome in group cognitive-behavioral therapy for insomnia. *Behavioral Sleep Medicine, 5*(3), 210–228.

Edinger, J. D., & Carney, C. E. (2008). *Overcoming insomnia: A cognitive behavior therapy approach therapist guide*. New York: Oxford University Press.

Edinger, J. D., & Fins, A. I. (1995). The distribution and clinical significance of sleep time misperceptions among insomniacs. *Sleep, 18*, 232–239.

Edinger, J. D., Wohlgemuth, W. K., Radtke, R. A., Marsh, G. R., & Quillian, R. E. (2001). Does cognitive-behavioral insomnia therapy alter dysfunctional beliefs about sleep? *Sleep, 24*, 591–599.

Lichstein, K., Durrence, H., Taylor, D., Bush, A., & Riedel, B. (2003). Quantitative criteria for insomnia. *Behavioral Research and Therapy, 41*, 427–445.

Lineberger, M., Carney, C., Edinger, J., & Means, M. (2006). Defining insomnia: Quantitative criteria for insomnia severity and frequency. *Sleep, 29*, 479–485.

Means, M. K., Edinger, J. D., Glenn, D. M., & Fins, A. I. (2003). Accuracy of sleep perceptions among insomnia sufferers and normal sleepers. *Sleep Medicine, 4*, 285–296.

Morgan, K., Thompson, J., Dixon, S., Tomeny, M., & Mathers, N. (2003). Predicting longer-term outcomes following psychological treatment for hypnotic-dependent chronic insomnia. *Journal of Psychosomatic Research, 54*(1), 21–29.

Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York: Guilford Press.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A. J., Buysse, D. J., & Bootzin, R. R. (1999). Nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine review. *Sleep, 22*, 1134–1156.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J., & Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychology and Aging, 8*, 463–467.

Morin, C. M., & Wooten, V. (1996). Psychological and pharmacological approaches to treating insomnia: Critical issues in assessing their separate and combined effects. *Clinical Psychology Review, 16*(6), 521–542.

Ong, J. C., Kuo, T. F., & Manber, R. (2008). Who is at risk for dropout from group cognitive-behavior therapy for insomnia? *Journal of Psychosomatic Research, 64*(4), 419–425.

Perlis, M., Aloia, M., & Millikan, A. (2000). Behavioral treatment of insomnia: A clinical case series study. *Journal of Behavioral Medicine, 23*, 149–161.

Spielman, A. J., Saskin, P., & Thorpy, M. J. (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep, 10*, 45–55.

Vincent, N., & Lionberg, C. (2001). Treatment preference and patient satisfaction in chronic insomnia. *Sleep, 24*, 411–417.

Vincent, N., & Walker, J. (2001). Anxiety sensitivity: Predictor of sleep-related impairment and medication use in chronic insomnia. *Depression and Anxiety, 14*, 238–243.

Wohlgemuth, W. (1999). How many nights are enough? The short-term stability of sleep parameters in elderly insomniacs and normal sleepers. *Psychophysiology, 36*(2), 233–244.

Chapter 7
Sleep-Related Cognitive Processes

Abstract The essence of a cognitive model of psychopathology is the implication of multiple cognitive processes (including schematic, attentional, and perceptual biases) that predispose and perpetuate a given disorder. Considerable research has amassed to support this model in insomnia; that is, people with insomnia have a range of cognitive–emotional processes that make it more likely for the insomnia to occur and continue (Behav Res Ther 40:869–893, 2002). Harvey (Behav Res Ther 40:869–893, 2002) presents a comprehensive contemporary cognitive model of insomnia, which includes a range of sleep-interfering cognitive processes including beliefs, perception, and attention. We discuss each component of Harvey's Cognitive Model of Insomnia and provide evidence in support of such a model. This chapter sets the stage for a detailed discussion of cognitive strategies in the subsequent chapter.

Harvey's (2002) Cognitive Model

Mr. S wakes up in the middle of the night and notices the clock reads 2:40 AM. He thinks, "I'm NEVER going to be able to sleep." While having this negatively valenced thought, Mr. S notices that he has a "nervous stomach" and that he feels tense. He can feel the distress mounting, and he begins to have other thoughts such as, "I have a big day tomorrow – I'm never going to be able to get through it if I don't get some sleep." He feels even more distressed, and the thoughts and anxiety feed into each other, creating a vicious cycle. The thought-distress cycle is purportedly fueled by a core belief (i.e., schema) that Mr. S is unable to cope with stress. While this is not something Mr. S normally thinks about (i.e., this belief is latent and otherwise out of awareness), performance-laden situations activate his fear that he cannot cope with and lead to thoughts that are consistent with this belief. In turn, he interprets the distress and activation he feels in the moment as evidence that he is not "good at coping" – thus further maintaining this belief. This distressing turn of events focuses his attention selectively on things that confirm that he will struggle with returning to sleep. He becomes vigilant for internal signs of evidence (i.e., a physical state such as anxiety) that would suggest he would have difficulty returning to sleep.

Perhaps he becomes focused on similar cues in the external environment that might not have been perceived had they not been aroused, for example, hearing a noise in the distance and worrying that it will prevent him from resuming sleep. Perhaps, he monitors the clock and makes calculations for how much more sleep he could obtain if he were to fall asleep right away. This selective attention and monitoring for stimuli that are threatening to his ability to produce sleep make it increasingly likely that these stimuli will be present. In an attempt to deal with this heightened state of cognitive–emotional arousal, Mr. S tries to manage the aversive situation by attempting to avoid it. He employs safety behaviors, such as taking an extra dose of sleeping medication. Unfortunately, this does not alleviate his anxiety, and he becomes fixated on the idea that he may feel groggier the next day. He decides to call the voicemail of his supervisor to leave a message that he is sick and unable to come in to work today. Mr. S feels a slight release in his tension. Although this may result in a temporary relief of the performance related anxiety, it also strengthens/confirms the helplessness belief that he cannot cope with and makes it more likely to become activated in a future similar situation.

It is important to note that the same cognitive process that occurs during the night operates during the day. Insomnia is most accurately regarded as a 24-h disorder. Thus, Mr. S's increased focused on sleep-threat related material will make it more likely for him to detect and pay careful attention to physical symptoms that will confirm the idea that he slept poorly. His thoughts about fatigue or other daytime symptoms of insomnia lead to distress, increased attention, and monitoring. Again, this is all fueled by the belief that he is helpless to cope with the effects of sleep loss. This might also be linked to safety behaviors such as overuse of caffeine to compensate for the presumed consequences of sleep loss. This logical safety behavior may have the unintended effect of perpetuating the insomnia because there would be a need later in the day to consume more caffeine since the withdrawal effects of caffeine include fatigue symptoms. Increased caffeine use may also disrupt subsequent sleep. The distress created by detecting the presumed (threatening) daytime consequences of insomnia would garner an unhelpful degree of attentional resources, such that Mr. S would be more likely to perceive further symptoms, which might normally go unnoticed and perhaps make inaccurate attributions about their cause. For example, he may feel tired and attribute that feeling to poor sleep when he may actually be dehydrated. This will lead to feeling more anxious and pressured to sleep well the next night, instead of simply drinking a glass of water and addressing the problem. But is there any evidence that Mr. S's experience is typical in insomnia? We attempt to answer this question by examining the evidence for Harvey's (2002) model.

The Role of Thoughts

One of the basic components of Harvey's model is the activational role of negative thoughts in a sequence characterized by increased arousal/distress and unhelpful behaviors. Thus, it seems prudent to start here. The majority of work in the area has focused on the content of presleep cognitions. In insomnia, the presleep thought

content is often negatively valenced (Harvey, 2002) and is characterized by thinking about "thoughts" or the sleeping environment (e.g., temperature or noise), planning, problem solving, ruminating about past events, and/or worrying about the inability to sleep (Harvey, 2000; Levey, Aldaz, Watts, & Coyle, 1991; Watts, Coyle, & East, 1994; Wicklow & Espie, 2000). The more negatively valenced thought content such as worry and rumination is strongly associated with increased sleep onset latency (Wicklow & Espie, 2000). In addition to the valence of thoughts, people with insomnia tend to have greater thought activation, that is, they have intrusive mental activity while trying to fall asleep. Cognitive overactivity is seen by those with insomnia as ten times more important than somatic tension in the perpetuation of their insomnia (Lichstein & Rosenthal, 1980). The content of overactive thinking is sometimes related to topics from the daytime that continue into the sleep period. Thus, there is a preponderance of thoughts related to work, family, and mundane topics, and such thoughts are often characterized by problem solving (Wicklow & Espie, 2000). In addition, people with insomnia tend to think specifically about sleeplessness (Fichten et al., 1998; Harvey, 2000; Kuisk, Bertelson, & Walsh, 1989; Watts et al., 1994; Wicklow & Espie, 2000).

Unwanted Repetitive Mental Activity

In insomnia, one of the most well known instructions in folk psychology to deal with repetitive thoughts is the advice to count sheep. The idea behind counting sheep actually makes sense because counting occupies space in the *articulatory loop*, a construct that refers to the brain's processing of ongoing information. There is a limited amount of space in the articulatory loop, so it is difficult to keep both old and new thoughts in the loop simultaneously. Thus, the focus on new thought content such as counting sheep essentially ejects previous (unwanted) material from the loop. The problem is that counting sheep is boring, so it may not readily stay in the articulatory loop. In a more sophisticated experimental version of counting sheep, people with insomnia were asked to repeat the word "the" over and over again in their mind while trying to fall asleep. Interestingly, saying "thethethethethethe" actually shortened the experience of wakefulness in bed (Levey et al., 1991). So why do not we prescribe this to those with insomnia? The practical problem with this is that saying "the" is about as exciting as counting sheep, so it may not be interesting enough to occupy the loop for long. Some people with insomnia instead try to suppress unwanted thoughts, but suppression attempts have the opposite effect; that is, the unwanted thoughts (and wakefulness) persist even longer (Ree, Harvey, Blake, Tang, & Shawe-Taylor, 2005). This cycle of unwanted recurring thoughts has been described in many different disorders as repetitive thinking (Segerstrom, Tsao, Alden, & Craske, 2000), and the most pertinent types of repetitive thinking in insomnia are rumination and worry (Carney, Edinger, Meyer, Lindman, & Istre, 2006; Harvey, 2002; Thoresen, Coates, Kirmil-Gray, & Rosekind, 1981). The intrusion of this type of thinking has prompted the testing of bedtime

arousal-decreasing strategies for processing intrusive material for the day via early evening problem solving (Carney & Waters, 2006; Espie & Lindsay, 1987) or a Pennebaker writing (Harvey & Farrell, 2003) assignment. These and other cognitive strategies will be discussed further in Chap. 8.

The Role of Distress

There have been many studies showing increased emotional arousal in those with insomnia (Carskadon et al., 1976; Coursey, 1975; Kales, Caldwell, Preston, Healey, & Kales, 1976; Monroe, 1967). In addition to reporting more distress, people with insomnia also report taking longer time to emotionally recover from daytime stressors (Waters, Adams, Binks, & Varnado, 1993). One further piece of evidence for Harvey's (2002) model is that the presleep thought content of people with insomnia tends to be negatively valenced (Kuisk et al., 1989). Manipulating presleep distress (e.g., instructing people that they will have to do a speech upon awakening in the morning) tends to disrupt sleep (Gross & Borkovec, 1982). Thus, there is support for distress and emotional arousal in those with insomnia.

Beliefs in Insomnia

Morin (1993) is largely responsible for importing Beck's Cognitive Theory of psychopathology (Beck, 1976) into the area of insomnia. In Beck's classic Cognitive Theory beliefs are the basis for the automatic thoughts. Early cognitive conceptualizations focused on unhelpful beliefs as perpetuating and potentially predisposing factors in insomnia (Morin, 1993). In insomnia, beliefs are thought to drive sleep-interfering behavior and maintain arousal/distress in the face of poor sleep. Beliefs also drive a tendency to seek out and pay more careful attention to information confirming the presumption that poor nighttime sleep will occur or daytime functioning will be impaired. Research has shown that, in contrast to good sleepers, people with insomnia have an unhelpful degree of beliefs about sleep that make them more prone to insomnia (Carney & Edinger, 2006). Collectively, these beliefs have been shown to respond to CBT for insomnia (Carney & Edinger, 2006; Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001b; Morin, Blais, & Savard, 2002). These beliefs also show some improvement with relaxation therapy, although not as much as with CBT (Edinger et al., 2001b). There is less cognitive improvement with pharmacotherapy when compared with CBT for insomnia (Morin et al., 2002). Presumably, these belief changes relate to decreased helplessness about their sleep problem (i.e., relaxation therapies and pharmacotherapy produce sleep improvements). However, despite sleep improvements in all of these treatments, these beliefs do not change as much as the change associated with a belief-targeted treatment such as CBT. These CBT belief improvements significantly relate to other indices of clinical improvement including PSG (Edinger et al., 2001b), sleep

diaries, and other sleep measures such as sleep self-efficacy and a global insomnia symptom questionnaire (Carney & Edinger, 2006).

Morin (1993) describes two main beliefs in insomnia: (1) that there is something wrong and there is a sense of lowered self-efficacy about the ability to produce sleep; and (2) worry about the consequences of poor sleep (Morin, 1993). These were echoed in Beck's later writings about beliefs across multiple disorders (Beck, 1999). These two beliefs are mainly concerned with a belief of helplessness to cope with sleep loss. This is arguably a trans-diagnostic belief that could conceivably predispose a person to comorbid disorders in addition to insomnia.

In addition to helplessness, a second common theme across such beliefs is the belief that effort is required to sleep (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006). Perhaps, it is the thwarted attempts at sleep effort that lead to the activation of helplessness related beliefs. Espie and his colleagues used the following quote from Frankl (1965) to demonstrate this phenomenon: "Sleep (is like) a dove which has landed near one's hand and stays there as long as one does not pay any attention to it; if one attempts to grab it, it quickly flies away." (Ansfield, Wegner, & Bowser, 1996) Sleep is something that occurs in the absence of effort. In Espie et al. (2006) attention–intention effort model, the good sleeper is seen as passive, and sleep behavior is determined by cues of sleepiness at night and waking cues in the morning. Such a pattern of reinforcement shapes the pattern without much thought or effort on the part of the good sleeper. Poor sleep occurs chronically when people begin to pay attention and exert effort over their sleep. There is evidence of increased sleep-related effort in those with insomnia relative to good sleepers via self-report (Broomfield & Espie, 2005) and experimental manipulations that show improved sleep when the instruction is to stay awake (Ascher & Turner, 1979; Broomfield & Espie, 2003). Clinically speaking, sleep effort is an important construct because it may be the motivation behind maladaptive sleep behaviors. Going to bed early to *catch-up* on lost sleep implies that one needs to *do* something to catch-up. Going to bed earlier can decrease the likelihood for sleep through its deleterious effect on homeostatic sleep drive. Thus, effort-related beliefs can contribute to important behavioral perpetuating factors for insomnia, and consequently must be targeted in CBT.

Attention and the "Threat" of Sleep

Do people with insomnia exhibit increased attention to their sleep? One of the criteria for Psychophysiologic Insomnia in the International Classification of Sleep Disorders, Diagnostic and Coding Manual, Second Edition (ICSD-2) is an "excessive focus" on sleep. Studies have supported attentional bias for sleep-related words (Taylor, Espie, & White, 2003) and sleep-related visual stimuli (Jones, Macphee, Broomfield, Jones, & Espie, 2005) in people with insomnia relative to normal sleepers. The importance of attention as a perpetuating factor is not a novel idea and is in fact invoked in anxiety disorder psychopathology models (Mathews & MacLeod, 1994). In anxiety disorders, the focus of the heightened attention is on

threat-related material (Mogg, Mathews, Bird, & MacGregor-Morris, 1990). Insomnia is no exception, and the material is presumed to be sleep threat-related stimuli. Indeed, in the ICSD-2 diagnostic classification scheme mentioned above, Psychophysiological Insomnia is characterized by heightened anxiety about sleep. Espie et al. (2006) points to sleep's prominent place in Maslow's (1943) Hierarchy of Human Needs to explain why sleeplessness is so threatening. If sleep is one of our most basic needs, then it would make sense for considerable resources to be utilized to remediate this need if sleeplessness were to occur. Several investigations have also found support for attention focus on sleep-related threat stimuli (Harvey, 2002; Semler & Harvey, 2004; Tang & Harvey, 2004). Sleep-related threat stimuli includes daytime threats such as scanning the body for fatigue or other symptoms thought to be associated with poor sleep, as well as nocturnal threats such as scanning the body for symptoms predictive of poor sleep, or focusing on the extended amount of time it takes to fall asleep (Harvey, 2002).

One example of a sleep-threat stimulus is the bedroom clock. Many people with insomnia will admit to watching the clock and becoming anxious as they make calculations of a shortened sleep opportunity. This is demonstrated in a clever experiment, wherein those with primary insomnia were assigned to one of two groups: a clock-monitoring group and a digital display unit monitoring group (Tang, Schmidt, & Harvey, 2006). The digital display unit was identical to the digital clock in the other condition; however, it was programmed to display random digits that changed every minute. On the monitoring night, the clock-monitoring group reported greater sleep-related worry, and longer estimated sleep onset latency (SOL), relative to baseline and relative to monitoring night data in the digital display unit monitoring group. The groups did not differ on objective (actiwatch) SOL estimates; however, in the clock monitoring group, there was a tendency to overestimate subjective SOL, when comparing sleep diaries to objective (actiwatch) SOL estimates.

