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Chapter

16

Behavioral Techniques and Biofeedback for Insomnia

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Insomnia has many different causes, including physical disorders, ingested or inhaled substances, circadian rhythm disturbances, psychological factors, poor sleep environments, and poor sleep habits (Bootzin, Mauber, Perlis, Salvio, & Wyatt, 1993). Because of the multifaceted etiology of insomnia, a thorough assessment is essential. The components of this assessment should include a history, physical examination, daily sleep diaries, and if it appears that other sleep disorders (e.g., periodic limb movements, sleep apnea) are contributing to the problem, polysomnography (PSG). For further information about assessment, see chapter 6, this volume, and the other chapters in part II.

Daily sleep diaries are a critical component of the assessment and treatment process. Diaries typically include entries for naps, sleep latency, number and duration of awakenings, total sleep, quality of sleep, feelings on awakening, and whether or not the night in question was a typical night. The patients fill out the diaries each morning.

Diaries are a practical, efficient means of obtaining information about the frequency of sleep problems in the patient's environment. They often have a therapeutic effect, as well. As a consequence of keeping daily diaries, patients may discover that their sleep problems are not as severe or as frequent as they thought. Daily diaries also provide a means of assessing whether or not interventions are having any effect. Without diaries, patients may become discouraged at the occurrence of even a single sleepless night. Keeping sleep diaries helps patients to focus on whether or not the frequency of the sleep problem is changing.

A number of behavioral and cognitive–behavioral treatments have been found to be effective for treating insomnia. These include sleep hygiene information, stimulus control instructions, sleep restriction, relaxation training, biofeedback, and cognitive therapy.

Sleep Hygiene Information

Basic information about sleep and sleep hygiene is usually provided as a core component of all treatments for insomnia. Inadequate sleep hygiene refers to daily living activities that are inconsistent with the maintenance of high-quality sleep and daytime alertness (American Sleep Disorders Association, 1990). Among the activities included in this category are the irregular use of daytime naps; extended amounts of time spent in bed; irregular sleep–wake schedules; inactivity; the routine use of products that interfere with sleep such as caffeine, nicotine, and alcohol; scheduling of exercise close to bedtime; engaging in exciting or emotionally upsetting activities close to bedtime; having a poor sleep environment such as an uncomfortable bed; or having a bedroom that is too bright, stuffy, hot, cold, or noisy. In addition, emphasis is frequently placed on the individual variability of sleep need and on changes in the nature and quality of sleep associated with aging.

In recent years, increased focus has been placed on the use of bright light to regularize the sleep–wake circadian rhythm (see Chapter 11, this volume). One component of the problem may be that the underlying sleep–wake circadian rhythm has shifted or has a reduced amplitude owing to the patient's irregular wake–sleep patterns. Evaluations of patients with insomnia have found that the sleep–wake circadian rhythm is shifted depending on the nature of the insomnia. Patients with sleep-onset insomnia, who have difficulty falling asleep and difficulty waking up in the morning, have been found to have a delayed sleep–wake circadian rhythm as measured by core body temperature (Morris, Lack, & Dawson, 1990). In contrast, patients with sleep-maintenance insomnia, who are sleepy in the early evening and have difficulty sustaining sleep, have been found to have an advanced sleep–wake circadian rhythm (Lack & Wright, 1993). Appropriately timed bright light exposure has been found to shift the sleep–wake rhythm; that is, bright light in the morning advances and bright light in the evening light delays the sleep–wake rhythm.

Encouraging results have been reported with the use of bright light for insomnia. Morning light has been found to reduce sleep-onset latency in sleep-onset insomnia (Lack, Wright, & Paynter, 1995), and evening light has been found to delay wake-up time, increase total sleep (Lack & Wright, 1993), and increase sleep efficiency (Campbell, Dawson, & Anderson, 1993) in sleep-maintenance insomnia.

The increasing literature on the effectiveness of bright light in resetting the sleep–wake circadian rhythm in a variety of settings has led many clinicians to add exposure to bright light as one of the sleep hygiene recommendations. In an evaluation of the effects of exercise or bright light added to sleep hygiene instructions, Guilleminault et al. (1995) found the strongest improvement in patients who received morning bright light instructions along with other sleep hygiene recommendations.

In many sleep clinic settings, sleep clinicians are asked to provide evaluations for patients who will be seen only for one or two treatment sessions. Given this constraint, many of the recommendations focus on sleep hygiene. In an evaluation of 62 patients with insomnia who received one session of evaluation and sleep hygiene recommendations, Hauri (1993) found that patients reported improved sleep at 1-, 3-, and 12-month telephone follow-ups and that 70% of those who tried a recommendation reported that it was helpful. As encouraging as these results are, a limitation in this study is that improvement was estimated by patients in retrospective, subjective reports rather than by means of prospective measures such as daily diaries. These results are best viewed as evidence of patient satisfaction, which is quite high, rather than as evidence of improved sleep.

In contrast to the positive results for sleep hygiene reported by Hauri (1993), a meta-analysis of studies that evaluated nonpharmacological treatments for insomnia against control conditions found that sleep hygiene recommendations, as a sole intervention, was not as effective as other treatments (Morin, Culbert, & Schwartz, 1994). No one would argue that information about sleep and sleep hygiene is unimportant; it is included in all multicomponent treatment packages. Caution should be exercised, however, in relying on sleep hygiene recommendations alone, because other behavioral treatments have been found to be more effective.

