The vicious cycle of insomnia and anticipatory anxiety

Insomnia, characterized by difficulty initiating, maintaining, or obtaining qualitatively satisfying sleep is a widespread health complaint. Like the common cold, most individuals have experienced at least transient bouts of nocturnal sleep difficulty due either to an impending stressful (e. g., final exam) or exciting (e. g., a long-awaited vacation trip) event or due to acute medical or environmental factors. However, slightly over one-third of the adult population complains of recurring, intermittent sleep difficulties whereas 9 to 10% endure chronic, unrelenting insomnia problems. Although many health care professionals as well as the lay public may minimize its significance, insomnia may have notable short- and long-term consequences. At a minimum, insomnia results in daytime fatigue, decreased mood, and general malaise. In more protracted cases it may cause impaired occupational and social functioning. In addition, there is substantial evidence that insomnia dramatically increases risks for medical complaints, alcohol and drug abuse, and serious psychiatric illnesses. Moreover, insomnia alone contributes to increased health costs and utilization among affected individuals and, in turn, escalates health care costs for society in general. Indeed, insomnia sufferers may collectively spend well over $285 million per year for prescription sleeping pills whereas the projected annual direct, treatment-related costs of insomnia to the U. S. population may be as high as $92. 5 billion. Thus, chronic insomnia represents a significant public health problem that warrants early detection and treatment.

The Vicious Cycle of Insomnia and Anticipatory Anxiety

The interaction of disturbances in sleep and wakefulness is clearly seen in the mutually reinforcing experiences of sleepless nights and anxious days.

Transient insomnia is nearly a universal experience. The tossing and turning, the racing mind and half-completed thoughts, the frustration at being unable to bring oneself relief, all of these experiences are extremely unpleasant and avoided if possible. During the day, insomniacs will wonder whether these experiences are again in store. A dread of the night to come may appear as evening approaches. This anticipation of a sleepless night produces anxiety and physiological arousal. Thus, fear of insomnia has itself produced sufficient arousal to perpetuate the sleep disturbance. This vicious cycle persists despite occasional nights of good sleep. Variability of sleep from night to night is characteristic of insomnia. This renders the sleep of insomnia unpredictable and provides the basis for the insomniac’s worry.

The Three P Model of Insomnia: Predisposing, Precipitating, and Perpetuating Factors in Insomnia

The nosological scheme of the International Classification of Sleep Disorders (revised edition) has produced a clear and consistent description of the sleep disorder’s clinical phenomena. Intervention strategies are not automatically derived from diagnosis. With regard to the insomnias, we have urged the use of a simple categorization of case material that helps focus on the roles of different factors in the pathogenesis of the disorder, thereby assisting in a rational approach to treatment. In the development of insomnia, characteristics of the person may serve as predisposing factors by increasing the vulnerability to develop a sleep disturbance. These characteristics might include susceptibility to anxious worrying or activation at night. Environmental features, such as noise and morning light exposure, may also predispose to insomnia. By definition, these characteristics are not sufficient

to produce insomnia, but they may set the stage for the development of a particular form of insomnia. Interventions that address these factors will help ameliorate the current insomnia and forestall the development of insomnia in the future.

The factors that trigger insomnia are at the center of the initial clinical evaluation. An understanding of the factors that precipitate a sleep disturbance is often sufficient for developing a successful treatment plan. For example, a scientist may become increasingly keyed up and alter her bedtime hours as the deadline for submission of a grant application approaches. When writing is going well, she will stay up late; when it is going poorly, in the middle of the day she will take a nap. These changes weaken the synchronization of circadian rhythms that is sustained by a regular sleep-wake cycle. While she may believe that nothing can be done about her sleeplessness until after the deadline, strict structuring of her bedtime may substantially improve the sleep problem.