One implication for this study is support for the role of threat monitoring in increasing anxiety and potential misperception in sleep estimation. The tendency to monitor the clock may be indicative of an attentional bias toward threats to sleep. The perception of a threat to sleep increases anxiety and worry because as time progresses, less time is available for sleep (Harvey, 2002). Similarly, sleep-related worry purportedly drives the process of sleep estimation distortion (Borkovec, 1982; Harvey, 2002). Another key implication of this study is support for the instruction that it may be useful to remove clocks from view as part of insomnia treatment (Hauri, 1991; Morin, 1993).

The Role of Attributions

There are several studies that support the presumed role of misattribution in cognitive models of insomnia (Harvey, 2002; Harvey, Tang, & Browning, 2005; Morin, 1993). An early study reported that those with insomnia who were told that a placebo pill would produce arousal symptoms fell asleep faster than insomnia sufferers

who were told that the same pill would produce a relaxation response (Storms & Nisbett, 1970). The explanation? The arousal-pill group attributed their arousal to the pill rather than to an endogenous (internal) source, thus decreasing anxiety and decreasing SOL. Or perhaps, when the expected relaxation response was not detected, it increased arousal, thus increasing SOL. Similarly, another study combined medication with behavior therapy and at the end of the first treatment week, half of study patients were told that they were receiving a suboptimal dose of the medication, and the other half were told that they were receiving an adequate dose (Davison, Tsujimoto, & Glaros, 1973). All participants stopped the drug therapy and continued with behavior therapy. Those who were told that they had received a suboptimal dose showed greater maintenance of their improvements than those who were told that the dose was optimal. Those who were told that the dose was suboptimal did not attribute their improvement to the drug (because they were told the dose was not therapeutic). The other group did not maintain their improvements after stopping the drug because they attributed their improvement to the drug. Indeed, sleep improvement attributions appear to be important in clinical trials, as those treated with CBT evidence greater improvements in their confidence in being able to sleep than those in a control treatment (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001a).

Attributions also play an important role in daytime functioning (Morin, 1993). As part of Harvey's (2002) Cognitive Model, there is a purported tendency to misattribute daytime symptoms of insomnia, negative mood, or cognitive difficulties to poor nighttime sleep. Research with the most frequently used measure of maladaptive beliefs about sleep, the Dysfunctional Beliefs, and Attitudes about Sleep Scale (Morin, 1993; Morin, Vallières, & Ivers, 2007) suggests that both primary and comorbid insomnia groups tend to misattribute daytime symptoms to sleep to a greater degree than good sleepers (Carney & Edinger, 2006; Morin, 1993). Misattributing other causes of daytime symptoms such as fatigue increases the pressure to produce sleep. That is, if feeling poorly during the day is attributed solely to sleeping poorly (rather than the multitude of possible explanations for fatigue at any given time during the day), then there will be an increased anxiety about sleep. Indeed, cognitive restructuring is frequently aimed at correcting such misattributions (Edinger & Carney, 2008).

Maintaining the Status Quo

This attentional bias for confirmatory information also makes it more likely that such information will be perceived. Thus, a latent belief such as, "I need 8 h to sleep or I cannot cope," will result in distressing thoughts if there is a delay in falling asleep. This may drive thoughts such as, "I am *never* going to sleep," as well as ongoing anxiety. The attentional bias toward sleep threat-related material will make it more likely that the person will perceive the distress and anxiety symptoms and view them as evidence that he/she will sleep poorly. This will reinforce worries

about a future consequence of not sleeping (i.e., that they will not be able to cope with the daytime symptoms of insomnia). This may drive a safety behavior such as drinking alcohol to hasten sleep or leaving a message on the boss' voicemail that they are sick and cannot come to work. Such safety behaviors will lessen the pressure to sleep and may quell the arousal/anxiety in the short term; unfortunately, they also reinforce the original belief that one is helpless to cope with less than 8 h of sleep. Indeed, unhelpful/rigid beliefs about sleep predict reported usage of safety behaviors (Woodley & Smith, 2006). The result is a system that is set up to maintain the status quo of insomnia.

Teasdale (1997) describes a process in depression whereby, once cognitive structures are activated, it is as if a wholly different state of mind is adopted. The content of this "mind-in-place" is negative and self-referent, and the process that perpetuates this type of thinking is automatic and operates on a relatively closed circuit. As a consequence, disconfirming information has little opportunity to get in. The repetitive thoughts revolve around a negative view of the self, and are reinforced by feedback loops involving the effects of depression on other cognitive systems (e.g., attention and memory processes) and on the body (e.g., symptoms of fatigue) (Teasdale, Taylor, Cooper, Hayhurst, & Paykel, 1995).This description would seem applicable to insomnia as well. As applied to insomnia, Teasdale's theory would suggest that a person with insomnia is of a *different* insomnia-focused mind when experiencing sleep disturbance. Attentional resources are directed at perceiving threatening sleep-specific information that confirms unhelpful beliefs about sleep. The repetitive thought process (i.e., worry or rumination) is similar, but the cognitive thought content may be more related to a fixation on bodily symptoms and their implication for poor functioning (Carney et al., 2006). Indeed, self-focused rumination (as described in depression) is not a factor in insomnia, but symptom-focused rumination discriminated between poor and good sleepers (Carney et al., 2006). As a result of this perpetuating cognitive process, people with insomnia often feel helpless; that is, helpless to cope with sleep loss and helpless to produce sleep naturally. Many people with insomnia think that their sleep system is *broken,* and they worry about frightening, future catastrophic consequences. The collection of cognitive strategies described in the next chapter is focused on remediating these processes.

There has been considerable evidence for a cognitive perspective in insomnia. Sleep threat-based information processing and resultant sleep-interfering behaviors of those with insomnia tend to set up a psychological status quo, such that it is difficult for such individuals to break free of those factors perpetuating their difficulties. This may explain the success of CBT for insomnia, a treatment that targets unhelpful beliefs and behaviors.

References

Ansfield, M. E., Wegner, D. M., & Bowser, R. (1996). Ironic effects of sleep urgency. *Behaviour Research and Therapy, 34*, 523–531.

Ascher, L. M., & Turner, R. M. (1979). Paradoxical intention and insomnia: An experimental investigation. *Behaviour Research and Therapy, 17*, 408–411.

Beck, A. T. (1999). Psychoevolutionary view of personality and axis I disorders. In R. C. Cloninger (Ed.), *Personality and psychopathology* (pp. 721–730). Washington: American Psychiatric Press.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York: International Universities Press.

Borkovec, T. D. (1982). Insomnia. *Journal of Consulting and Clinical Psychology, 50*, 880–895.

Broomfield, N. M., & Espie, C. A. (2003). Initial insomnia and paradoxical intention: An experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. [References]. *Behavioural and Cognitive Psychotherapy, 31*(3), 313–324.

Broomfield, N. M., & Espie, C. A. (2005). Towards a valid, reliable measure of sleep effort. *Journal of Sleep Research, 14*(4), 401–407.

Carney, C., Edinger, J., Meyer, B., Lindman, L., & Istre, T. (2006). Symptom-focused rumination and sleep disturbance. *Behavioral Sleep Medicine, 4*(4), 228–241.

Carney, C. E., & Edinger, J. D. (2006). Identifying critical dysfunctional beliefs about sleep in primary insomnia. *Sleep, 29*(4), 440–453.

Carney, C. E., & Waters, W. F. (2006). Effects of a structured problem-solving procedure on pre-sleep cognitive arousal in college students with insomnia. *Behavioral Sleep Medicine, 4*(1), 13–28.

Carskadon, M. A., Dement, W. C., Mitler, M., Guilleminault, C., Zarcone, V. P., & Spiegel, R. (1976). Self-reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. *The American Journal of Psychiatry, 133*, 1382–1388.

Coursey, R. D. (1975). Personality measures and evoked responses in chronic insomniacs. *Journal of Abnormal Psychology, 84*(3), 239–249.

Davison, G. C., Tsujimoto, R. N., & Glaros, A. G. (1973). Attribution and the maintenance of behavior change in falling asleep. *Journal of Abnormal Psychology, 82*, 124–133.

Edinger, J. D., & Carney, C. E. (2008). *Overcoming insomnia: A cognitive behavior therapy approach therapist guide*. New York: Oxford University Press.

Edinger, J. D., Wohlgemuth, W. K., Radtke, R. A., Marsh, G. R., & Quillian, R. E. (2001a). Cognitive behavioral therapy for treatment of chronic primary insomnia: A randomized controlled trial. *JAMA: The Journal of the American Medical Association, 285*, 1856–1864.

Edinger, J. D., Wohlgemuth, W. K., Radtke, R. A., Marsh, G. R., & Quillian, R. E. (2001b). Does cognitive–behavioral insomnia therapy alter dysfunctional beliefs about sleep? *Sleep, 24*, 591–599.

Espie, C., Broomfield, N., MacMahon, K., Macphee, L., & Taylor, L. M. (2006). The attention-intention-effort pathway in the development of psychophysiologic insomnia: A theoretical review. *Sleep Medicine Reviews, 10*(4), 215–245.

Espie, C. A., & Lindsay, W. R. (1987). Cognitive strategies for the management of severe sleep maintenance insomnia: A preliminary investigation. *Behavioral Psychotherapy, 15*, 388–395.

Fichten, C. S., Libman, E., Creti, L., Amsel, R., Tagalakis, V., & Brender, W. (1998). Thoughts during awake times in older good and poor sleepers – The self-statement test: 60+. *Cognitive Therapy and Research, 22*(1), 1–20.

Gross, R., & Borkovec, T. (1982). Effects of cognitive intrusion manipulation on the sleep onset latency of good sleepers. *Behaviour Therapy, 13*, 112–116.

Harvey, A. G. (2000). Pre-sleep cognitive activity: A comparison of sleep-onset insomniacs and good sleepers. *The British Journal of Clinical Psychology, 39*, 275–286.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40*, 869–893.

Harvey, A. G., & Farrell, C. (2003). The efficacy of a Pennebaker-like writing intervention for poor sleepers. *Behavioral Sleep Medicine, 1*(2), 115–123.

Harvey, A. G., Tang, N. K. Y., & Browning, L. (2005). Cognitive approaches to insomnia. *Clinical Psychology Review, 25*, 593–611.

Hauri, P. J. (1991). Sleep hygiene, relaxation therapy, and cognitive interventions. In P. J. Hauri (Ed.), *Case studies in insomnia* (pp. 65–84). New York: Plenum.

Jones, B. T., Macphee, L. M., Broomfield, N. M., Jones, B. C., & Espie, C. A. (2005). Sleep-related attentional bias in good, moderate, and poor (primary insomnia) sleepers. *Journal of Abnormal Psychology, 114*(2), 249–258.

Kales, A., Caldwell, A. B., Preston, T. A., Healey, S., & Kales, J. D. (1976). Personality patterns in insomnia. *Archives of General Psychiatry, 33*, 1128–1134.

Kuisk, L. A., Bertelson, A. D., & Walsh, J. K. (1989). Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. *Perceptual and Motor Skills, 69*(3), 1219–1225.

Levey, A. B., Aldaz, J. A., Watts, F. N., & Coyle, K. (1991). Articulatory suppression and the treatment of insomnia. *Behaviour Research and Therapy, 29*(1), 85–89.

Lichstein, K. L., & Rosenthal, T. L. (1980). Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. *Journal of Abnormal Psychology, 89*, 105–107.

Mathews, A., & MacLeod, C. (1994). Cognitive approaches to emotion and emotional disorders. *Annual Review of Psychology, 45*, 25–50.

Mogg, K., Mathews, A., Bird, C., & MacGregor-Morris, R. (1990). Effects of stress and anxiety on processing of threat stimuli. *Journal of Personality and Social Psychology, 59*, 1230–1237.

Monroe, L. J. (1967). Psychological and physiological differences between good and poor sleepers. *Journal of Abnormal Psychology, 72*, 255–264.

Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York: Guilford Press.

Morin, C. M., Blais, F., & Savard, J. (2002). Are changes in beliefs and attitudes related to sleep improvements in the treatment of insomnia? *Behaviour Research and Therapy, 40*, 741–752.

Morin, C. M., Vallières, A., & Ivers, H. (2007). Dysfunctional Beliefs and Attitudes About Sleep (DBAS): Validation of a Brief Version (DBAS-16). *Sleep, 30*(11), 1547–1554.

Ree, M. J., Harvey, A. G., Blake, R., Tang, N. K. Y., & Shawe-Taylor, M. (2005). Attempts to control unwanted thoughts in the night: Development of the thought control questionnaire-insomnia revised (TCQI-R). *Behaviour Research and Therapy, 43*, 985–998.

Segerstrom, S. C., Tsao, J. C. I., Alden, L. E., & Craske, M. G. (2000). Worry and rumination: Repetitive thought as a concomitant and predictor of negative mood. *Cognitive Therapy and Research, 24*, 671–688.

Semler, C. N., & Harvey, A. G. (2004). Monitoring for sleep-related threat: A pilot study of the sleep associated monitoring index. *Psychosomatic Medicine, 66*, 242–250.

Storms, M. D., & Nisbett, R. E. (1970). Insomnia and the attribution process. *Journal of Personality and Social Psychology, 16*, 319–328.

Tang, N. K. Y., & Harvey, A. G. (2004). Effects of cognitive arousal and physiological arousal on sleep perception. *Sleep, 27*(1), 69–78.

Tang, N. K. Y., Schmidt, D. A., & Harvey, A. G. (2006). Sleeping with the enemy: Clock monitoring in the maintenance of insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 38*(1), 40–55.

Taylor, L. M., Espie, C. A., & White, C. A. (2003). A comparative experimental investigation of attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behavioral Sleep Medicine, 1*, 200–212.

Teasdale, J. D. (1997). The relation between cognition and emotion: The mind-in-place in mood disorders. In D. M. Clark & C. G. Fairburn (Eds.), *Science and practice of cognitive behavior therapy* (pp. 67–93). Oxford: Oxford University Press.

Teasdale, J. D., Taylor, M. J., Cooper, Z., Hayhurst, H., & Paykel, E. S. (1995). Depressive thinking: Shifts in construct accessibility or in schematic mental models? *Journal of Abnormal Psychology, 104*(3), 500–507.

Thoresen, C. E., Coates, T. J., Kirmil-Gray, K., & Rosekind, M. R. (1981). Behavioural self-management in treating sleep maintenance insomnia. *Journal of Behavioral Medicine, 4*, 41–52.

Waters, W. F., Adams, S. G., Binks, P., & Varnado, P. (1993). Attention, stress and negative emotion in persistent sleep-onset and sleep-maintenance insomnia. *Sleep, 16*(2), 128–136.

Watts, F. N., Coyle, K., & East, M. P. (1994). The contribution of worry to insomnia. *British Journal of Clinical Psychology and Aging, 33*, 211–220.

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research and Therapy, 38*, 679–693.

Woodley, J., & Smith, S. (2006). Safety behaviors and dysfunctional beliefs about sleep: testing a cognitive model of the maintenance of insomnia. *Journal of Psychosomatic Research, 60*(6), 551–557.

Chapter 8
Cognitive Strategies for Managing Anxiety and Insomnia

Abstract The previous chapter discussed the importance of cognitive processes (described by Harvey, Behaviour Research and Therapy 40:869–893, 2002) in perpetuating a sleep-interfering cognitive–emotional cycle as well as setting the stage for sleep-interfering behaviors. The motivation for sleep-interfering behaviors appears to relate to reciprocating beliefs for the need to exert sleep effort (described by Espie et al., Sleep Medicine Review 10:215–245, 2006) and feeling helpless when these efforts have no effect or actually worsen sleep (Morin, Insomnia: Psychological assessment and management. New York: Guilford Press, 1993); thus, we present step-by-step instructions for strategies for targeting these cognitive dispositions. These include a review of worry management strategies (including early evening and presleep strategies), cognitive restructuring (e.g., psychoeducation, Thought Records, behavioral experiments), and relapse prevention. Such strategies are aimed at managing and, in some cases, modifying perpetuating cognitive problems that contribute to insomnia problems.

We have discussed the importance of cognitive processes in perpetuating a sleep-interfering cognitive–emotional cycle as well as setting the stage for sleep-interfering behaviors. The motivation for sleep-interfering behaviors appears to relate to reciprocating beliefs for the need to exert sleep effort (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006) and feeling helpless when these efforts have no effect or actually worsen sleep (Morin, 1993). This process is arguably best described in Harvey's (2002) the model discussed in the previous chapter. In this chapter, we present some strategies for managing and, in some cases, modifying perpetuating cognitive problems that contribute to insomnia problems.

Worry Management Strategies

When discussing insomnia in the context of prominent anxiety, it may be best to start with strategies to manage presleep cognitive arousal, and then focus on a specific form of presleep arousal, namely worry. Managing presleep cognitive arousal

involves strategies that focus on (1) somatic relaxation such as PMR (Lichstein & Johnson, 1993), (2) eliminating arousal from the bed by leaving the bedroom when it occurs (stimulus control) (Bootzin, 1972), (3) scheduling worry and problem-solving outside of the bedroom (Carney & Waters, 2006; Espie & Lindsay, 1987), and (4) facilitating processing of the day's events (Harvey & Farrell, 2003). Stimulus control and relaxation techniques are covered in the Chaps. 6 and 9, respectively, so we will focus on scheduled worry and facilitated processing techniques in this chapter.