Predisposing Conditions

A variety of circumstances and conditions predispose patients to insomnia, including personality, age, genes, and intrinsic neurobiologic factors. Tense, anxious, nervous, and worried persons; those who tend to ruminate; those who internalize problems; and those who tend to have somatic responses to stress are at higher risk for insomnia than relaxed, phlegmatic types. Advancing age also predisposes persona to insomnia. Sometimes, relatively minor events associated with little or no obvious stress precipitate insomnia, such as a change to night work or to a rotating shift work schedule. An uncomfortable bed, excessive noise, a bedroom that it too hot or too cold, or

other changes in the sleeping environment may also precipitate insomnia in predisposed person. Predisposing characteristics are often present for years before chronic insomnia takes hold. Many are thought to be congenital, such as tendencies toward physiologic or cognitive hyperarousal, or innate preferences for activity in the evening versus the morning. The 3P model allows for acquired predisposing factors as well. For example, residual pain following an injury may not in itself be accompanied by chronic insomnia, but it can lower the threshold for the disorder's appearance.

Precipitants

Precipitants are usually revealed by the patient's life circumstances; in one study, 74% of insomniacs reported a stressful event at the onset of insomnia, and almost half of insomniacs note that worries make their sleep worse. Typical life events that precipitate insomnia include death or illness of a loved one, divorce or separation, a move to a new location, and a change in occupational status. Depression and other psychiatric disorders can precipitate insomnia, and spousal bereavement in older persons often leads to insomnia that may persist for more than 1 year (see Chap. 16). Medical illnesses may also precipitate insomnia via their effects on bodily systems (e. g., insomnia caused by cardiac failure), their symptoms (e. g., pain and stiffness of arthritic conditions), or their treatment (e. g., (3-adrenergic agents for treatment of asthma). For a discussion of the medical disorders that can cause sleep disruption, see Chapter 17. Sometimes, relatively minor life events associated with little or no obvious stress precipitate insomnia, such as a change to night work or to a rotating shift work schedule. In one study, subjects who were expected to give a brief talk on a specific topic

after awakening had more difficulty falling asleep than a control group. 19 An uncomfortable bed, excessive noise, a bedroom that is too hot or too cold, or other changes in the sleeping environment may also precipitate insomnia in predisposed persons.

By the time people have labeled themselves ” poor sleepers” and presented this complaint to their physician, the precipitating events identified as triggers of their sleeplessness are often long resolved. This can be a source of consternation. A patient may appear years after a divorce and demonstrate convincingly that she has moved on with her life yet still be unable to count on a good night’s sleep. In this case, perpetuating attitudes and practices, the third component of the 3P model, have likely become predominant. As we have seen, the experience of sleep disturbance on a chronic basis becomes self-sustaining. Poor sleepers begin to associate bedtime and their bedrooms with an anxious hyperaroused state, and they settle for short-term relief from the effects of sleep loss through ultimately maladaptive measures such reliance on caffeine or frequent napping (Fig. 2).

Perpetuating Circumstances

From a therapeutic perspective, the perpetuating factors are critical because they may be most amenable to change. Anxiety about insomnia, negative conditioning, poor sleep habits, the use of hypnotics and alcohol, and secondary gain associated with insomnia are important perpetuating factors. Anxiety about insomnia and about its effects on daytime function often perpetuates insomnia. Concern or overconcern about the impact of insomnia on daytime function may lead to performance anxiety, whereby the patient feels required to perform the function or duty of falling asleep. Unfortunately,

sleep cannot be willed to occur, and as the patient tries hard to fall sleep, it becomes increasingly difficult to fall asleep, which in turn leads to increased anxiety. The increased anxiety then makes falling asleep even more difficult. Sterman and associates20 demonstrated the role of conditioning by using classic conditioning to induce sleep in cats. The animals received paired stimuli of a tone and electrical stimulation of the preoptic basal forebrain that induced sleep. After repeated pairing of the stimuli, the experimenters found that the tone alone could induce sleep.

With most people who fall asleep easily, similar conditioning probably takes place with the bed, the bedclothes, and the act of getting into bed and preparing to sleep. In patients with chronic insomnia, however, the process of preparing for sleep, getting into bed, and turning out the lights is no longer associated with falling asleep but may instead become tied to anxiety, sleeplessness, and fear of failure to fall asleep. The bed itself then becomes a source of anxiety and a stimulus for arousal. In such patients, sleep is often better away from home or on a couch, when the negative associations with the bed are absent and the expectation that sleep will be worse in an unfamiliar setting reduces the performance anxiety usually associated with attempts to sleep.

After insomnia begins, some patients develop poor sleep habits that perpetuate insomnia. They may exercise at night in order to feel more tired, spend more time in bed or go to bed at irregular times in an attempt to obtain more sleep, or increase their daytime caffeine consumption in order to feel more alert. Other patients have never had good sleep habits but were able to sleep well as young persons' because of robust sleep mechanisms.

With age-related impairment of sleep, their poor habits lead to or perpetuate insomnia. Secondary gain associated with insomnia may also perpetuate the symptom. Insomnia may be used as a reason for nighttime snacks, alcohol, or TV watching, which then act as reinforcers for poor sleep. Time off work may be an additional source of secondary gain. For some, insomnia may contribute to the role of “ sickly child” or dependent adult. For others, particularly those with marital or relational problems, insomnia or the associated perceived need for a quiet time before bed may provide a rationale for avoiding sexual relations.

Use of alcohol and hypnotics may perpetuate sleep disturbance. Short-acting hypnotics, such as triazolam and zolpidem, may wear off before the end of the night, leading to early-morning insomnia, and can also produce “ rebound insomnia” on the following night if the hypnotic is not ingested. Insomniacs often discover that alcohol promotes sleep onset, and some insomniacs develop a conditioned association between alcohol use and falling asleep that leads them to believe they cannot sleep without a “ nightcap.” For heavy alcohol users, anxiety and symptoms of mild alcohol withdrawal that develop if alcohol is not consumed contribute to their impression that they cannot sleep without alcohol. Unfortunately, although alcohol can hasten the onset of sleep, it also can lead to sweaty, restless sleep during the second half of the night and frequent awakenings from dreams with difficulty returning to sleep (see Chap. 16). With chronic use at bedtime or during the night, the sleep-inducing effect of alcohol may be reduced, whereas its effects on late-night sleep continue or are increased, leading to daytime fatigue and sleepiness. Alcohol also suppresses rapid eye

movement (REM) sleep and probably makes sleep less restful. In some patients, a precipitating event is difficult to identify; it appears that poor sleep may develop gradually in these persons as the anxiety about occasional poor nights of sleep leads to progressively increasing concern about sleep.

Perpetuating factors are what telescope acute sleep disruption into chronic insomnia; as such, they often present the most opportune targets for behavioral treatment. Their presence is actually grounds for optimism. When patients have become disheartened by the entanglement of their sleep with seemingly intractable problems such as chronic illness or the loss of financial security, addressing perpetuating factors can yield moderate improvement relatively quickly.

TABLE COMMON PRACTICES AND RESPONSES TO INSOMNIA

THAT PERPETUATE SLEEPLESSNESS

- Irregular timing of retiring and arising

- Excessive time in bed

- Napping at irregular times

- Worry that insomnia will produce daytime deficits

- Expectation of a bad night’s sleep

- Increased caffeine consumption

- Use of hypnotic medication and alcohol

- Maladaptive conditioning

- “ Sleeping in” on weekends

Insomnia may last for decades. When it persists beyond a transient period, the clinician may have to go beyond the uncovering of predisposing and precipitating factors. As insomnia becomes a chronic experience, the individual may instigate compensatory practices to deal with the problem. Returning to the frantic grant writer, if a habit of napping at irregular hours continues after the deadline is long past, this may maintain her insomnia. Or if she increases her caffeine consumption to buttress her flagging alertness and then continues this habit, her insomnia may persist. In these cases, the precipitating circumstance has long subsided yet the secondary factors are sufficient to maintain the insomnia. Perpetuating factors may go unnoticed, especially when clear predisposing and precipitating aspects are still present. Therefore, one must thoroughly evaluate the common practices and experiences (SEE TABLE ABOVE) that may accrue onto any insomnia so that a comprehensive treatment plan may be designed.