Even in the absence of an anxiety disorder diagnosis, presleep worry can contribute to presleep arousal and delay the onset of sleep. It is well documented that people with insomnia tend to worry before bed and in bed (Harvey, 2000; Wicklow & Espie, 2000). Even in good sleepers, experimentally induced presleep worry can interfere with sleep (Gross & Borkovec, 1982; Hall, Buysse, Reynolds, Kupfer, & Baum, 1996). Some presleep activity is not necessarily affectively laden (e.g., focusing on environmental stimuli like noise). However, the presleep content is often characterized by anxious thoughts about daytime worries and sleeplessness (Fichten et al., 1998; Wicklow & Espie, 2000). Stimulus control may be enough to deal with unintended mental habits like thinking about the sleeping environment, but when negatively valenced mental activity persists, it may help to try an early evening procedure intended to deal with the day's "unfinished business."

Espie and Lindsay (1987) were among the first to suggest that those with insomnia should schedule a time in the evening to address, and then set aside concerns that may interfere with subsequent nocturnal sleep. Carney and Waters (2006) tested a similar procedure called Constructive Worry in an analog sample. Although these strategies have not been tested in anxiety disorders populations, these two studies suggest that this is a useful intervention for reducing sleep-interfering presleep arousal in those with insomnia.

The instructions for Constructive Worry are straightforward and described in full elsewhere (Carney & Waters, 2006; Edinger & Carney, 2008). Briefly, the instructions are to set aside some time in the early evening, when the person is at their "problem-solving best." Arriving at a suitable time can take some discussion as some people believe they do not have the time for such an experiment. Yet, with adequate consideration of scheduling options, usually a time can be found for this activity. During this scheduled time, the individual should identify and then address worries using the worksheet shown on the following page. Those worries or concerns that have the potential to keep the person awake that night should be recorded on the first column of a 2-column form. In the adjacent second column should be the "next" steps towards solving this problem. Carney and Waters (2006) emphasize that the focus should not be on the ultimate or end solution as people can become anxious and ultimately overwhelmed if the final solution will take many steps to solve. Instead, the most proximal steps are the steps that are written down so that something could potentially be done the next day to start solving the problem.

Constructive Worry Worksheet Example

Worry	Next possible step
Need to finish my annual report	Can make a to-do list right now
	I need to call Bob tomorrow and get the final figures
	I need to email Debra about how to complete Sect. 4
	I can cut and paste from last year's report

It is most important to orient people towards problem solving. Arguably, worry is problem-solving thwarted by anxiety (Davey, 1994). If the individual can set aside time to engage in effective problem solving, there is less likelihood of becoming overwhelmed/anxious and greater probability of successfully dealing with sleep-disruptive topics. Not all problems have an easily identified solution or even a solution at all. In such cases, it is important for people to consider asking someone else for help or advice, accepting that there might not be a solution, or acknowledging that there is not an immediate solution but they can write down that they will revisit the problem tomorrow evening to see if a solution presents itself. At the conclusion of the exercise, the individual should acknowledge having dealt with the problem list as well as can be expected during a time of day when problem-solving is optimal, and the individual should also recognize that trying to solve it at night will be ineffective, anxiety-provoking, and sleep-interfering. Some find it useful to fold the worksheet over and ceremoniously put it away for the evening. The Constructive Worry procedure appears to have good implementation feasibility as the effectiveness of participants' solutions was rated as high using the Means-End Problem Solving Procedure (Platt & Spivack, 1975) in the Carney and Waters (2006) study.

A similar intervention is one that attempts to process information that is likely to intrude into the sleep period. This intervention for insomnia (Harvey & Farrell, 2003) is based on the Pennebaker writing exercise described and tested in the health and trauma literature (Francis & Pennebaker, 1992; Pennebaker, Kiecolt-Glaser, & Glaser, 1988). An investigation of a Pennebaker writing exercise showed that poor sleepers who complained about excessive mental activity in bed fell asleep more quickly if they were instructed to focus on writing about emotional content than did those who were not instructed to write (Harvey & Farrell, 2003). Just as with Constructive Worry, this intervention has not been evaluated in those with insomnia and an anxiety disorder. The instructions are simple and similar to the Constructive Worry procedure. The rationale provided to users of this strategy is that there are expected benefits to writing down thoughts or concerns before bed, and then "letting them go". They are encouraged to explore their deepest emotions and thoughts during this exercise. The Pennebaker and Constructive Worry procedures typically request that 20–30 min are set aside to complete the exercise. In contrast to the Constructive Worry procedure that is completed several hours before bed, the Pennebaker writing procedure is typically completed just prior to getting into bed. Espie and Lindsay (1987) suggested a procedure that targets presleep arousal at an

earlier time in the evening and this approach may be more effective than procedures conducted at bedtime. Sleep onset latency (on the PSQI) was measured, before and after the Pennebaker exercise, whereas anxiety has not been assessed in this setting, so it is possible that this presleep exercise could be activating in a clinical sample. In the analog investigation of the Pennebaker procedure mentioned, the decrease in sleep onset latency might suggest that this is not anxiety-provoking, but this needs to be evaluated in a future study. It is not clear how successful people in the Pennebaker experiment were with emotional processing; indeed, those in the control group who wrote about their hobbies did not differ from the writing about emotional topics group.

Cognitive Restructuring

Insofar as maladaptive beliefs about sleep or the self (i.e., helplessness) are perpetuating factors in insomnia, we need techniques to modify and/or manage these cognitions. Arguably, the three most common tools in cognitive restructuring are (1) psychoeducation about sleep, (2) Thought Records and in-session Socratic questioning, and (3) behavioral experiments. We will discuss Thought Records and behavioral experiments in detail in the ensuing sections. In the current section, we consider the use of psychoeducation in cognitive therapy.

Psychoeducation remains an important part of insomnia treatment. Early CBT trials used psychoeducation exclusively as a cognitive tool (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001). Psychoeducation is merely the verbal provision of corrective sleep-related information. Some examples include an explanation of the variability of sleep needs and the idea that humans do not necessarily require 8 h of sleep. We provide this information because it is a common misconception among people with insomnia; when in actuality, some people require more sleep, and some less. Even among those who require more than 8 h, there may be current circumstances that will shorten sleep need temporarily. Thus, it is important for people not to focus on a magical number as it is often incorrect and subject to change. It is also helpful to provide people with an explanation of how their homeostatic and circadian systems function to regulate sleep (described in Chap. 6). This explanation may highlight how increased sleep effort interferes with normal homeostatic and circadian sleep regulation and consequently contributes to insomnia. Discussing how sleep need decreases with advancing age and decreased activity as well as considering various causes for fatigue (i.e., boredom, circadian factors, diet, caffeine crash, virus, or dehydration) other than deficit sleep is also useful to correct common misconceptions and misattributions.

It is also important to provide normative values for sleep as some people with insomnia have sleep that is actually within normal limits. Hearing that their sleep is within the normal range can alleviate anxiety for some people so that no further treatment may be necessary. For example, it is normal to take up to 30 min to fall asleep and to be awake in the middle of the night up to 30 min. Psychoeducation

alone may adequately address many problematic sleep beliefs that contribute to insomnia. Nonetheless, there are many instances where this form of intervention is not sufficient and a more potent cognitive intervention may be needed. In such cases, a more intense and structured intervention as described in the following text should be considered.

Thought Record

The Thought Record is a tool for modifying thoughts and beliefs. In the short term, it is meant to target the thought–distress connection and interrupt the automaticity of this process. In the long term, the goal is belief modification, which would presumably decrease future cognitive vulnerability to insomnia. The Thought Record itself is fairly simple and enjoys many years of use as a cognitive therapy tool across multiple disorders. Although various renditions of Thought Records exist, the main components are the recording of the situation in which the troubling thought or mood occurred, the mood, and the associated problematic thoughts. The Thought Record also typically includes a column or space wherein the veracity, utility, or accuracy of the thought is challenged. Most Thought Records ask for a rerating of mood after the disputation to assess if the thought modification was associated with a mood improvement. We will discuss the type of Thought Record used in our (CEC and JDE) clinics and clinical trials. The exact record form we use is shown in Fig. 8.1 (Edinger & Carney, 2008).

Situation: This column asks for the situation in which the person noticed a troublesome mood or thought. One of the reasons for recording the situation is that sometimes there is a relationship between certain situations or settings and recurrent troubling thoughts or moods. In such cases, one can develop a preemptive plan to decrease the likelihood that these thoughts and moods might arise. For example, Mr. R monitored his thoughts, mood, and the situation in which it occurs on the first three columns of a Thought Records for 1 week (Fig. 8.1). When Mr. R returned to therapy, he discussed his Thought Records and noticed that he tended to worry about whether he was going to sleep well when he sat down to watch television at night. He noticed that it was the first time each day that his surroundings were quiet, and something about that situation made him anxious about his ensuing night's sleep. In addition to teaching Mr. R how to challenge catastrophic thoughts on the Thought Record, he began a relaxation practice at this time in the evening and noticed that he no longer had these anxious thoughts.

Mood: The mood column is used to record emotions and their intensity. The main challenge facing people when completing this column is the tendency to confuse thoughts with moods. Moods are often best described using a single word rather than a phrase. Thus, feeling "blasé" may be a mood but "I feel like I can't get anything accomplished," may be best conceptualized as a thought about feeling "blasé." This column also provides a rating of the intensity of the mood (usually 0–100%). This rating can serve

Situation	Mood (Intensity 0-100%)	Thoughts	Evidence for the thought	Evidence against the thought	Adaptive/Coping statement	Do you feel any differently?
Sitting on my couch watching evening news	Anxious (90%)	It feels like I'm going to have a panic attack. Something's wrong. I should have a beer. I need to find a way to calm down. I have a really big day tomorrow. What am I going to do? I can't keep going on like this. I'll go "crazy" if this continues. **I am never going to get to sleep.***	I have trouble sleeping when I feel anxious like this. It just feels like I won't sleep, so I won't.	It is not 100% true that I will NEVER be able to sleep. I'll definitely sleep at least a little. Just because it feels like I won't sleep doesn't mean I won't—that emotional reasoning. I feel really sleepy, so it is possible I could sleep well. There is likely a cost to telling myself that I'll never get to sleep. It could become a self-fulfilling prophesy.	Telling myself that I'll never sleep makes me feel more anxious. The truth is that I will absolutely sleep and I can't know how well. I've noticed that yoga helps with my anxiety, maybe if I do something relaxing, I'll improve my odds of sleeping well.	Anxious (40%)

Fig. 8.1 Thought Record for Mr. R

as a precognitive challenge value that can be compared to the postcognitive challenge mood rating to assess whether mood improved. In the case of Mr. R, he rated anxiety as his prominent mood and rated it quite high (90%).

Thoughts: This column is where people record their thoughts for analysis in subsequent columns of the record. Some of these thoughts are automatic and perhaps out-of-awareness. It may take some encouragement to expose what is truly occurring underneath the thought process in the situation. In cases wherein it is difficult for someone to identify many thoughts, it may prove helpful to use techniques such as the "downward arrow." Some of the questions that facilitate further exploration can include "And then what?" For example, if someone records the thought, "I am going to get sick if this continues," it may be helpful to ask "So you are concerned that you might get sick? And then what would happen? What would happen if you were to get sick?" When people examine their thoughts in this way, it often uncovers catastrophic thinking. In the example above, it would not be uncommon to uncover fears about becoming disabled, committing suicide, becoming seriously mentally ill, or finding out that insomnia is linked to a fatal condition. In the case of Mr. R, he acknowledged a fear of "going crazy." Catastrophic thoughts can be typical in those with insomnia (Harvey & Greenall, 2003), and the downward arrow technique can make these fears more explicit. Other helpful questions include: "If this thought is true, what's so bad about that?" or "What's the worst part about that?" or "What does that thought mean to you?" It is important for the person to

fully explore their thoughts on the issue without censorship, or the Thought Record will become a superficial and not particularly helpful exercise.

A common strategy when using Thought Records is to circle the "hot" thought (bolded in the case of Mr. R in Fig. 8.1); that is, the thought most tied to the intense mood in the Mood column. In some cases, there is not a thought that is most linked to the identified mood state in which case it may be that the mood has not been adequately characterized. For example, someone may choose to record angry feelings, ignoring sad or hurt feelings. Thus, the thoughts may be more linked with loss and sadness while the mood is rated as angry. The other, more likely explanation is that the thoughts recorded do not include the most troubling thought, but simply "scratch the surface" and avoid the most difficult cognitions. Asking some of the questions above can make the Thought Record more productive. In addition, there are several helpful Cognitive Therapy resources available to elicit "data-rich" thoughts on the Thought Record (Beck, 2005; Beck, 1995; Greenberger & Padesky, 1995).

Evidence for the Thought: Gathering the evidence for the thought is not included in all versions of the Thought Record. The purpose is to acknowledge the kernel of truth in the person's thoughts. It can be validating for individuals to hear that their thoughts are not ridiculous and there is a "reason" why they find the thoughts so compelling. This exercise may be helpful in combating resistance that can develop during the disputation or challenge part of the Thought Record. Thus, the evidence for the thought that "I am going to become sick" may be that a depression developed subsequent to the insomnia. It is important that evidence written in this column be factual. Thus, Mr. R's evidence that "It just feels like I won't sleep, so I won't" is an example of emotional reasoning and not factual. Socratic Questioning (that is, using questions to elicit particular responses) focused on factual evidence in this column, which can be used to challenge cognitive errors and communicate the idea that thoughts are not facts. We generally ask people to modify the piece of evidence listed in this column so that it is factual, or we ask them to consider crossing it out if it is not really an evidence. In other cases, we ask them to address the cognitive error in the evidence against the thought column.

Evidence against the Thought: This is the column most associated with Cognitive Therapy as it is the column wherein the disputation occurs. It is here wherein the veracity or the utility of the thought comes under scrutiny. Traditional Cognitive Therapy focuses on thoughts being erroneous. In some cases, clients might receive a list of Cognitive Errors along with reasons for why they are incorrect to aid them in disputing thoughts. For example, they may receive information that "Catastrophizing" is an example of a cognitive error because it overestimates the likelihood of the most extreme possible alternative occurring. Or "all or none/dichotomous thinking" is an error because it considers only two extreme outcomes and ignores the far more likely moderate "everything in between." For example, the thought that "I didn't sleep at all last night" is highly unlikely as most people sleep sometime (however, briefly) during a 24-h day. It may be that the person only slept for 2 h but claims to have not slept *at all*. This type of thinking fuels anxiety, so we ask people to modify it to something more accurate and thus more helpful.

In Mr. R's Thought Record, he addresses the emotional reasoning listed in the evidence for the thought by acknowledging that it was not a fact in the evidence against the thought column and added that just because something feels as though it is true does not necessarily mean that it is true.

There are many questions that can help with the evidence against the thought column. For example, a classic restructuring question is whether something is true 100% of the time. Mr. R acknowledged that it is not 100% certain that he would not sleep well. Another common question is to ask whether there is a particular downside to having that particular hot thought. We do not advocate focusing on "errors" exclusively as it is often more helpful to focus on the adaptiveness or cost of buying into particular thoughts instead. In Mr. R's case, we do not focus on whether it is accurate that he will have a panic attack. Arguably, we could say that he is catastrophizing by assuming that his anxiety symptoms will culminate into the worst possible outcome – a panic attack, especially since these symptoms did not actually end in a panic attack in this instance. Instead, we focus on the *cost* of focusing on whether he will have a panic attack – in his case, the cost is feeling more anxious, thus making it more likely that he could have had a panic attack. Focusing on the accuracy of thoughts can become tricky when people are accurately perceiving negative outcomes, but focusing on the adaptiveness of particular thoughts is a useful endeavor (i.e., that some thoughts are anxiety-provoking and thus not helpful).

Adaptive, balanced, coping thought: This column attempts to acknowledge the kernel of truth in the evidence for the thought while focusing on the evidence against the thought. Thus, the thought is modified into a more helpful, adaptive, and balanced cognition. We refer to it as a coping thought because we encourage people to write down the thoughts that are most helpful to consider when they are particularly distressed. Mr. R's examples are: "Telling myself that I'll never sleep makes me feel more anxious. The truth is that I will absolutely sleep and I can't know how well." Mr. R also focused on something that would be helpful in the situation: "I've noticed that yoga helps with my anxiety, so maybe if I do something relaxing, I'll improve my odds of sleeping well." We usually ask the person to rate the "believability" of each adaptive thought. When believability is low, we usually spend more time revising these statements in session until they are more credible to the person.

Post-Thought Record Mood rating: The last step is to rerate the original mood. This process acts as a postintervention point of comparison. The expectation is that there should be some mood improvement even if it is small. A lack of improvement most often means that the Thought Record columns should be revisited. Possible problems include: (1) moods and thoughts were confused; (2) the hot thought was not uncovered/explored fully; (3) the evidence against the thought was not fully explored; (4) integrating the evidence into a more adaptive thought was not done convincingly enough. Sometimes, mood ratings fall victim to all-or-none thinking, in that a negative mood is either present or 100% absent, and the person does not perceive the range of degrees within a mood. Similarly, little movement in mood can also occur in those who tend toward perfectionism. For example, the mood

change is not "good enough" to warrant recording. Thus, it is important to carefully query very low or no mood rating changes.

As stated above, the benefits of Thought Records are manifold: (1) there should be an immediate reduction in arousal (i.e., mood improvement); (2) it should challenge thinking (and beliefs) that drive sleep-related anxiety; and (3) the repeated process of completing these records impose structure on the tendency to think negatively and become distressed – that is, it breaks the sleep-disruptive mental habit. When someone has an unwanted habit, one of the first steps is to interrupt it. We rarely ask someone to simply stop the habit outright because it often occurs without prior thought/intent; it is automatic. However, if we ask someone to interrupt the process and engage in an exercise that encourages thinking about information other than the automatic thought (e.g., focusing on evidence against the thought), then we increase the likelihood that awareness may begin before the automatic sequence begins. The more this practice occurs, the more likely it is that a disturbing thought will be met with a more realistic appraisal and challenge rather than an automatic downward spiral of negative thoughts and emotions.