Chronic Pain and Insomnia

Pain and insomnia are among the most common complaints in our society so that the likelihood that the two conditions coincide in the same person should not be surprising. Our common experience is that any painful condition will disturb sleep and if prolonged could have a negative effect upon mood, thinking, energy and behavior. In a recent Gallop Poll Survey, 56 million Americans complained that nighttime pain interfered with their falling asleep or promoted awakenings during the night or awakenings in the early

morning. A Canadian population survey indicated that 44% of people with any painful disorder have sleep problems. This epidemiological study showed that the greater the severity of pain, the higher the likelihood of having insomnia or unrefreshing sleep (Moldofsky 2001). Sleep is a physiological state usually characterized by isolation from the environment, except when an unpleasant, potentially harmful or life-threatening event occurs. During sleep, sensory perception is attenuated to prevent sleep disruption by non-relevant input in order to promote sleep consolidation. The perception of pain in sleep should rather be termed nociception, since sleep is associated with an altered state of consciousness. The presence of pain during wakefulness, as well as the intrusion of pain in the sleeping period, is potentially associated with fatigue and significantly lower sleep quality (e. g. complaints of non-restorative sleep), daytime sleepiness and risk of accidents, and low memory performance.

Individuals suffering from chronic pain often develop pain-related insomnia that can develop into a primary clinical concern. For most patients with chronic pain, fragmented sleep is a secondary and highly distressing component of their condition resulting in increased levels of disability and emotional distress (Morin, 1993). The relationship between sleep disturbance and pain might be reciprocal, such that pain disturbs sleep continuity or quality and poor sleep further exacerbates pain. Some investigators have proposed that behavioral factors may contribute to the maintenance of sleep disturbances among patients with chronic pain (Haythornthwaite, Hegel, & Kerns, 1991). A number of behavioral changes interfere with effective sleep. For example, pain patients may spend increasing time in their bedrooms and

the classically conditioned association between bedroom and sleep may be lost. Pain patients often perform activities in the bedroom or while reclining that are typically performed in other areas inside or outside of the home (Fordyce, Shelton & Dundore, 1982; Loeser, 1991). Instead of using the bedroom just for sleep or sexual activity, they read, watch television, talk with family members, and further dilute the conditioned facilitating effect of the bedroom on sleep. As patients spend more time in bed, their basic circadian cycle is disrupted. Patients often report a very irregular sleep-wake schedule and spend more time reclining, frequently awakening through the night, and sleeping in daytime. As a result, the basic physiologic rhythm of sleep is lost. More specifically, patients typically develop problematic behaviors such as remaining in bed awake for long periods of time, often resulting in increased efforts to sleep, heightened frustration and anxiety about not sleeping, further wakefulness and negative expectations, and distorted beliefs and attitudes concerning the disorder and its consequences. Negative learned responses may develop and become key perpetuating factors that can be targeted psychological and behavioral therapies. Treatments which address these core components play an important role in the management of co-morbid insomnias.

Chronic pain is a major cause of sleep disturbances and complaints. Its major influence is to increase the magnitude and/or the frequency of arousal and awakening in sleep. A day with intense pain could be followed by sleep of poor quality, and poor sleep may be followed by more pain on the next day. Pain that occurs acutely (such as wound pain immediately after surgery, angina, or a severe toothache) can disrupt sleep by delaying sleep onset,

causing awakening from sleep or poor sleep quality. However, this type of pain is usually short-term and once treated, the effects on sleep are immediately reversible. Chronic pain, even if low-grade and long-lasting, can lead to a vicious cycle where sleep is disrupted leading to poor sleep and increased pain sensitivity the next day. This cycle then continues and affects mood, energy, behavior, and one’s general safety during the day. In most cases, a new pain episode will precede complaints of poor sleep (Morin et al. 1998; Riley III et al. 2001). By contrast, when chronic pain sets in (e. g. burn pain after a few days) a vicious circle is reported: a day with high pain is followed by a night of poor sleep, and sleep of poor quality is followed by reports of higher pain the next day (Raymond et al. 2001).

Sleep Architecture and Pain

A normal sleep period is characterized by alternated sleep stages (light St 1&2 to deep St 3&4 to Rapid Eye Movement (REM) sleep) that occur 3-5 times in a normal sleep period of 7-9 hours. We usually spend approximately 50-65% of a night in St 1&2, 20-25% in St 3&4, and 10-20% in REM sleep. The roles of sleep are to recover from fatigue, maintain cognitive function (e. g. memory consolidation and performance, concentration) and help overall biological regeneration. Sleep disruption (e. g. fragmentation) that interferes with sleep continuity is reported to induce some complaints of fatigue and poor cognitive performance the next day. Chronic pain results in sleep fragmentation and arousals leading to poor quality, unrefreshing sleep. Sleep fragmentation can result in an absolute increase in stage 1 and stage 2 sleep in relation to the other stages of sleep. This means that there is less slow wave sleep which is considered restorative to physical processes.

People with chronic pain also experience many microawakenings during the night related to body movement. There also appears to be overactivation of the sympathetic nervous system in people with chronic pain, which leads to increased anxiety, and problems with maintaining and initiating sleep, leading to insomnia. Sleep can be disrupted in two ways: Sleep fragmentation or deprivation. Sleep fragmentation is a brief intrusion in sleep that causes a transitional change in the sleep process (sleep stage shift, micro-arousal or awakening without a conscious response from the subject). Sleep deprivation is either a total prevention of sleep or is limited to a specific sleep stage (e. g. St 3&4). Both fragmentation and deprivation have potential consequences for functioning the next day (e. g. fatigue, sleepiness, boredom, irritability, poor memory performance) that could influence pain reports or clinician assessment of pain. Interruption of any sleep stage by isolated or repetitive events such as sleep stage shifts (deeper to lighter), micro-arousal or awakening, short duration of deep St 3 & 4 sleep, or Alpha EEG wave intrusion. As a result, EEG frequency is in the fast range, heart rate is increased, muscle tone higher with occasional body movements. As a consequence, sleep continuity may be impaired and sleep complaints are frequent (e. g. un-refreshing).

Sleep is also a state normally associated with a reduction in heart rate variability, due to a change in the balance of components of the autonomic nervous system. In light and deep sleep, a parasympathetic dominance “ slows down” the cardiac activity, while during the awake or REM sleep state there is a cardiac sympathetic dominance, characterized by a higher cardiac activity/variability. The absence of a reduction in cardiac activity during light

or deep sleep may cause un-refreshing or non-restorative sleep. This suggestion is supported by findings showing that chronic pain and insomniac patients maintain a high sympathetic cardiac activity (Martínez-Lavín et al. 1998; Moldofsky 2001; Brousseau et al. 2003).

Pain and Sleep

Sleep physiological studies show that people with chronic low back pain have an arousal disturbance in EEG sleep that interferes with restful sleep (Moldofsky 2001). These sleep physiological disturbances interfere with the natural restorative properties of sleep, so that there is an adverse effect upon daytime functioning. About 75% of people with various painful rheumatic disorders report sleep problems. Fatigue, which is almost universal in people with rheumatic disorders, is largely explained by pain, sleep disturbance and depression. Sleep disturbances are important in patients with rheumatoid arthritis (Moldofsky 2001). Their fatigue is associated with poor sleep, functional disability, joint pain and depression. Along with increased weakness and diminished energy, there is an alpha EEG brain wave pattern riding in non-REM sleep that indicates an arousal disturbance during the sleep of acutely ill arthritic patients.