Decreasing Safety Behaviors and Behavioral Experiments

While it may seem unusual to place a behavioral strategy in a cognitive chapter, we have opted to include it here because of its presumed reinforcement role for beliefs (Bennett-Levy et al., 2004). The cognitive model suggests that safety behaviors play a prominent role in insomnia (Harvey, 2002). Safety behaviors have mainly been examined in the anxiety disorders literature and are conceptualized as an attempt to prevent a feared or unwanted outcome from occurring. For example, some people with insomnia might consume alcohol to avoid a lengthy time to fall asleep (Harvey, Tang, & Browning, 2005). Across Axis I disorders, safety behaviors emanate from unhelpful beliefs about the probability of a feared outcome and/ or an underestimation of one's ability to cope with an unwanted situation.

Safety behaviors are reinforced when these strategies alleviate short-term distress; however, the long-term consequence is the maintenance of beliefs that sleep-related situations and situations in which there is a performance demand are threatening and must be avoided. It also prevents contact with disconfirming evidence. In our example above, drinking alcohol as a sleep-related safety behavior may allow the person to avoid a prolonged struggle with delayed sleep onset, but it is unknown as to whether the person necessarily would have had sleep onset difficulties. In addition, drinking alcohol interferes with later sleep continuity. Preliminary studies suggest that those with insomnia engage in safety behaviors (Ree & Harvey, 2004a; Woodley & Smith, 2006). In the anxiety disorder literature, reducing safety behaviors is associated with improved outcomes (Salkovskis, Clark, Hackmann, Wells, & Gelder, 1999; Wells et al., 1995). Additionally, an open trial of Cognitive Therapy for insomnia (Harvey, Sharpley, Ree, Stinson, & Clark, 2007)

suggests that reducing safety behaviors may be an effective component of insomnia treatment. The instruction for reducing safety behaviors is simple:

1. Provide an explanation of the role of safety behaviors and avoidance in general. That is, engaging in these behaviors makes things worse in the long term and sends a message that you believe that you are incapable of coping with poor sleep or its daytime consequences.
2. Construct a list of currently operating safety behaviors. The Safety-Related Behaviors Questionnaire (Ree & Harvey, 2004b) may be helpful. Carney and Manber (2009) suggest the use of a worksheet that identifies the link between an unhelpful belief and a safety behavior (Carney & Manber, 2009). For example, for someone who consumes alcohol before bed, the assumption might be: "I cannot sleep without alcohol." Carney and Manber (2009) encourage the design of an experiment meant to test the assumption by resisting the safety behavior and tracking the experience with resistance. For example, "I will refrain from alcohol consumption this week and see if I sleep."
3. Work collaboratively to construct a plan for resisting engagement in the safety behavior.
4. Monitor successes and challenges to implementing the plan so that "cheerleading" for the successful prevention of safety behaviors can occur, or adjustments can be made if necessary.

Behavioral experiments are an important part of cognitive restructuring as they may be even more important than verbal methods (Tang & Harvey, 2006). When actigraphs were used to show people the discrepancy between how they thought they slept and how they actually slept (rather than simply telling people verbally that there was a discrepancy), there are greater cognitive shifts towards perceiving the "missing" sleep on subsequent nights (Tang, Schmidt, & Harvey, 2006). In addition to correcting sleep misperception, behavioral experiments have been used in cognitive therapy for insomnia (Harvey et al., 2007) to modify beliefs about having a limited ability to cope with fatigue or beliefs that poor sleep is dangerous (Ree & Harvey, 2004a). Harvey and colleagues (2004), for example, encouraged a 45-year-old man with insomnia to conduct an experiment that would test the idea that he needed to conserve energy because he had, in his view, a limited amount of coping resources. This man was asked to spend 3 h conserving energy (e.g., resting and avoiding anything taxing), and then 3 h generating energy (e.g., returning phone calls, going for a short walk, socializing, getting a drink). He later repeated the experiment but in reverse order and monitored the effects of the experiment on his mood and energy levels. He reported that his mood and energy levels were actually improved by expending/generating energy. Similarly, to test the idea that poor sleep is dangerous, Harvey and colleagues (2004) encouraged a 49-year-old woman with GAD to delay her bedtime and restrict her time in bed to 6.5 h. The woman was encouraged to monitor the effects of the experiment on her sleep on sleep diaries and also to monitor her coping ability, her tiredness, productivity, and mood. The woman reported that she was surprised to learn that (contrary to her prediction) she coped fairly well

and that she did not trigger poor sleep the next night. These are examples of the power that behavioral experiments can exert on cognitive change. Behavioral experiments like the ones described were part of a trial of CT for insomnia that showed efficacy for this therapy when used as a stand-alone intervention (Harvey et al., 2007).

Focus on Relapse Prevention

Whereas insomnia has not traditionally been conceptualized as a recurrent condition, evidence would suggest that it tends to be a persistent condition characterized by relapses. In a large 3-year follow-up study, insomnia was shown to have a fairly long course and to be recurrent in almost one third of those with insomnia at baseline (Morin et al., 2009). If insomnia tends to recur in those vulnerable to it, it would make sense to follow the model of other persistent disorders such as Major Depressive Disorder and incorporate a relapse prevention component to treatment. In treatment, this can be as simple as reminding people of what worked and also reminding them that they can return for a refresher appointment if needed (Edinger & Carney, 2008). Alternatively, you can design a worksheet to provide people when preparing for termination. This can be a standard document given to everyone you treat (see sample sheet), or it is often useful to devise a blank form that you construct jointly with the patient that will look like the sample provided below. The general components of a relapse prevention strategy include the following:

1. Provide guidelines for determining if insomnia has returned. This is generally not a problem for someone with insomnia. In fact, in our clinical experience, the opposite problem often occurs. That is, someone becomes alarmed about not sleeping when in actuality their sleep efficiencies and time spent awake in bed are within normal limits. Nonetheless, it is usually a productive exercise to construct a list of red flags
2. Foster a new sense of sleep self-efficacy. We know that CBT increases self-efficacy (Carney & Edinger, 2006), so it is important to remind people of their newfound sleep-related confidence. People need to form a new self-concept of themselves as good sleepers who occasionally have sleep problems that can be simply managed if/when they emerge. Termination-related conversations should foster this concept by encouraging them to remember that they mastered their sleep and can do it again in the future.

Action Plan for Addressing Insomnia

Insomnia can return, but now that you have the tools to address it, you need not worry about the possibility of its recurrence. Below we will outline some signs of chronic insomnia that alert you to the need to put the skills you learned back into place.

Red Flags for a Possible Problem with Insomnia

1. You are spending more than 30 min awake while in bed trying to sleep at least half the days. Alternatively, if you calculate your average sleep efficiency and determine it is less than 85% (i.e., on average you are sleeping for less than 85% of the time you spend in bed) or, if your sleep feels chronically nonrestorative (i.e., you wake up tired each morning and it persists throughout the day).
2. You have trouble during the day on at least half of the days of the week: For example, you feel tired, your mood is anxious/depressed/agitated/upset, you have difficulty concentrating or paying attention, and it is more difficult to function (at work, home or school).
3. You notice some uncharacteristic thinking; that is, you are preoccupied with worries about not sleeping or not being able to function the next day. This type of thinking is probably linked with feeling distressed too.

What Tools Work for This Problem?

- Restrict your time in bed to the amount of sleep you are currently producing. Do this by calculating your average amount of sleep and by adding 30 min to this average. Schedule this new amount of time in bed by picking the schedule that works best for your body and/or works best for your obligations such as work.
- Set this rise time and "earliest" bedtime 7 days per week. Adjusting these times for the weekend can produce a jetlag syndrome that will persist into the week. Be sure to set an alarm. You should go to bed no earlier than your scheduled bedtime, but you should not go to bed unless you are sleepy.
- Get out of bed whenever it is obvious you would not be able to sleep for the next 20–30 min. If you are upset, you should immediately get out bed. Do something relaxing and nonarousing until you are sleepy and then return to bed. Do not try to make up for this time you are spending out bed.
- Avoid wakeful activities in bed or the bedroom. This means that anything you would do when awake should not be done in your bed/bedroom. Sex can be an exception to this rule.
- Challenging the negative thought distress connection by completing a Thought Record.
- Complete a Behavioral Experiment that challenges whether a safety behavior is helpful in the long term.
- If anxiety is a significant problem, enact your anxiety management strategies during the day to reduce the likelihood that they will be an issue at night.
  - If the anxiety is specific to sleep, use Thought Records throughout the day to interrupt the negative thought–emotion cycle and use Constructive Worry in the evening.
  - Consider starting a daily relaxation practice particularly if it has been helpful in the past.

- Maintain healthy sleep behaviors such as refraining from caffeine, alcohol, or tobacco consumption within hours of bedtime.

If I have enacted all of these strategies and continue to have problems, I will contact my health provider and schedule a refresher session.

If I notice new sleep-related symptoms, I will contact my health provider and schedule an appointment. Such symptoms can include:

– Loud snoring
– Stopping breathing, breathing pauses, gasping or snorting during sleep
– Falling asleep unintentionally during the day
– A creepy-crawly sensation in your lower legs in the evening accompanied by an irresistible urges to move your legs to alleviate the sensation
– Very frequent leg jerking during the night
– Other unusual new experiences

Remember, you mastered the insomnia before, and you will master it again.

References

Beck, J. S. (1995). *Cognitive therapy: Basics and beyond*. New York: Guilford Press.

Beck, J. S. (2005). *Cognitive therapy for challenging problems: What to do when the basics don't work*. New York: Guilford Press.

Bennett-Levy, J., Westbrook, J., Fennell, M., Cooper, M., Rouf, K., & Hackmann, A. (2004). Behavioural experiments: Historical and conceptual underpinnings. In J. Bennett-Levy, G. Butler, M. Fennell, A. Hackman, M. Mueller, & D. Westbrook (Eds.), *Oxford guide to behavioural experiments in cognitive therapy* (pp. 1–19). Oxford: Oxford University Press.

Bootzin, R. R. (1972). *Stimulus control treatment for insomnia. Proceedings of the 80th Annual Meeting of the American Psychological Association, 7*, 395–396.

Carney, C. E., & Edinger, J. D. (2006). Identifying critical dysfunctional beliefs about sleep in primary insomnia. *Sleep, 29*(4), 440–453.

Carney, C. E., & Manber, R. (2009). *Quiet Your Mind and Get to Sleep: Solutions to insomnia for those with depression, anxiety or chronic pain*. Oakland, CA: New Harbinger.

Carney, C. E., & Waters, W. F. (2006). Effects of a structured problem-solving procedure on pre-sleep cognitive arousal in college students with insomnia. *Behavioral Sleep Medicine, 4*(1), 13–28.

Davey, G. C. L. (1994). Pathological worrying as exasercbated problem-solving. In G. C. L. Davey & F. Tallis (Eds.), *Worrying: Perspectives on theory, assessment and treatment* (pp. 35–59). New York: John Wiley and Sons.

Edinger, J. D., & Carney, C. E. (2008). *Overcoming insomnia: A cognitive behavior therapy approach therapist guide*. New York: Oxford University Press.

Edinger, J. D., Wohlgemuth, W. K., Radtke, R. A., Marsh, G. R., & Quillian, R. E. (2001). Cognitive behavioral therapy for treatment of chronic primary insomnia: A randomized controlled trial. *The Journal of the American Medical Association, 285*, 1856–1864.

Espie, C., Broomfield, N., MacMahon, K., Macphee, L., & Taylor, L. M. (2006). The attention-intention-effort pathway in the development of psychophysiologic insomnia: A theoretical review. *Sleep Medicine Reviews, 10*(4), 215–245.

Espie, C. A., & Lindsay, W. R. (1987). Cognitive strategies for the management of severe sleep maintenance insomnia: A preliminary investigation. *Behavioral Psychotherapy, 15*, 388–395.

Fichten, C. S., Libman, E., Creti, L., Amsel, R., Tagalakis, V., & Brender, W. (1998). Thoughts during awake times in older good and poor sleepers-The self-statement test:60+. *Cognitive Therapy and Research, 22*(1), 1–20.

Francis, M. E., & Pennebaker, J. W. (1992). Putting stress into words: The impact of writing on physiological, absentee, and self-reported emotional well-being measures. *American Journal of Health Promotion, 6*, 280–287.

Greenberger, D., & Padesky, C. A. (1995). *Mind over mood*. New York: The Guilford Press.

Gross, R., & Borkovec, T. (1982). Effects of cognitive intrusion manipulation on the sleep onset latency of good sleepers. *Behaviour Therapy, 13*, 112–116.

Hall, M., Buysse, D. J., Reynolds, C. F., Kupfer, D. J., & Baum, A. (1996). Stress-related intrusive thoughts disrupt sleep onset and contiguity. *Sleep Research, 25*, 163.

Harvey, A. G. (2000). Pre-sleep cognitive activity: A comparison of sleep-onset insomniacs and good sleepers. *British Journal of Clinical Psychology, 39*, 275–286.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40*, 869–893.

Harvey, A. G., & Farrell, C. (2003). The efficacy of a Pennebaker-like writing intervention for poor sleepers. *Behavioral Sleep Medicine, 1*(2), 115–123.

Harvey, A. G., & Greenall, E. (2003). Catastrophic worry in primary insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 34*, 11–23.

Harvey, A. G., Sharpley, A. L., Ree, M. J., Stinson, K., & Clark, D. M. (2007). An open trial of cognitive therapy for chronic insomnia. *Behaviour Research and Therapy, 45*(10), 2491–2501.

Harvey, A. G., Tang, N. K. Y., & Browning, L. (2005). Cognitive approaches to insomnia. *Clinical Psychology Review, 25*, 593–611.

Lichstein, K. L., & Johnson, R. S. (1993). Relaxation for insomnia and hypnotic medication use in older women. *Psychology and Aging, 8*, 103–111.

Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York: Guilford Press.

Morin, C. M., Vallières, A., Guay, B., Ivers, H., Savard, J., & Mérette, C. (2009). Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: A randomized controlled trial. *The Journal of the American Medical Association, 301*(19), 2005–2015.

Pennebaker, J. W., Kiecolt-Glaser, J., & Glaser, R. (1988). Disclosure of traumas and immune function: Health implicaitons for psychotherapy. *Journal of Consulting and Clinical Psychology, 56*, 239–245.

Platt, J. J., & Spivack, G. (1975). Unidimensionality of the Means-End Problem-Solving (MEPS) procedure. *Journal of Clinical Psychology, 31*, 15–16.

Ree, M. J., & Harvey, A. G. (2004a). Insomnia. In J. Bennett-Levy, G. Butler, M. Fennell, A. Hackman, M. Mueller, & D. Westbrook (Eds.), *Oxford guide to behavioural experiments in cognitive therapy* (pp. 287–305). Oxford: Oxford University Press.

Ree, M. J., & Harvey, A. G. (2004b). Insomnia: The development of the Sleep-related Behaviours Questionnaire (SRBQ). *Behaviour Change, 21*(1), 26–36.

Salkovskis, P. M., Clark, D. M., Hackmann, A., Wells, A., & Gelder, M. G. (1999). An experimental investigation of the role of safety-seeking behaviours in the maintenance of panic disorder with agoraphobia. *Behavior Research and Therapy, 37*(6), 559–574.

Tang, N. K. Y., & Harvey, A. G. (2006). Altering misperception of sleep in insomnia: Behavioral experiment versus verbal feedback. *Journal of Consulting and Clinical Psychology, 74*(4), 767–776.

Tang, N. K. Y., Schmidt, D. A., & Harvey, A. G. (2006). Sleeping with the enemy: Clock monitoring in the maintenance of insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 38*(1), 40–55.

Wells, A., Clark, D. M., Salkovskis, P., Ludgate, J., Hackmann, A., & Gelder, M. (1995). Social phobia: The role of in-situation safety behaviors in maintaining anxiety and negative beliefs. *Behavior Therapy, 26*(1), 153–161.

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research and Therapy, 38*, 679–693.

Woodley, J., & Smith, S. (2006). Safety behaviors and dysfunctional beliefs about sleep: Testing a cognitive model of the maintenance of insomnia. *Journal of Psychosomatic Research, 60*(6), 551–557.

Chapter 9
Other Issues in Managing the Sleep of Those with Anxiety

Abstract We have presented the core treatment strategies of CBT for insomnia in the previous chapters, but there are potential challenges unique to those suffering from comorbid anxiety problems that should be discussed. Herein, we present specific instructions/protocols for managing sleep problems in the context of anxiety and anxiety disorders, including relaxation-based strategies (focusing specifically on Progressive Muscle Relaxation), Cognitive Behavioral Treatment of Nocturnal Panic (Craske et al., Behavior Therapy 36:43–54, 2005), treating claustrophobia for those using CPAP for sleep apnea, and dream/nightmare rescripting. While a major goal of this text is that of providing practitioners guidance in the use of psychological strategies for the management of sleep problems with anxiety as a prominent feature, the problems discussed may represent only a subset of the varied forms of sleep disturbances that may present as primary or comorbid sleep disorders. Many people with such conditions require and benefit from one or more consultations with a sleep specialist. Hence, we provide discussion and a resource for use in determining whether the type of sleep problem and circumstances warrant a sleep specialty referral.