An experimental study employed to determine whether specific stages in electroencephalographic (EEG) sleep were affected by pain showed that all stages of sleep are disrupted by noxious stimulation of muscles and that quality of sleep was impaired (Lavigne et al. 2004). While painful conditions may interfere with sleep, the corollary is also true. That is, healthy people who were exposed experimentally to several nights of noise induced

arousals from stages 3and 4 (slow wave or deep) non rapid eye movement (non-REM) sleep caused them to experience unrefreshing sleep, nonspecific generalized muscle aching and fatigue (Moldofsky 2001). Furthermore, sleep deprivation counteracts analgesic effects of drugs that affect opioidergic and serotoninergic neural mechanisms (Kundermann et al. 2004). While traditional analgesic, anti-anxiety or antidepressant medications are often used empirically to address pain and mood symptoms, the potential adverse effects of such medications upon sleep and daytime functioning should be considered in the assessment and overall management program (Moldofsky 2002).

CBT for Insomnia Secondary to Chronic Pain

Cognitive-behavioral models of insomnia propose that acute medical or psychiatric illnesses or significant stressful events are common precipitants of acute insomnia (Smith & Perlis, 2001). However, chronic insomnia is perpetuated by factors that serve to maintain the disturbed sleep including extending sleep opportunity (i. e., going to bed early, sleeping in, or napping), engaging in sleep-interfering behaviors (i. e., worrying, working, or watching television), and inappropriately using alcohol or stimulants (Smith & Perlis, 2001). Dysfunctional cognitions about sleep and maladaptive sleep behaviors exacerbate insomnia and disrupt homeostatic processes resulting in poorly timed and unconsolidated sleep (Smith & Perlis, 2000). Thus, cognitive and behavioral treatments target dysfunctional cognitions and maladaptive sleep behaviors. Cognitive and behavioral treatments for sleep difficulties aim to improve sleep by changing poor sleep habits and challenging negative thoughts attitudes and beliefs about sleep. More

specifically, the cognitive-behavioral component aims to change incorrect beliefs and attitudes about sleep (e. g., unrealistic expectations, misconceptions, amplifying consequences of sleeplessness); techniques include reattribution training (i. e., goal setting and planning coping responses), decatastrophizing (aimed at balancing anxious automatic thoughts), reappraisal, and attention shifting (Morin, 1993). Thus, CBT attempts to alter patterns of negative thoughts and dysfunctional attitudes in order to foster more healthy and adaptive thoughts, emotions, and actions.

The empirically validated cognitive-behavioral interventions for insomnia include stimulus control (SCT; Bootzin, 1972), sleep restriction therapy (SRT; Spielman, Saskin, & Thorpy, 1987), relaxation therapies (RT; Lichenstein, Riedel, Wilson, Lester, & Aguillard, 2001), and multi-component approaches (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001). SCT serves to re-associate the bed and bedroom environment as a primary stimulus for sleep. SRT consolidates fragmented sleep by first carefully restricting sleep (i. e., controlled sleep deprivation) and then extending sleep opportunity while maintaining appropriate sleep efficiency (i. e., total sleep time/time in bed). RT attempts to reduce sleep-interfering physiological arousal. Multi-component approaches incorporate cognitive and behavioral interventions to modify maladaptive sleep-related beliefs, manage intrusive pre-sleep cognitions, or provide education to reduce maladaptive sleep behaviors (i. e., sleep hygiene).