Relaxation-Based Strategies

Since cognitive and physiological arousal is a hallmark symptom of anxiety disorders in general, psychological treatments designed to reduce arousal have long been popular for the management of such conditions. In fact, as early as the 1930s, Jacobson noted the usefulness of a structured progressive muscle relaxation (PMR) exercise for reducing arousal symptoms. During the latter half of the twentieth century, various forms of relaxation therapies evolved from Jacobson's early observations and became popular for managing the arousal symptoms in the various anxiety disorders. Meta-analytic studies and systematic reviews have generally supported their efficacy for

anxiety management. These reports have indicated that the relaxation therapies are effective as either stand-alone treatments or adjunctive measures for the management of conditions such as generalized anxiety disorder, panic disorder, social anxiety, and phobic conditions (Clum, Clum, & Surls, 1993; Futterman & Shapiro, 1986; Jorm et al., 2004; Norton & Price, 2007; Siev & Chambless, 2007; Stetter & Kupper, 2002). Thus, relaxation therapy has become a staple one among the psychological treatments offered to those with anxiety problems.

As noted in Chap. 2, cognitive and physiological arousal (resulting from behavioral conditioning), tendencies to worry in bed, and sleep-related performance anxiety all are well-recognized perpetuating mechanisms for chronic insomnia. Consequently, relaxation therapies would seem an obvious treatment choice for insomnia management. In fact, relaxation approaches were among the first behavioral treatments applied to insomnia problems. Initially, there was success in treating someone with sleep-onset insomnia using a form of relaxation therapy known as autogenic training (Schultz & Luthe, 1959). A few years later, there were similar results in an insomnia case treated with progressive muscle relaxation training (Jacobson, 1964). However, not until the early 1970s were the first randomized clinical trials conducted to document the efficacy of relaxation approaches (Borkovec & Fowles, 1973; Nicassio & Bootzin, 1974). Nonetheless, these early reports were sufficient to foster substantial research and clinical interest in the use of relaxation therapies for insomnia treatment during the past several decades.

Currently, there is sufficient evidence to conclude that relaxation training is a well-established and efficacious treatment for insomnia management. Results of meta-analyses (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995), for example, suggest that relaxation therapy, in general, results in moderate to large treatment effect sizes (compared to control conditions) when common subjective sleep measures such as sleep onset latency, total sleep time, sleep quality, etc., are considered. Furthermore, the sleep improvements resulting from relaxation therapy compare favorably with those obtained with common hypnotic medications. Figure 9.1 shows findings from meta-analyses for relaxation therapy (Murtagh & Greenwood, 1995) and hypnotic medications (Nowell et al., 1997). Specifically, this figure compares the effect sizes obtained from the two types of therapies for subjective estimates of sleep onset latency (SOL), number of nocturnal awakenings (NNA), total sleep time (TST), and sleep quality. These findings suggest that hypnotic medications have modest advantages over relaxation approaches on measures of TST and NNA, but relaxation therapies produce much larger improvements in SOL and sleep quality ratings than do the medications. Moreover, it should be noted that the results shown in the Figure were derived from short-term therapy (average = seven 7 days) with hypnotics, and there are currently no data to show that brief hypnotic intervention leads to long-term sleep improvements once such medication is withdrawn. In contrast, results obtained with relaxation tend to endure well after acute treatment (i.e., therapist contact) ends. Hence, due to the durability

Fig. 9.1 Effect sizes for relaxation and hypnotic therapies
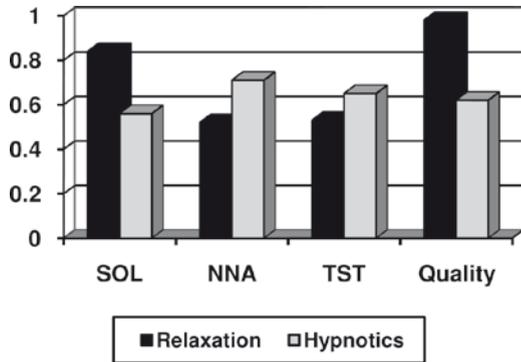
of its treatment effects, relaxation therapy may be preferred over hypnotic medications for the management of patients with chronic insomnia.

Treatment approach: Over the years, various forms of relaxation therapy have been tested for insomnia management. Included among these are techniques such as progressive muscle relaxation, passive relaxation, autogenic training, deep breathing, EMG and EEG biofeedback, various forms of meditation, and use of relaxing imagery. Head-to-head studies designed to identify the best approach generally have been absent from the literature, and cross-study comparisons have failed to suggest that one approach is a clear winner over the other approaches for treating insomnia (Murtagh & Greenwood, 1995). However, a perusal of the literature shows that progressive muscle relaxation (PMR) training has been the most commonly employed form in previous treatment studies. Given that observation, we have chosen to include instructions for that form of relaxation training in this chapter.

Both the literature and our own clinical experience suggest that a number of factors should be considered when selecting and preparing patients for this form of treatment. First, it should be noted that relaxation therapy has its most pronounced positive effects among those with sleep onset difficulties (Murtagh & Greenwood, 1995) and may be less effective as a stand-alone approach than are multicomponent CBT protocols for sleep maintenance difficulties (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001). Secondly, people with insomnia generally appear more receptive to relaxation treatment if they are provided a convincing rationale for this intervention. In this regard, a brief explanation of the role of excessive arousal as a perpetuating mechanism and the effectiveness of relaxation for reducing arousal is often useful. Finally, this treatment requires a significant amount of home-based practice between treatment sessions to achieve the treatment effects desired. For that reason, people must be relatively committed and adherent to the treatment process. Those who have demonstrated adherence difficulties to other aspects of treatment may not be the best candidates for this form of intervention.

Assuming proper candidacy for PMR, it is usually useful to introduce the therapy with the following sort of rationale:

> "To understand the reason for using the strategy we are about to discuss, it is important that you learn how a sleep problem like yours develops. Usually, insomnia problems begin as a result of stressful life events or disruption of one's usual sleep patterns. At first, an individual may ignore or minimize the sleep difficulty. However, if the stressor or disrupting agent lasts long enough, concern about the sleep disruption usually develops. This concern usually takes the form of worrying about not sleeping, becoming anxious, watching the clock, tossing and turning, etc. By repeatedly pairing these activities with attempts to sleep, the act of trying to sleep becomes associated with a heightened state of arousal that only serves to make sleep difficult to initiate. Since this "conditioned arousal" is learned over time, it can also be unlearned. One method for accomplishing this is by pairing a state of relaxation, rather than arousal, with your attempts to sleep. Over the course of this treatment you will be taught relaxation skills that will help you overcome any heightened arousal you experience when you are awake in bed. These skills should help you fall sleep more quickly on a routine basis."

This treatment rationale helps clarify the role of PMR in the management of insomnia and typically increases the likelihood that the person will both accept the approach and continue the practice outside of the therapy session.

In conducting the therapy, it is helpful to provide a therapeutic environment that is conducive to the PMR process. As noted in the classic PMR manual (Bernstein & Borkovec, 1973), people should wear sufficiently comfortable clothing so that their attire does not hinder the relaxation process. In preparing for the RT exercise, eyeglasses or jewelry should be removed that could distract during the training. Also, they should be encouraged to use the restroom prior to each training session, so that they are most comfortable when undergoing RT training. A quiet room equipped with a recliner chair is desirable for PMR sessions. It may be optimal to sit in a recliner with eyes closed and legs raised off of the floor during the delivery of instructions by the therapist. While PMR instructions are being presented, the room lighting may be dimmed if possible to facilitate the training process.

Consistent with the training protocol described by Bernstein and Borkovec (1973), PMR begins by teaching exercises involving the alternate tensing and relaxing of 16 skeletal muscle groups. Subsequently, they are taught how to combine the initial 16 muscle groups into 7 and later 4 muscle groups. Finally, the tensing portion of the exercise is dropped, and patients learn how to relax their muscles without first tensing them. The outline below shows the progression from one exercise to the next across the 6 RT treatment sessions.

PMR Treatment Outline

Session 1

Present rationale

Introduction of progressive muscle relaxation

PMR exercise – 16 muscles (tension-release)

(continued)

(continued)

Home practice instructions
Session 2
Review home practice
PMR exercise – 16 muscles (tension-release)
Home practice instructions
Session 3
Review home practice
PMR exercise – 7 muscles (tension-release)
Encourage use of RT to combat nocturnal wakefulness
Session 4
Review home practice
RT exercise – 4 muscles (tension-release)
Same as C. for Session 3
Sessions 5 and 6
Same as Session 3
RT exercise – 4 muscles (relax only)
Same as C. for Session 3

In most respects, PMR training in those with insomnia is no different from training conducted with other types of patients deemed suitable for this intervention. Although some attention to the insomnia problem is usually included in presenting treatment instructions, most of the protocol includes the standard PMR instructions that are employed with myriad anxiety and stress-induced conditions. The following script and instructional guidelines demonstrate how PMR is fashioned for insomnia sufferers:

"The exercise you are about to learn is called progressive muscle relaxation. This exercise should help you overcome the conditioned arousal you experience when you awaken during the night so that you can return to sleep more easily. Relaxation therapy is useful for a variety of stress-related conditions including tension headache, high blood pressure, general anxiety and insomnia.

During this first treatment session you will be taught an exercise that requires you to alternately tense and relax 16 major muscle groups throughout your body. In doing so, you will become sensitized to your muscle tension and better able to rid yourself of unwanted tightness. Also, by tensing each muscle group first, you will give yourself a 'running start' toward deep relaxation because the tension release will provide you some momentum toward this state.

Before beginning this exercise, you should understand that relaxation is a skill. Like any other skill such as riding a bicycle, typing, knitting or playing golf, progressive relaxation requires practice. You will, hence, be asked to practice this procedure at home every day. Only through such practice will you become able to use relaxation to combat your nocturnal awakenings. Now, before we begin the exercise, do you have any questions?

After pausing to answer any questions:

OK, now let's begin. I would like you to first make yourself comfortable in the chair by raising the footrest to a comfortable height. Now close your eyes and clear your mind so that you can concentrate only on my voice (10 sec. pause). To begin, focus all of your attention on the muscles in your dominant hand and forearm. When I tell you, you will tighten these muscles by making a tight fist. OK, tense these muscles now (pause 7 sec.) Now relax your hand and forearm by opening your fist. As you do so, study the sensations

in your hand and forearm. Notice how the tension feels different from the relaxation. Continue to relax your hand and forearm and study the sensations (after 30-40 sec of relaxation "patter" the tension [7 sec] and relaxation [30-40 sec.] is repeated with the hand and forearm).

Now that you have tensed and relaxed your hand and forearm, focus your attention on the bicep in your dominant arm. You will tense this muscle by bending your arm at the elbow and pushing your elbow against the chair. Ok tense your bicep now and study how the tension feels (pause 7 sec.). Now release the tension and notice how different the tension and relaxation feel. Also compare the relaxation you feel in your bicep with the relaxation you achieved in your hand and forearm. Continue to relax your bicep and when you feel that it is as relaxed as your hand and forearm signal me by raising the index finger of your dominant hand slightly (after 30-40 sec. of relaxation, the tension [7 sec.] and relaxation [30-40 sec.] is repeated with the bicep.)"

At this point in the exercise, the above script is repeated substituting the nondominant hand/forearm and bicep in these instructions before moving on to the following instructions involving the face muscles.

"Having tensed and relaxed the muscles in your hands and arms, we will now move on to the muscles in your face. You should begin by focusing on the muscles in your upper face. You will tense these muscles by raising your eyebrows as high as possible. OK tense these muscles now. Feel the tightness in your upper face as you do that (7 sec. pause). Now relax your upper face. Let your eyebrows drop and feel the tension releasing for those muscles. Continue to relax those muscles in your face and when they feel as relaxed as the muscles in your arms and hands signal me as before (after a 30–40 relaxation cycle the 7 sec. tension and 30-40 sec. relaxation cycle is repeated).

Now you should focus your attention on the muscles in the central part of your face. You will tense these muscles by squinting your eyes as tightly as you can and simultaneously wrinkling your nose. OK tense your central face muscles now. Notice how the tension feels as you do that. Feel the tightness (7 sec. pause). Now relax the muscles in the center of your face and notice how it feels to release the tension. Continue to relax these muscles and when they feel as relaxed as the muscles in your upper face signal me as before (after 30–40 sec relaxation phase the 7 sec. tension and 30-40 sec. relaxation cycle is repeated).

Now focus your attention on the muscles in your lower face. You will tense these muscles by biting down hard and, at the same time, pulling back the corners of your mouth. OK, tense those muscles now. Feel the tightness as you do that (7 sec. pause). Now relax those muscles in your lower face and notice the difference between tension and relaxation. Continue to relax your lower face and signal me when those muscles feel as relaxed as the other muscles in your face (after 30-40 sec. of relaxation, the tension-relaxation cycle is repeated)".

Now focus on the muscles in your neck. You will tense these muscles by pulling your chin toward your chest and at the same time keep it from touching your chest. OK tense your neck now. Notice how it feels to tense your neck (7 s). Now relax your neck. Notice how relaxation feels and how different it is from tension. As before, when your neck feels as relaxed as your face muscles, signal me with your finger (after 30–40 s of relaxation, the tension-relaxation cycle is repeated).

"We will now consider the muscles in your upper torso. You will tense these muscles by pulling your shoulder blades together. Ok, tense these muscles now. Notice how they feel when you tense them (7 sec delay). Now relax those muscles. Let go of all of the tension and notice how different that feels. As before, when your upper torso feels as relaxed

as your neck muscles, signal me with your finger (after 30-40 sec. of relaxation, the tension-relaxation cycle is repeated).

Now turn your attention to the muscles in your abdomen. You will tense these muscles by making your stomach as hard as you can make it. Notice how tight that feels as you tense your stomach (7 sec. pause). Now relax your stomach. Notice the difference between tension and relaxation as you let go of the tension in your stomach. When your stomach feels as relaxed as your upper torso, signal me with your finger (after 30-40 sec, of relaxation the tension-relaxation cycle is repeated).

Now we will focus on the muscles in your dominant leg. To begin, focus your attention on the muscles in your upper leg. You will tense these muscles by trying to straighten your leg and at the same time trying to bend your leg at the knee. Your leg should not move, but you should feel tension as a result of the opposing muscles working against each other. OK tense your upper leg now. Notice the tightness in your leg muscles as you do that (7 sec.). Now release the tension in your upper leg. Notice how different your leg feels as you relax it. Let it relax very deeply and when it feels as relaxed as your stomach signal me as before (after 30-40 sec. of relaxation patter the tension-relaxation cycle is repeated).

We will now focus on the muscles in your dominant calf. You will tense these muscles by pulling your toes toward your head. OK, tense your calf now and notice how it feels when it is tense (7 sec.). Now relax your calf and notice how different that feels from being tense. When your calf feels as relaxed as your upper leg, signal me as before (after 30-40 sec. of relaxation the tension-relaxation cycle is repeated).

Now we will move to your dominant foot. You will tense the muscles in your foot by pointing and curling your toes as you turn your foot inward. OK tense your foot now. Notice how it feels to tense your foot (7 Sec.). Now relax your foot and notice how different it feels from tensing your foot. Let it relax very deeply and when it feels as relaxed as your calf signal me as before (after 30-40 sec of relaxation the tension-relaxation cycle is repeated)".

At this point, the above three paragraphs are repeated with the nondominant upper leg, calf, and foot in sequence to conclude the exercise. After the exercise is concluded, there should be an inquiry into any difficulties encountered in following any of the instructions. Also, the patient should be asked about such common side effects as floating sensations, disorientation, muscle twitches, restlessness, etc experienced during the session. Discussing that these side effects are usually transient can provide assurance that with continued practice such side effects will subside. However, if there are any bothersome or anxiety-provoking side effects, these should be discussed at length and the therapist should decide whether PMR training should be continued. If there was a favorable PMR response during the session, it is often helpful to provide a recording of the exercise to assist home practice efforts. An actual recording of the PMR training session can be made for this purpose, or the patient can be referred to one of the many recordings that are commercially available. Whatever home aids are used, the person should be instructed to practice the initial PMR exercise one time each day at least 2 h before bedtime.

At the second PMR session, there should be an inquiry into adherence to home practice instructions. Those reporting adherence difficulties should be provided in-session assistance in problem-solving their difficulties. Specifically, those with adherence difficulties should be assisted in identifying barriers to adherence and in determining a time each day when they might most easily engage in the exercise.

Once these issues are addressed, the patient should be guided through the same 16-muscle exercise presented during session 1.

Upon returning for session 3, adherence with home practice of RT should again be reviewed. Subsequently, the patient should be presented an abridged relaxation exercise that combines the original 16 muscle groups into 7 larger muscle groupings. The PMR instructions for the new groupings are presented below. The therapist should guide through the tension and relaxation of each of these muscle groups just as was done for the 16-muscle exercise. Once again, it is usually useful to assist the at-home practice via the use of recorded instructions of this revised exercise.

Muscle group	Tension procedure
Dominant arm	Make fist/press elbow down
Nondominant arm	Make fist/press elbow down
Face	Squint, raise eyebrows, wrinkle nose, bite down, pull mouth back
Neck	Same as for 16 groups
Torso	Pull shoulders back, take deep breath, tighten stomach
Dominant leg/foot	Lift leg and curl toes
Nondominant leg/foot	Lift leg and curl toes

Once this exercise has been presented, the therapist should suggest using relaxation skills to combat nocturnal wakefulness. Specifically, the person can attempt to use their developing relaxation skills to facilitate sleep onset whenever they experience a prolonged period of wakefulness in bed.

Session 4 should be identical to session three except that the RT exercise will be reduced to the 4 major muscle groups listed below.

Four muscle groups
Right and left arms/hands
Face/neck muscles
Torso muscles
Right and left legs/feet

Sessions 5 and 6 should be identical to session 4 except the tension component of the tension-release instructions is dropped from the instructional set. In this procedure, the therapist only provides instructions to focus on each muscle group, and then to recall the feelings associated with the release of tension from that muscle group. Again, 2 presentations of each of the 4 muscle groups are conducted and each relaxation phase lasts 30–40 s.

Those who manifest excessive bedtime arousal are usually good candidates for PMR, and those who also show good treatment adherence typically receive some sleep benefits from this intervention. Generally speaking, those with the types of anxiety disorders discussed in this text may be considered good treatment candidates. However, it should be remembered that insomnia is often a complex problem that is perpetuated by a number of cognitive, physiological, and behavioral factors. Because of this fact, relaxation training may not represent an omnibus treatment for many people with insomnia. Nonetheless, there may be benefits to combining

relaxation techniques with the other approaches typically included in CBT insomnia protocols. When employing relaxation therapy as part of a complex, multicomponent insomnia intervention, it may be desirable to use an alternate, less time-intensive protocol than the PMR instructions presented here. For more information about those approaches, the reader may wish to consider a number of available texts largely or specifically devoted to the relaxation therapies (Benson, 1984; Lichstein, 1988; Smith, 1990).

Cognitive Behavioral Treatment of Nocturnal Panic

There is a validated Cognitive Behavioral Therapy treatment available for nocturnal panic that has shown some impressive improvements in those treated with CBT relative to a waitlist control group (Craske, Lang, Aikins, & Mystkowski, 2005). At posttreatment, three quarters of the CBT group reported zero nocturnal panic attacks and an absence of worry about nocturnal panic. The CBT recipients also reported decreased severity of their panic disorder and improved sleep satisfaction. Those in the CBT group evidenced less reactivity on posttreatment anxiety-inducing laboratory procedures. These improvements were maintained at a 9-month follow-up. Admittedly, additional randomized controlled clinical trials are needed to validate these findings but given the promise of this protocol, we provide an overview of it here.

Treatment Approach: The Craske protocol for nocturnal panic (Craske et al., 2005) is delivered over approximately 10–13 weeks of hourly sessions

Craske's Nocturnal Panic Protocol

Session	Content
1	Introduction to panic and anxiety
2	Physiology of anxiety
3	Hyperventilation and breathing retraining
4	Breathing retraining
	Cognitive restructuring: overestimates of danger
5	Cognitive restructuring: evaluating consequences
6	Deconditioning
	Sensation induction testing
	Identification of feared/avoided activities
7	Cognitive restructuring practice
	Deconditioning hypothesis testing
	Interoceptive exposure
8	Cognitive restructuring practice
	Deconditioning: causal analysis
	Interoceptive exposure

(continued)

Session	Content
	(continued)
9	Cognitive restructuring practice
	Deconditioning: management of intense anxiety
	Interoceptive exposure to activities
10	Cognitive restructuring practice
	Deconditioning: management of worst panic and fear of panic
	Interoceptive exposure to activities
11	Review and planning for termination
12 and 13	Include progress review sessions at 3 and 6-month posttreatment

In actuality, much of the treatment of NP is the same as that of PD, although there are some added sleep-specific psychoeducation and sleep hygiene components. Session one provides an overview of what to expect over the course of the treatment, as well as an introduction to the Cognitive Behavioral model of anxiety. Patients already have an explanation of panic as sensitivity to physical signs of fear. As a result, there is an increased attention on physical changes, such that even small changes in the body that other people would not notice are perceived. Despite this increased attention, PA sufferers may not be consciously aware that they are reacting to these small physical symptoms. Attributing negative or catastrophic meaning to these normal fluctuations increase the likelihood of attentional bias. The example in the manual is that of being in a large noisy room full of talking people. When someone mentions your name, you may detect that meaningful bit of information very clearly, above the din of the other noise (Craske et al. 2005). In the same way, physical cues can be meaningful information because of the fear that they signal panic symptoms. This same process can occur in sleep. Craske's manual provides the example of a mother perceiving sounds from her new born baby out of a sleep because of the meaning such sounds have for her. During sleep, there are a variety of normal physical fluctuations in breathing, heart rate, and muscle activity that may be perceived more easily in those predisposed to mistakenly think of these symptoms as danger cues. Panic during the night is thus conceptualized the same as panic during the day. As a result, psychoeducation focuses on the idea that physical sensations do not pose a genuine threat. The homework assignment for the first week is to monitor awareness of their experience as they wake out of sleep in a panic. This can include thoughts, physiological sensations, or imagery.

In session 2, patients are taught about the physiology of anxiety and the autonomic nervous system. They also receive a handout on sleep hygiene which details the following sleep rules: (1) stay in bed for as long as sleep is needed but no longer, (2) maintain a regular, consistent wake up time, and obtain exposure to bright light during the day; (3) engage in quiet presleep activities such as reading or taking a hot bath; (4) engage in regular late afternoon exercise; (5) maintain a comfortable sleeping environment (e.g., no extremes in temperature, no noise etc.); (6) consider a light bedtime snack (e.g., dairy or crackers); (7) limit caffeine, tobacco, or alcohol use close to bedtime. The idea behind these recommendations is to optimize sleep habits and decrease the likelihood of sleep deprivation; sleep deprivation is thought

to increase sleep-related anxiety and increase susceptibility to panic (Mellman & Uhde 1990; Roy-Byrne, 1986).

In session 3, patients are asked to undergo a voluntary hyperventilation experiment to learn about the effects of overbreathing. This sets the stage for a breathing retraining technique (BRT); a practice taught over the next few sessions. BRT implements a counting procedure with the end goal of slowing breathing to three seconds on the inhale and 3 on the exhale. Session 4 continues with BRT and also focuses on restructuring thoughts about the overestimate of danger. Starting in session 2, there is a focus on monitoring thoughts about panic, thus there is data available during session 4 for discussion. The tendency toward overestimating risk is presented as an exacerbating factor in panic. There is a careful exploration of thoughts that overestimate the likelihood of danger and a countering of the overestimation of risk. Possible techniques include asking to: (1) treat such thoughts as hypotheses for which data should be gathered to test if it is true; (2) generate a list for what alternative possibilities exist; (3) consider whether there might be an error in their assessment of risk; (4) estimate the "real" odds for something happening. Unrealistic statements are challenged because they can turn *possibilities* into certainties and thus create anxiety (Craske et al. 2005).

Session 5 continues the focus on cognitive restructuring; more specifically, the tendency toward evaluating consequences as catastrophes. A countering technique is taught, wherein the person is encouraged to critically evaluate the actual severity of the situation and personal resources for coping with the presumed "catastrophe." For example, if someone is afraid of passing out upon awakening from a panic attack, the focus may be on the fact that such an event would be unlikely, and if it were to occur, it would be "time-limited and manageable." Craske suggests that in cases wherein the "catastrophe" involves truly significant loss (e.g., death), then countering the probability overestimation (i.e., as presented in the preceding paragraph) is more appropriate. Session 6 encourages experiencing feared physical sensations (e.g., hyperventilation-related symptoms). The rationale for this strategy is that the more people experience feared sensations, the less likely it is that they will react with panic when experiencing them in the future. The therapist models a series of exercises, and then the patient is invited to repeat the same exercises. For each of the exercises, the person identifies: (1) the sensations experienced; (2) the intensity of the sensations; (3) the intensity of anxiety; and (4) the similarity of the experience to naturally occurring panic sensations.

Exercises
Shaking the head from side to side for 30 s
Running on the spot for 90 s
Holding one's breath for as long as possible
Complete body muscle tension for 1 min or holding a pushup position for as long as possible
Spinning in a chair for 1 min
Hyperventilation for 1 min
Breathing through a straw (with nostrils held together) for 2 min or breath as slowly as possible for 2 min
Focusing on a specific bodily sensation (e.g., swallowing) for 90 s

(continued)

Exercises
Focusing on a specific cognitive image (e.g., going crazy) for 90 s
Meditative relaxation (i.e., repeat a word like calm over and over) for 5 min
Quiet relaxation for several minutes interrupted by a buzzer sound

In addition, patients construct a hierarchy of feared/avoided activities (e.g., exercising, saunas, drinking hot beverages) for exposure in future sessions. Sessions 7–10 primarily focus of cognitive restructuring, deconditioning, exploration and exposure to avoided activities (from their hierarchy), and continued interoceptive exposure. The final session focuses on the review and planning for termination. While future studies are needed, Craske's treatment appears to provide an effective treatment for those with nocturnal panic.

Treating Claustrophobia Associated with Sleep Apnea Treatment

Sleep apnea is a fairly common disorder characterized by loud snoring and repeated episodes of breathing interruptions occurring in sleep. Typically, these breathing disturbances result in frequent arousals and contribute to poor sleep quality and such daytime sequelae as overwhelming sleepiness, reduced concentration, memory dysfunction, cardiopulmonary complications, and impaired occupational and social functioning. Although various treatment approaches may be considered for the management of this condition, most diagnosed with moderate or severe sleep apnea are treated with an apparatus known as a continuous positive airway pressure device – CPAP. This apparatus consists of a nasal or oral/nasal mask. The mask is attached via plastic hosing to an electric air pump designed to force air into the airway during sleep in order to eliminate sleep-related breathing disturbances. The compressed air flows into the airway and acts as a splint to hold back the tongue and open the soft tissue obstructing the airway. When CPAP is used, breathing becomes more regular, snoring stops, restful sleep is restored, and daytime symptoms are reduced or alleviated entirely.

CPAP has proven very effective for eliminating sleep-related upper airway obstruction, reducing excessive daytime somnolence (EDS), and improving cardiopulmonary function among patients with sleep apnea (Engleman & Wild, 2003; Kribbs et al., 1993; Rauscher, Popp, Wanke, & Zwick, 1991; Sanders, Gruendl, & Rogers, 1986). Unfortunately, many treated with CPAP fail to adhere to this therapy (Beecroft, Zanon, Lukic, & Hanly, 2003; Engleman & Wild, 2003; Jenkins, Mrad, & Walsh, 1991; Kribbs et al., 1993; Rauscher et al., 1991; Sanders et al., 1986). Factors most often cited as contributing to CPAP intolerance include the cost and inconvenience of the CPAP apparatus, the physical discomfort experienced from wearing the CPAP mask, dryness in the nose and throat, and, in some cases, chronic rhinitis associated with CPAP use. However, a substantial proportion of those who fail CPAP therapy report panic or claustrophobic reactions to the nasal mask

(Chasens, Pack, Maislin, Dinges, & Weaver, 2005; Means, 2002; Rolfe, Olson, & Saunders, 1991). This is particularly the case in those who otherwise have a history of claustrophobia or other severe anxiety disorders (e.g., panic disorder; PTSD). Those with claustrophobic or panic reactions to CPAP typically report a pronounced and uncomfortable sense of confinement and fears of suffocation while wearing their CPAP masks. Attempts to wear CPAP while falling asleep only heighten this anxiety and arousal, making it difficult if not impossible to fall asleep. Thus, despite the important benefits of CPAP therapy, some may decline or reject this treatment as a function of the enhanced anxiety and insomnia it causes them.

To the extent that anxiety and panic reactions to CPAP therapy actually represent "phobic" responses to wearing the CPAP mask, such reactions should be treatable with an anxiety deconditioning therapy such as desensitization or graded exposure. Initial support for this contention comes from an early case study (Edinger & Radtke, 1993), in which someone with history of claustrophobia and consequent rejection of CPAP therapy was treated with a paradigm involving gradual exposure and home-based practice with the CPAP apparatus. While this person was not using CPAP at all when treatment began, he gradually became capable of using the CPAP throughout each night's sleep by the end of treatment. Follow-up with this person showed continued CPAP use through an ensuing 6-year period. In a more recent case series study (Means & Edinger, 2007), 11 people with pronounced anxiety reactions to CPAP underwent graded exposure treatment to enhance their CPAP tolerance/adherence. As a result of intervention, 8 (72.7%) of the 11 people showed a pre-to-posttherapy increase in the number of nights they used CPAP, whereas 9 (81.8%) of the 11 showed pre-to-posttherapy increases in their hours on CPAP when they actually used this apparatus. Although only 4 achieved a predetermined desirable level of CPAP use, all but one showed improvements in either hours of CPAP use or percent of nights CPAP was used. In addition to these promising findings with adults, it is noteworthy that other investigators (Koontz, Slifer, Cataldo, & Marcus, 2003; Rains, 1995) have successfully used similar graded exposure therapies for acclimating children with significant anxiety to CPAP therapy. Thus, despite the limited number of reports attesting to its efficacy, graded exposure appears to be a promising and conceptually reasonable treatment for addressing CPAP refusals resulting from the claustrophobic and panic reactions it elicits in some people.

Treatment approach: The treatment approach presented herein is based on the strategies described in previous reports (Edinger & Radtke, 1993; Means & Edinger, 2007). The primary treatment components include a series of graded CPAP exposure exercises that are accomplished at each patient's preferred pace via homework assignments. The graded series of exposure exercises involve a gradual introduction (or reintroduction) to CPAP usage to help people slowly acclimate to CPAP and to reduce CPAP-related anxiety. The specific treatment employs a standard exposure hierarchy that can be individually tailored for each person. A sample hierarchy is provided in Table 9.1. As can be seen, the hierarchy requires acclimation to holding the CPAP mask against the face and practice breathing with

Table 9.1 Sample CPAP exposure hierarchy

1. Connect the CPAP mask to the air compressor, turn it on, and hold the mask over your nose, without strapping it to your head. Attempt to gradually increase the time you are able to tolerate breathing through the CPAP mask until you can do so for at least 15 min without anxiety.
2. Connect the CPAP mask to the air compressor, turn it on, and attach the mask to the headgear. Practice wearing the mask with the headgear while you breathe through the CPAP mask. Do this for increasing periods of time, starting with brief periods and building up to 30 min or more.
3. Connect the CPAP mask to the air compressor, turn it on, attach the mask to the headgear and put the CPAP in place on your face with the headgear. Now practice taking short daytime naps with the CPAP in place. Start with brief naps of 15 min or so and increase the time up to 1 h as you feel able to do so.
4. Begin wearing CPAP at nighttime as originally prescribed. You may first try to wear it for a portion of the night and then gradually increase your time on CPAP until you can tolerate it for your full night of sleep

it without attaching it to the headgear and actually wearing the mask. Once the person is comfortable with this step and can tolerate breathing with the CPAP in this way for extended periods, the next step (i.e., practice breathing with the CPAP while wearing the CPAP mask with the headgear) is introduced. Once the first two steps of this hierarchy are mastered while awake, the next step involving using CPAP during a brief daytime nap is introduced. After they are able to successfully nap with CPAP for up to 1 h without anxiety or other difficulties, they are encouraged to begin using CPAP during the main sleep period at night time. Since patients implement the graded exposure regimen independently in their homes, they are able to progress at their own rates and may discontinue each practice session whenever they choose to do so. These features provide patients a sense of control and safety as treatment progresses and minimize chances for strong anxiety reactions that might confound the treatment process.

Before beginning this treatment, it is useful to assess readiness and motivation for treatment. Given the amount of "homework" involved in this therapy, those who are not particularly interested in pursuing such treatment are generally not good treatment candidates. However, because sleep apnea may have serious long-term medical sequelae (cardiac disorders, increased risks for stroke, etc.) interventions such a motivational interviewing (Aloia, Arndt, Riggs, Hecht, & Borrelli, 2004) may be a necessary prelude to graded CPAP exposure. It is also important to assess comfort with the type of CPAP equipment he/she is using. For example, some may report that their CPAP mask does not fit properly or is otherwise uncomfortable. Others may complain that the CPAP air is too dry and irritates their airways. It is important to know that such complaints can be addressed by providing choices from the myriad of CPAP masks available on the market and by attaching a humidifier unit to the CPAP air compressor. Such ancillary supplies can usually be obtained from home healthcare companies that deliver the CPAP machine, so collaborating with this vendor to resolve these issues can be beneficial to the overall CPAP adherence intervention.