Health Anxiety and Pre-Sleep Arousal

Patients with insomnia are characterized by excessive arousal and an inability to sleep. Current management approaches are focused on reducing this hyperarousal and its behavioral manifestations using a range of behavioral treatments (Gunstein, 2002). While a person's subjective complaint of sleep disturbance is central to the diagnosis of insomnia (Morin, 1993), it is important to confirm and delineate the causal links between health anxiety, affective pain, and objective insomnia symptoms. Health anxiety is increasingly recognized as a key feature of a large proportion of chronic pain patients (MacDonald, Linton, & Jansson-Fröjmark, 2008). Tang, Wright, and Salkovski (2007) investigated the prevalence and correlates of insomnia secondary to chronic pain. They found excessive concern and worry over health produces a sleep-interfering effect. Moreover, health anxious pain patients are more likely to show selective attention to bodily sensations, detect heightened physical symptoms, report more intense pain, exhibit lower pain tolerance, report greater anxiety, and engage in catastrophizing thinking (MacDonald, Linton, & Jansson-Fröjmark, 2008). Catastrophizing is defined as a negative cognitive process characterized by a tendency to ruminate on the pain experience, to exaggerate the threat of pain, to adopt a helpless orientation, and to negatively evaluate one's ability to deal with pain (Sullivan et al., 2001).

Health anxiety may trigger or aggravate insomnia by inducing arousal and by activating cognitive-behavioral processes. Likewise, affective pain responses involving negative pain interpretation may serve to potentiate insomnia by activating the arousal system and sleep-interfering processes

(MacDonald, Linton, & Jansson-Fröjmark, 2008). According to Smith and Perlis (2001), cognitive arousal was found to be more predictive than somatic arousal in the development and maintenance of insomnia. These findings substantiate the view that insomnia associated with chronic pain may operate on the same cognitive mechanisms as primary insomnia (Smith & Perlis, 2001). Thus, chronic insomnia secondary to pain may be similar to primary insomnia. Although the precipitating factors may differ, the maintaining factors and subjective experience may not. Thus, chronic pain patients may benefit from the cognitive-behavioral interventions that specifically target insomnia and pre-sleep cognitive or physiological arousal.

Insomnia Secondary to Chronic Pain

Sleep disturbance is one of the most prevalent complaints of patients with chronic pain conditions. According to Morin, Gibson, and Wade (1998), the prevalence of sleep disturbances ranges from 50% to 70% among patients with chronic medical conditions. Sleep disorders in individuals with chronic pain remain under-reported, under-diagnosed and under-treated, which may lead to the frequent development of chronic sleep disorders (Stiefel & Stagno, 2004). Developing effective treatment modalities for patients with insomnia secondary to chronic pain is an overlooked area of study (Lacks & Morin, 1992). Despite unsupported long-term efficacy (King & Strain, 1990), pharmacotherapy remains the most widely used treatment for sleep disturbances secondary to chronic medical conditions (Aronoff, Wagner, & Spangler, 1986). Reliance on sedative medications to control insomnia in chronic pain does not address the patient’s distress concerning sleep-related

functional impairments (Carey, Wilson, Pontefract, & deLaplante, 2000) or the patient’s preference for non-drug treatment alternatives (Morin, 1993).

Despite the recognition of sleep as an essential component of good quality of life, pain clinics do not normally have the resources or expertise to provide comprehensive sleep assessment or treatment for patients complaining of insomnia (Roth, 2009). Most pain programs offer lifestyle changes, usually suggested in a list of sleep hygiene measures such as reducing coffee consumption or exercising but have limited support as stand-alone interventions for the treatment of insomnia (Perlis & Smith, 2001). These brief interventions are typically delivered in a less comprehensive manner and include watered-down version of stimulus control, sleep restriction, and sleep hygiene. Often times, these interventions are provided as a handout to patients and lack formal instruction and implementation (Stiefel & Stagno, 2004). According to Roth (2009) a sleep disorder caused by pain requires a multi-disciplinary appro