When implementing the graded exposure treatment, it is useful to provide a general rationale for the treatment. Most understand that gradual exposure to feared situations is an effective means of becoming more comfortable in such situations, so describing CPAP exposure therapy in this context usually is helpful. We have found it useful to present such rationale before introducing the specific treatment instructions outlined above and in Table 9.2. Some are able to accept the treatment rationale and follow through on the treatment instructions fairly independently and seemingly require just one therapy visit. However, many if not most benefit from one or more follow-up visits spaced at several week intervals from the initial visit. During follow-up sessions, it is useful to consider subjective reports of increasing comfort with CPAP. However, it is equally useful to monitor actual CPAP usage when possible particularly during the latter stages of this treatment. Most current CPAP devices have internal monitors that record actual time of usage, thus providing objective adherence information. Although most readers will not likely have the means to access these data, it may

Table 9.2 Symptoms and characteristics of sleep disorders that warrant sleep specialty referral

Sleep disorder	Common symptoms and characteristics[a]
Sleep apnea	Loud snoring
	Observed breathing pauses during sleep/or
	Gasping or choking in sleep
	Excessive daytime sleepiness
	Unrefreshing nighttime sleep and daytime naps
	Awakening with a headache and/or dry mouth
	Medical history may include hypertension or cardiac arrhythmia
	Obesity is a common characteristic
	Middle aged men most prone
	Women more prone after menopause
Narcolepsy	Excessive daytime sleepiness
	Periods of sudden muscular weakness coupled to strong emotion
	Frightening images (hallucinations) at sleep onset or offset
	Awakening from sleep with temporary paralysis
	Insomnia complaints may or may not be present
Restless legs syndrome	Irresistible urges to move the legs
	Urges to move the legs often result from annoying sensations in the legs such as crawling, tingling, drawing, or electric sensations
	Symptoms more pronounced in the late afternoon and evening hours
	Movement of legs or walking provides momentary relief
Night terrors	Sudden episodes of terror during sleep beginning with a loud cry or scream
	Autonomic and behavioral manifestations of fear usually present during event
	It is difficult to awaken the individual during the event
	The individual is confused upon awakening from the event
	There is often amnesia for the event on the following morning
	Dangerous or self-injurious behaviors may occur during the event
	Most such events occur during the first half of the sleep episode

[a]Symptoms and characteristics extracted from the International Classifications of Sleep Disorders, 2nd Edition: Diagnostic and Coding Manual (American Academy of Sleep Medicine, 2005)

be useful to collaborate with the sleep laboratories or home healthcare companies that respectively initially prescribe or set-up the CPAP unit. Usually, one or both of these entities can access and print out reports summarizing the objective adherence data. These data are particularly important for monitoring treatment outcomes since patients often tend to over-report their CPAP usage (Grunstein & Sullivan, 2000). Such data help corroborate the patients' self-reports and aid in determining when treatment can be terminated. Whereas no dose-response studies have been conducted to ascertain the ideal number of follow-up sessions for this therapy, our clinical experience suggests that those who respond to this therapy usually do so with one to four follow-up sessions spaced at several week intervals.

Dream/Nightmare Rescripting

Nightmares, defined as disturbing dreams that awaken the sleeper, are emotionally upsetting, sleep-disruptive phenomena that are highly pervasive in the general population. Indeed, the lifetime prevalence of a nightmare experience likely approaches 100% (Nielsen & Zadra, 2005). For most individuals, nightmares are no more than an occasional nuisance that have no clinically significant effects on their overall sleep patterns or daytime functioning. However, 2–5% of younger adults and 1–2% of older adults in the general population regard nightmares as a "current problem" (Partinen, 1994). Whereas only about 4% of those patients seen in general medical practices present nightmare complaints (Bixler, Kales, & Soldatos, 1979), nightmares are commonly reported by psychiatric patients including those with depression (Cartwright, Young, Mercer, & Bears, 1998), schizophrenia (Levin, 1998), substance abuse issues (Cernovsky, 1985, 1986), and those who seek psychiatric care through emergency room visits (Brylowski, 1990). Arguably, among the more affected groups are those with posttraumatic stress disorders who display nightmare prevalence rates as high as 50–88% (Forbes, Phelps, & McHugh, 2001; Kilpatrick et al., 1998; Neylan et al., 1998; Schreuder, van Egmond, Kleijn, & Visser, 1998). Thus, nightmare complaints are relatively common among the general population at large and especially among those with anxiety and other disorders.

The morbidity associated with nightmares can vary depending upon their frequency, intensity, and the level of sleep disturbance and daytime anxiety they cause. In milder forms, nightmares may cause occasional sleep disruption and lingering anxiety or fear. In more protracted cases, nightmares may contribute to significant psychological distress and sleep impairment (Berquier & Ashton, 1992; Kales, Soldatos, & Caldwell, 1980; Krakow, Tandberg, Scriggins, & Barey, 1995; Zadra & Dondri, 2000). Nightmares may be viewed as exacerbating phenomena that contribute to the overall perceived severity of such comorbid conditions as anxiety disorders, depression, and PTSD (Berquier & Ashton, 1992; Kales et al., 1980; Krakow et al., 2002; Zadra & Dondri, 2000). Often, learned sleep-preventing associations develop among nightmare sufferers. For example, frequent and recurent nightmares may lead to fear of the bed/bedroom, fear of going to sleep initially, or a fear of returning to sleep following a

nightmare-induced awakening (Krakow et al., 2000). These fears in turn may lead to sleep-disruptive practices such as keeping the TV and/or bedroom lights on throughout the night or altering sleep schedules to reduce nightmare opportunities. Anecdotal observations of combat veterans with PTSD often show such patterns as markedly curtailing nighttime sleep and napping in the afternoon when dream and nightmare propensity is reduced. When sleep becomes chronically disrupted by such practices, notable ongoing daytime impairment including reduced concentration, excessive sleepiness/fatigue, enhanced irritability, and mood disturbance may emerge. In turn, the individual may suffer marked social and occupational functioning as a result. Thus, nightmares often represent a clinically significant problem that merits effective management strategies.

Traditional views of nightmares posit that such phenomena are uncontrollable processes emerging from the unconscious mind and represent unresolved conflicts (Hartmann, 1984; Lansky, 1995; Mack, 1974) or unfinished business (Foa, Riggs, Dancu, & Rothmaum, 1993) related to stressful or traumatic experiences. Although this view implies that psychotherapy designed to produce conflict resolution should be an effective nightmare therapy, this treatment approach currently has little empirical support (Nielsen & Zadra, 2005). Since nightmares commonly occur in the context of comorbid psychiatric conditions such as PTSD, some (Mack, 1974) have argued that nightmares are merely secondary symptoms of a primary psychiatric disorder. Given this view, it would be expected that effective treatment of the primary comorbid condition presumed to cause the nightmare(s) would resolve the nightmare problem. However, at least one report showed that a substantial number of PTSD patients continue to suffer from ongoing residual nightmares even after completing an intensive 3-month group program for their condition (Forbes, Creamer, & Biddle, 2001). Hence, it is not apparent that these traditional conceptualizations of nightmares have led to effective therapies for their amelioration.

Over the past several decades, clinicians and researchers have increasingly adopted the view that nightmares are learned phenomena that merit their own specific therapies. From this viewpoint, nightmares that emerge immediately following a trauma or stressful event may initially serve a useful role by allowing for helpful emotional processing and/or motivate the individual to alter behavior so as to stay out of harm's way (Krakow & Zadra, 2006). Those who show a proper balance in their thoughts, emotions, and imagery surrounding the trauma are thought to recover most effectively from traumatic events. However, some individuals may tend to "think too much and spend less time with their feelings and images because the latter are more unpleasant and less manageable" (Krakow & Zadra, 2006). In such individuals, nightmares persist over time as a habit and replay over and over much like a broken record (Krakow & Zadra, 2006). Thus, nightmares related to trauma can become a habit (Krakow, Hollifield et al., 2001). In such cases, the nightmare persists as a noxious conditioned stimulus leading to such conditioned responses as awakenings from bad dreams to reduce or avoid unpleasant affect (Krakow, Johnston et al., 2001). Inasmuch as the awakenings allow for "escape" from the noxious nightmares, such arousals are reinforced by the momentary anxiety reduction they produce. This process, in turn, may contribute

to unhelpful beliefs about sleep (i.e., sleeping leads to nightmares so sleep is not safe) and the above-mentioned sleep safety and avoidance behaviors to minimize nightmare exposure.

Given the possibility that nightmares and bad dreams are learned phenomena, investigators have increasingly turned to the use of cognitive-behavioral interventions that specifically target these phenomena themselves. A few early studies demonstrated that systematic desensitization, with or without concurrent relaxation training, is effective for reducing nightmare frequency (Cellucci & Lawrence, 1978; Miller & DiPilato, 1983). However, seemingly the most popular and arguably promising nightmare therapy has been the treatment known as imagery rehearsal therapy (IRT). Although, several renditions of IRT have been proposed (Krakow & Zadra, 2006; Neidhardt, Krakow, Kellner, & Pathak, 1992; Rybarczyk et al., 2005) all of these include instructions to re-script disturbing dreams/nightmares using more acceptable storylines and to rehearse the rescripted dreams via imagery during scheduled daytime rehearsal sessions. The role of nightmare rehearsal has been touted as the core therapeutic element since it results in exposure, abreaction, and mastery of disturbing dream content (Marks, 1978). However, it has also been argued that exposure and abreaction are less important to therapeutic outcome than is the sense of mastery over the dream process (Bishay, 1985). In either case, IRT challenges the common view that dreams cannot be controlled/altered and offers nightmare sufferers the option to influence the dreams they experience.

Since the early 1990s, a number of case reports as well as uncontrolled and controlled clinical trials that support the efficacy of IRT have been published. Perhaps, the largest and best controlled study was the randomized clinical trial by Krakow, Hollifield et al. (2001), in which female sexual assault victims with nightmares were randomized to IRT or a wait list condition. Results of this trial showed that those receiving IRT had significantly greater improvements on measures of nightmare frequency, overall sleep quality, and PTSD symptoms in general. Other case series and uncontrolled trials have supported the efficacy of this therapy with such groups as adult crime victims (Krakow, Johnston et al., 2001), combat veterans (Forbes et al., 2003; Forbes, Phelps et al., 2001), adults with poly-trauma histories (Rybarczyk et al., 2005), and victims of physical or sexual abuse during childhood or adolescence (Krakow, Sandoval et al., 2001). In addition, one case series report suggests that IRT is effective for reducing nightmares and improving sleep among adults with idiopathic nightmares and no associated trauma histories. Although the literature would benefit by more randomized controlled trials testing this therapy, the available data suggest that IRT is a viable therapy that holds much promise for assisting nightmare sufferers.

Treatment protocol: To date, most of the information concerning IRT implementation comes from the work of Krakow and colleagues (Krakow, Hollifield et al., 2001; Krakow, Kellner, Pathak & Lambert, 1995; Krakow & Zadra, 2006; Neidhardt et al., 1992). Much of what has been published by this group describes the delivery of IRT in a group therapy format although Krakow and Zadra (2006) suggest that treatment delivery via individual therapy format may be similarly effective. Treatment duration

has varied from abridged one-session formats (Neidhardt et al., 1992) to full day (i.e. 6 h) workshops (Krakow, Sandoval et al., 2001) and four, 2-hour group therapy sessions (Krakow & Zadra, 2006). The abridged treatment model includes instructions to: (1) write out a targeted troublesome nightmare in exacting detail; (2) change the storyline of the nightmare (usually to include a more positive theme); and (3) engage in imagery rehearsal of the rescripted nightmare on a daily basis. Specific rescripting instructions have consisted of changing the nightmare "anyway you wish" or "change the ending of the nightmare" and both approaches appear to work equally well (Krakow, Kellner et al., 1995). The unabridged versions of this treatment include rescripting strategies as well as extensive preparatory psychoeducation concerning the role of nightmares in emotional processing and self protection, the concept of chronic nightmares as a learned albeit unwanted habits, the role of nightmares in contributing to insomnia, and the general rationale for treating nightmares directly (see Krakow and Zadra, 2006 for more details). The more intensive renditions of Krakow's treatment approach also typically include guided imagery training to enhance the imagery skills of those with nightmares. Central to the approach of Krakow and colleagues is the avoidance of all discussion of past traumatic events or traumatic content of current nightmares. This approach is taken to enhance the sense of safety among group participants who may feel too uneasy to discuss such information in a group setting. Instead, participants are instructed to engage in all nightmare rescripting and rehearsal in their homes between sessions so as to minimize and control exposure to trauma phenomena and enhance mastery and self-control themes pertinent to the therapy process.

There have been questions about the utility of minimizing the exposure component of IRT (Rybarczyk et al., 2005) given the documented efficacy of exposure techniques for reducing PTSD symptoms (Connor et al., 2000). Given this observation, the suggestion has been for an altered IRT protocol that combines exposure, relaxation, and rescripting strategies to treat nightmares. Accordingly, this so-called ERRT protocol employs a group therapy format and requires patients to write out a detailed nightmare description, read it to the group, and engage in-group discussion about the specific trauma themes the nightmare includes. Like IRT, ERRT asks people to rescript their nightmares in a favorable manner incorporating the original trauma themes. However, this rescripting is also completed within the group and the rewritten nightmares are shared with group members as well. Homework subsequently includes imagery rehearsal with the rescripted nightmare followed by completion of a relaxation exercise. Unlike IRT, the ERRT protocol suggests completion of the imagery rehearsal and subsequent relaxation just prior to retiring to bed. Although the inclusion of the exposure and relaxation components in ERRT seems reasonable, it remains unclear whether people with nightmares will be as accepting of ERRT as they seemingly have been of the original IRT approach. Moreover, there have not yet been any direct comparisons of the IRT and ERRT protocols, so it remains unknown whether the latter approach is any more effective than the former one.

From the authors' clinical experience, it does appear useful to provide some preliminary psychoeducation that reviews that dream content is learned from

daytime experiences, and often disturbing dreams/nightmares reiterate negative or stressful life events. It is usually useful to ask if they can recall some dreams or nightmares that include some daytime experiences or concerns they have had. Usually they can, and reflection on those provides them some evidence that learning does play a role in the development of bad dreams and nightmares. Given this realization, people are usually more open to the interpretation that recurrent bad dreams/nightmares are bad mental habits that need to be altered through specific targeted treatment efforts on their parts. It is also useful to review and challenge the belief that dreams are random and uncontrollable by suggesting that the strategies of IRT are specifically designed to directly influence dream content, and thus provide eventual mastery over dream experiences. Once open to this idea, the actual strategy of altering dream content by rewriting and rehearsing a selected disturbing dream can be introduced. Krakow et al. have suggested starting with a less threatening nightmare when first employing these strategies. However, in our experience, some reasonably motivated people seem to be able and eager to begin with a relatively distressing and persistent nightmare. Hence, the level of motivation and comfort with addressing the most distressing and persistent nightmares should be assessed at the start of this therapy so as to better gear the pace of treatment to each individual.

As noted by Krakow et al., people can be given instructions to "change the dream anyway you prefer" or "change the ending of the dream." Whichever of these two instructional sets is used, the person should be encouraged to practice the rescripted dream at least one time each day. There are no current data to suggest how long this practice should last, but we have had success with recommending a minimum of 20 min per day. The rate at which the improvements occur can vary. Some show fairly rapid improvement, whereas others show more gradual reductions in nightmare severity and frequency. From clinical observations, we speculate that factors such as the nature of the bad dream/nightmare, nature of the original causative experiences (e.g., trauma severity), and diligence in adhering to treatment instructions are among the more important factors in predicting the pace of treatment and eventual outcome. Those who benefit from treatment usually achieve a subjective endpoint at which they report a markedly reduced frequency and intensity of the nightmares. Simply put, they report that they are having the nightmare much less frequently and that when they do have it, it does not bother them greatly. While it is useful to track global subjective appraisals through the course of IRT, we also find it useful to prospectively monitor nightmare activity by use of a nightmare log. When circumstances permit, it is useful to acquire "baseline" data concerning nightmare frequency over two to four weeks and then use these data as reference points. Comparison of these baseline logs with subsequent logs acquired during and after treatment provides useful information about the degree to which nightmare frequency and intensity change from treatment.

The following case example demonstrates the application of the core IRT methods for addressing nightmares in a military veteran.

Mr. C was a 57-year-old self-employed man with a long history of nightmares and sleep disturbance since his Vietnam War experiences. He reported a recurrent

disturbing dream, in which he relieved a traumatic combat related event. The event in question involved his military unit being subjected to repeated mortar fire while attempting to unload a supply truck. Mr. C recalls hearing the sound of the mortar fire and realizing from the sound that their supply truck was about to be hit. He recalls yelling out in efforts to warn his fellow soldiers, but the warning came too late for some of his comrades. A mortar shell hit the supply truck and resulted in a fiery explosion that led to many of his comrades being killed. Although he and several of his comrades successfully escaped the explosion without injuries, Mr. C was bothered by intense remorse over his inability to warn everyone in time. Upon his presentation for treatment, he reported a frequent (weekly) occurrence of a nightmare that essentially replayed this very frightening and troubling event in his life.

As treatment for his condition, Mr. C agreed to a trial of IRT. He was, thus, instructed to: (1) write out his nightmare in exacting detail, the next time it occurred; (2) to rewrite the nightmare with a new and more desirable ending, and (3) to rehearse the rescripted nightmare via use of visual imagery at least 20 min each day. He followed these instructions and altered the script of his nightmare to include a new ending, in which his efforts to warn others were officially recognized and commended by his commanding officers in a manner that suggested he did everything he was humanly able to do during the mortar attack to help his comrades. Mr. C practiced this revise dream at home and was seen for check-in sessions by the therapist every 3–4 weeks over the course of a 3-month period. He eventually reported that his nightmare frequency and intensity diminished appreciably, and he was satisfied with his outcome. He subsequently requested and was scheduled for a follow-up visit so that he could "check in" with the therapist. Mr. C's follow-up visit unexpectedly occurred about 1 month after the 9/11/01 terrorist attack on the World Trade Center and Pentagon. Nonetheless, Mr. C reported no relapse in his symptoms, and he continued to be satisfied with his status at the time of follow-up.

While not all will respond dramatically to IRT, this treatment does offer benefit to many who are able to adhere to it.

Is it Time for a Sleep Specialist Consultation?

A major goal of this text is that of providing practitioners guidance in the use of psychological/behavioral strategies for the management of insomnia and other sleep-related complaints common among patients with anxiety disorders. Inasmuch as the range of strategies discussed herein are all evidence-based, these techniques collectively should provide the mental health practitioner a sizable armamentarium of approaches for managing patients presenting with comorbid anxiety and sleep-related complaints. Nonetheless, the problems discussed represent only a subset of the myriad forms of sleep disturbances that may present as primary or comorbid sleep disorders. Many people with such conditions require and benefit from one or more consultations with a sleep specialist. Hence, it is useful to consider the types of sleep problems and circumstances that would warrant a sleep specialty referral.

Various people with primary sleep disorders present with symptoms that can be confused with symptoms of an anxiety or insomnia disorders. For example, some with sleep-disordered breathing (e.g., sleep apnea) report awakening gasping for breath. Since it is not uncommon for such people to report feeling anxious and sensing rapid heart rate upon such awakenings, it is easy to misinterpret such symptoms as indicative of nocturnal panic. Furthermore, many people with sleep apnea or narcolepsy may present complaining of poor quality or fragmented nocturnal sleep that they view as contributory to their daytime sleepiness. In some of the more persuasive patients of this type, there is a risk of attributing the noted daytime symptoms to the purported insomnia problem. Other people who suffer from restless legs syndrome may present primarily with sleep onset complaints resulting from their feeling restless and fidgety when in bed at night. While it may seem reasonable to assume that the person is suffering from sleep-related performance anxiety and conditional arousal to the bed and bedroom, such symptoms may actually have a neurologic basis. Finally, some people who report that they often awaken screaming with a pronounced sense of fear may be easily misdiagnosed as having nocturnal panic or a nightmare disorder when, in fact, they suffer from a condition known as night terrors.

It is important to be able to identify those with such disorders since these individuals most often require the attention of a certified sleep specialist for proper management. Those with suspected sleep apnea and narcolepsy as well as many individuals who suffer from night terrors require specific forms of diagnostic sleep monitoring that are conducted mainly in a sleep disorders center. Such diagnostic testing is essential in such cases to correctly diagnose these conditions and gauge their severities. Moreover, sleep specialists present in such centers are much more likely to understand the specific treatment needs of these patients than are general practitioners. Although those with restless legs syndrome usually do not require specialized diagnostic sleep monitoring, they often are difficult to manage and benefit from the knowledge of a sleep specialist to arrive at the most proper and effective management strategy. Because the types of patients mentioned should be considered for referral to a sleep specialist, it is useful to have a ready reference that can be used to help identify those with one or more of these conditions. Thus, Table 9.2, which lists the defining symptoms of each of these disorders, is provided as a reference guide to aid in the identification of those who should be considered for a sleep specialty consultation.

Of course, reliance on the information in this table will not result in identification of all those who may warrant the diagnostic or treatment services a sleep specialist can offer. In some instances, people may present with difficult to discern occult primary sleep disorders. Many such cases may have such occult conditions as a cause of or in addition to their more obvious insomnia symptoms. They may present the previously described cognitive and behavioral targets that make them appropriate candidates for a trial of the evidence-based insomnia therapy strategies discussed herein. However, when patients show a poor or marginal response to a trial of the cognitive and behavioral techniques described in this text, they may merit referral to a sleep specialist for further diagnostic evaluation. This is particularly the case when treatment adherence appears good and significant sleep/wake

symptoms persist. In fact, current practice parameters of the American Academy of Sleep Medicine suggest that referral for a diagnostic sleep laboratory study should be considered for insomnia patients when they fail their first line treatment (Littner et al., 2003). Many such cases sufferer from underlying subtle, albeit clinically significant, sleep-disordered breathing, or other medical/neurological sleep-specific disorders than can only be detected via sleep monitoring. Thus, assuming treatment adherence is satisfactory, nonresponse to the insomnia therapies described in this text also may serve as adequate rationale for sleep specialist consultation.

References

Aloia, M., Arnedt, J., Riggs, R., Hecht, J., & Borrelli, B. (2004). Clinical management of poor adherence to CPAP: Motivational enhancement. *Behavioral Sleep Medicine, 2*, 205–222.

Beecroft, J., Zanon, S., Lukic, D., & Hanly, P. (2003). Oral continuous positive airway pressure for sleep apnea: Effectiveness, patient preference, and adherence. *Chest, 124*, 2200–2208.

Benson, H. (1984). *Beyond the relaxation response: How to harness the healing power of your personal beliefs*. New York: Berkley Books.

Bernstein, D., & Borkovec, T. D. (1973). *Progressive relaxation training*. Champaign, Ill: Research Press.

Berquier, A., & Ashton, R. (1992). Characteristics of the frequent nightmare sufferer. *Journal of Abnormal Psychology, 101*, 246–250.

Bishay, N. (1985). Therapeutic manipulation of nightmares and the management of neuroses. *British Journal of Psychaitry, 147*, 67–70.

Bixler, E., Kales, A., & Soldatos, C. (1979). Sleep disorder encountered in medical practice: A national survey. *Behavioral Medicine, 6*, 13–21.

Borkovec, T. D., & Fowles, D. C. (1973). Controlled investigation of the effects of progressive relaxation and hypnotic relaxation on insomnia. *Journal of Abnormal Psychology, 82*, 153–158.

Brylowski, A. (1990). Nightmares in crisis: Clinical applications of lucid dreaming techniques. *Psychiatry Journal of the University of Ottawa, 15*, 79–84.

Cartwright, R., Young, M., Mercer, P., & Bears, M. (1998). Role of REM sleep and dream variables in prediction of remission from depression. *Psychiatry Research, 80*, 249–255.

Cellucci, A., & Lawrence, P. (1978). The efficacy of systematic desensitization in reducing nightmares. *Journal of Behavior Therapy and Experimental Psychiatry, 9*, 109–114.

Cernovsky, Z. (1985). MMPI and nightmares in male alcoholics. *Perceptual and Motor Skills, 61*, 841–842.

Cernovsky, Z. (1986). MMPI and nightmares reports in women addicted to alcohol and other drugs. *Perceptual and Motor Skills, 62*, 717–718.

Chasens, E. R., Pack, A., Maislin, G., Dinges, D., & Weaver, T. (2005). Claustrophobia and adherence to CPAP treatment. *Western Journal of Nursing Research, 27*, 307–321.

Clum, G., Clum, G., & Surls, R. (1993). A meta-analysis of treatments for panic disorder. *Journal of Consulting and Clinical Psychology, 61*(2), 317–326.

Connor, K. M., Davidson, J. R., Churchill, L. E., Sherwood, A., Foa, E. B., & Weisler, R. H. (2000). Psychometric properties of the Social Phobia Inventory (SPIN): A new self-rating scale. *British Journal of Psychiatry, 176*, 379–386.

Craske, M. G., Lang, A. J., Aikins, D., & Mystkowski, J. L. (2005). Cognitive behavioral therapy for nocturnal panic. *Behavior Therapy, 36*, 43–54.

Edinger, J., Wohlgemuth, W., Radtke, R., Marsh, G., & Quillian, R. (2001). Cognitive behavioral therapy for treatment of chronic Primary Insomnia: A randomized controlled trial. *The Journal of the American Medical Association, 285*, 1856–1864.

Edinger, J. D., & Radtke, R. A. (1993). Use of in vivo desensitization to treat a patient's claustro-phobic response to nasal CPAP. *Sleep, 16*(7), 678–680.

Engleman, H., & Wild, M. (2003). Improving CPAP use by patients with the sleep apnoea/hypopnoea syndrome (SAHS). *Sleep Medicine Reviews, 7*, 81–99.

Foa, E., Riggs, D., Dancu, C., & Rothbaum, B. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress, 6*, 459–473.

Forbes, D., Creamer, M., & Biddle, D. (2001). The validity of the PTSD checklist as a measurs of symptomatic change in combat-related PTSD. *Behaviour Research and Therapy, 39*, 977–986.

Forbes, D., Phelps, A., McHugh, A., Debenham, P., Hopwood, M., & Creamer, M. (2003). Imagery rehearsal in the treatment of posttraumatic nightmares in Australian Veterans with chronic combat-related PTSD: 12-month follow-up data. *Journal of Traumatic Stress, 16*(5), 509–513.

Forbes, D., Phelps, A., & McHugh, T. (2001). Treatment of combat-related nightmares using imagery rehearsal therapy: A pilot study. *Journal of Traumatic Stress, 14*(2), 433–442.

Futterman, A., & Shapiro, D. (1986). A review of biofeedback for mental disorders. *Hospital and Community Psychiatry, 37*(1), 27–33.

Grunstein, R., & Sullivan, C. (2000). Continuous positive airway pressure for slep breathing disorders. In M. Kryger, T. Roth, & W. Dement (Eds.), *Principles and practice of sleep medicine* (3rd ed., pp. 894–912). Philadelphia: W. B. Saunders Company.

Hartmann, E. (1984). *The nightmare: The psychology and biology of terrifying drams*. New York: Basic Books.

Jacobson, E. (1964). *Anxiety and tension control*. Philadelphia: Lippincott.

Jenkins, N., Mrad, R., & Walsh, J. (1991). Long term nasal CPAP use and follow-up care. *Sleep Research, 20*, 56.

Jorm, A., Christensen, H., Griffiths, K., Parslow, R., Rodgers, B., & Blewitt, K. (2004). Effectiveness of complementary and self-help treatments for anxiety disorders. *Medical Journal of Australia, 181*(7 suppl.), S29–S46.

Kales, A., Soldatos, C., & Caldwell, A. B. (1980). Nightmares: Clinical characteristics and personality patterns. *American Journal of Psychiatry, 137*, 1197–1201.

Kilpatrick, D., Resnick, H., Freedy, J., Pelcovitz, D., Resick, P., & Roth, S. (1998). Posttraumatic stress disorder field trial: evaluation of the PTSD construct-criteria A through E. In T. Widiger & A. Frances (Eds.), *DSM-IV sourcebook*. Washington, DC: American Psychiatric Press.

Koontz, K. L., Slifer, K. J., Cataldo, M. D., & Marcus, C. L. (2003). Improving pediatric compliance with positive airway pressure therapy: The impact of behavioral intervention. *Sleep, 26*, 1010–1015.

Krakow, B., Hollifield, M., Johnson, L., Koss, M., Schrader, R., Warner, T., et al. (2001). Imagery rehearsal therapy for chronic nightmares in sexual assault survivors with posttraumatic stress disorder: A randomized controlled trial. *The Journal of the American Medical Association, 286*, 537–545.

Krakow, B., Hollifield, M., Schrader, R., Koss, M., Tandberg, D., Lauriello, J., et al. (2000). A controlled study of imargery rehearsal for chronic nightmares in sexual assault survirors with PTSD: A preliminary report. *Journal of Traumatic Stress, 13*(4), 589–609.

Krakow, B., Johnston, L., Melendrez, D., Hollifield, M., Warner, T., Chavez-Kennedy, D., et al. (2001). An open-label trial of evidence-based cognitive behavior therapy for nightmares and insomnia in crime victims with PTSD. *American Journal of Psychiatry, 158*(12), 2043–2047.

Krakow, B., Kellner, R., Pathak, D., & Lambert, L. (1995). Imagery rehearsal treatment for chronic nightmares. *Behaviour Research and Therapy, 33*(7), 837–843.

Krakow, B., Sandoval, D., Schrader, R., Keuhne, B., McBride, L., Yau, C., et al. (2001). Treatment of chronic nightmares in adjudicated adolescent girls in a residential facility. *Journal of Adolescent Health, 29*, 94–100.

Krakow, B., Schrader, R., Tandberg, D., Hollifield, M., Koss, M., & Yau, C. (2002). Nightmare frequency in sexual assault survivors with PTSD. *Journal of Anxiety Disorders, 16*, 175–190.

Krakow, B., Tandberg, D., Scriggins, L., & Barey, M. (1995). A controlled comparison of self-rated sleep complaints in aute and chronic nightmare sufferers. *Journal of Nervous and Mental Disease, 183*, 623–627.

Krakow, B., & Zadra, A. (2006). Clinical management of chronic nightmares: Imagery rehearsal therapy. *Behavioral Sleep Medicine, 4*(1), 45–70.

Kribbs, N., Pack, A., Kline, L., Smith, P., Schwartz, A., & Schubert, N. (1993). Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *American Review of Respiratory Disease, 147*, 887–895.

Lansky, M. (1995). *Post truamatic nightmares: Psycho-dynamic explorations*. Hillsdale, NJ: The Analytic Press.

Levin, R. (1998). Nightmares and schizotypy. *Psychiatry and Clinical Neurosciences, 61*, 206–216.

Lichstein, K. L. (1988). *Clinical relaxation strategies*. Hoboken, NJ: John Wiley and Sons Ltd.

Littner, M., Hirshkowitz, M., Kramer, M., Kapen, S., Anderson, W., & Bailey, D. (2003). Practice parameters for using polysomnography to evaluate insomnia: An update. *Sleep, 26*(6), 754–760.

Mack, J. (1974). *Nightmares and human conflict*. Boston: Houghton Mifflin.

Marks, I. (1978). Rehearsal relief of a nightmare. *British Journal of Psychiatry, 135*, 461–465.

Means, M. (2002). Mind over matter: Optimizing CPAP compliance. *Advance for Managers of Respiratory Care, 11*, 10–13.

Means, M., & Edinger, J. (2007). Graded exposure therapy for addressing claustrophobic reactions to continuous positive airway pressure: A case series report. *Behavioral Sleep Medicine, 5*, 105–116.

Mellman, T. A., & Uhde, T. W. (1990). Patients with frequent sleep panic: Clinical findings and response to medication treatment. *Journal of Clinical Psychiatry, 51*(12), 513–516.

Miller, W., & DiPilato, M. (1983). Treatment of nightmares via relaxation and desensitization: A controlled evaluation. *Journal of Consulting and Clinical Psychology, 51*, 870–877.

Morin, C. M., Culbert, J. P., & Schwartz, S. M. (1994). Nonpharmacological interventions for insomnia: A meta-analysis of treatment efficacy. *American Journal of Psychiatry, 151*, 1172–1180.

Murtagh, D. R., & Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: A meta-analysis. *Journal of Consulting and Clinical Psychology, 63*, 79–89.

Neidhardt, E., Krakow, B., Kellner, R., & Pathak, D. (1992). The beneficial effects of one treatment session and recording of nightmares on chronic nightmare sufferers. *Sleep, 15*(5), 470–473.

Neylan, T., Marmar, C., Metzler, T., Weiss, D., Zatzick, D., & Delucchi, K. (1998). Sleep disturbances in the Vietnam generation: Findings from a nationally representative sample of male Vietnam veterans. *American Journal of Psychiatry, 155*, 929–933.

Nicassio, P., & Bootzin, R. R. (1974). A comparison of progressive relaxation and autogenic training as treatments for insomnia. *Journal of Abnormal Psychology, 83*, 253–260.

Nielsen, T., & Zadra, A. (2005). Nightmares and other common dream disturbances. In M. Kryger, T. Roth, & W. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed.). Philadelphia: Elsevier Saunders.

Norton, P., & Price, E. (2007). A meta-analytic review of adult cognitive-behavioral treatment outcome across the anxiety disorders. *Journal of Nervous and Mental Disease, 195*(6), 521–531.

Nowell, P. D., Mazumdar, S., Buysse, D. J., Dew, M. A., Reynolds, C. F. I., & Kupfer, D. J. (1997). Benzodiazepines and zolpidem for chronic insomnia: A meta-analysis of treatment efficacy. *Journal of the American Medical Association, 278*, 2170–2177.

Partinen, M. (1994). Epidemiology of sleep disorders. In M. Kryger, T. Roth, & W. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.). Philadelphia: WB Saunders.

Rains, J. C. (1995). Treatment of obstructive sleep apnea in pediatric patients: Behavioral intervention for compliance with nasal continuous positive airway pressure. *Clinical Pediatrics, 34*, 535–541.

Rauscher, H., Popp, W., Wanke, T., & Zwick, H. (1991). Acceptance of CPAP therapy for sleep apnea. *Chest, 100*, 1019–1023.

Rolfe, I., Olson, L., & Saunders, N. (1991). Long-term aceptance of continuous positive airway pressure in obstructive sleep apnea. *American Review of Respiratory Disease, 144*, 1130–1133.

Roy-Byrne, P. P., Uhde, T. W., & Post, R. M. (1986). Effects of one night's sleep deprivation on mood and behavior in panic disorder: Patients with panic disorder compared with depressed patients and normal controls. *Arch Gen Psychiatry, 43*(9), 895–899.

Rybarczyk, B., Stepanski, E., Fogg, L., Lopez, M., Barry, P., & Davis, A. (2005). A placebo-controlled test of Cognitive-Behavioral Therapy for comorbid insomnia in older adults. *Journal of Consulting and Clinical Psychology, 73*(6), 1164–1174.

Sanders, M., Gruendl, C., & Rogers, R. (1986). Patient compliance with nasal CPAP therapy for sleep apnea. *Chest, 90*, 331–333.

Schreuder, B., van Egmond, M., Kleijn, W., & Visser, A. (1998). Daily reports of posttraumatic nightmares and anxiety dreams in Dutch war victims. *Journal of Anxiety Disorders, 12*, 511–524.

Schultz, J. H., & Luthe, W. (1959). *Augenic training*. New York: Grune & Stratton.

Siev, J., & Chambless, D. (2007). Specificity of treatment effects: Cognitive therapy and relaxation for generalized anxiety and panic disorders. *Journal of Consulting and Clinicial Psychology, 75*(4), 513–522.

Smith, J. (1990). *Cognitive-behavioral relaxation training: A new system of strategies for treatment and assessment*. New York: Springer Publishing Company.

Stetter, F., & Kupper, S. (2002). Autogenic training: A meta-analysis of clinical outcome studies. *Applied Psychophysiology and Biofeedback, 27*(1), 45–98.

Zadra, A., & Dondri, D. (2000). Nightmares and bad dreams: Their prevalence and relationship to well-being. *Journal of Abnormal Psychology, 109*, 273–281.

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