Stimulus Control Instructions

Stimulus control instructions (Bootzin, 1972, 1977; Bootzin & Nicassio, 1978) are a set of instructions designed to help the patient with insomnia to establish a consistent sleep-wake rhythm, strengthen the bed and bedroom as cues for sleep, and weaken them as cues for activities that might interfere with sleep. This treatment was derived from an operant learning analysis in which falling asleep is conceptualized as an instrumental act emitted to produce reinforcement (i.e., sleep). Stimuli associated with sleep become discriminative stimuli for the occurrence of reinforcement. Difficulty in falling asleep, or in falling back to sleep after awakening, may be due to inadequate stimulus control. Both strong discriminative stimuli for sleep may not have been established or discriminative stimuli for activities incompatible with sleep may be present.

To develop discriminative stimuli for sleep, it is often helpful to establish a bedtime routine. This has been particularly effective in the treatment of children with bedtime disturbances (e.g., Richman, Douglas, Hunt, Lansdown, & Levere, 1985). It is equally important to weaken the cues for activities and emotional reactions that might interfere with sleep. Many people with insomnia seem to organize their entire existence around their bedroom, with television, telephone, books, and food within easy reach (Bootzin & Nicassio, 1978). For others, bedtime is the first quiet time during the day available to think about the day's events and to worry and plan for the next day. Under these conditions, bed and bedtime become cues for arousal rather than cues for sleep. Another source of arousal for the patient is that the bedroom can become a cue for the anxiety and frustration associated with *trying* to fall asleep (Bootzin & Nicassio, 1978). People with insomnia often can sleep anyplace other than their own bed. They might fall asleep in an easy chair or on a couch, and they often have little trouble sleeping when away from home.

There have been many studies from many different investigators evaluating the effectiveness of stimulus control instructions and other behavioral treatments for insomnia. Reviews of those studies indicate that stimulus control instructions constitute one of the most effective, if not the most effective, single-component therapy (Lacks & Morin, 1992; Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995). It is useful, therefore, to have stimulus control instructions as a core treatment element, around which other elements can be added.

The following rules constitute the stimulus control instructions (Bootzin, Epstein, & Wood, 1991):

1. Lie down intending to go to sleep only when you are sleepy.
2. Do not use your bed for anything except sleep; that is, do not read, watch television, eat, or worry in bed. Sexual activity is the only exception to this rule. On such occasions, the instructions are to be followed afterward, when you intend to go to sleep.
3. If you find yourself unable to fall asleep, get up and go into another room. Stay up as long as you wish and then return to the bedroom to sleep. Although we do not want you to watch the clock, we want you to get out of bed if you do not fall asleep immediately. Remember the goal is to associate your bed with falling asleep *quickly*! If you are in bed more than about 10 minutes without falling asleep and have not gotten up, you are not following this instruction.
4. If you still cannot fall asleep, repeat Step 3. Do this as often as necessary throughout the night.
5. Set your alarm and get up at the same time every morning irrespective of how much sleep you got during the night. This will help your body to acquire a consistent sleep rhythm.
6. Do not nap during the day.

The focus of the instructions is primarily on sleep onset. For sleep-maintenance problems, the instructions are to be followed after awakening when the patient has difficulty falling back to sleep. Although stimulus control instructions appear to be simple and straightforward, compliance is better if the instructions are discussed individually and a rationale is provided for each rule (Bootzin et al., 1991):

Rule 1. The goal of this rule is to help patients become more sensitive to internal cues of sleepiness so that they will be more likely to fall asleep quickly when they go to bed.

Rule 2. The goals here are to have activities that are associated with arousal occur elsewhere and to break up patterns that are associated with disturbed sleep. If bedtime is the only time patients have for thinking about the day's events and planning the next day, it is recommended that they spend some quiet time doing that in another room before they go to bed. Many people without sleep problems read or listen to music in bed without problems. However, this is not the case for those with insomnia. This instruction is intended to help those who have sleep problems establish new routines to facilitate sleep onset.

Rules 3 and 4. To help them associate the bed with sleep and disassociate it from the frustration and arousal of not being able to sleep, patients are instructed to get out of bed after about 10 minutes (20 min for those over 60 years old). This is also a means of coping with insomnia. By getting out of bed and engaging in other activities, patients are taking control of their problem. Consequently, the problem appears more manageable and the patient is likely to experience less distress.

Rule 5. People with insomnia often have irregular sleep rhythms because they try to make up for poor sleep by sleeping late or napping the next day. Keeping consistent wake times helps patients develop consistent sleep rhythms. In addition, adhering to a set wake time means that the patient will be somewhat sleep-deprived after a night of insomnia. This will make it more likely that they will fall asleep quickly the following night, strengthening the cues of the bed and bedroom for sleep. Often patients with insomnia want to follow a different sleeping schedule on weekends or nights off than they do during the work week. It is important to have as consistent a schedule as possible, 7 nights a week. In our experience, a deviation of no more than 1 hour in the wake time on days off does not produce problems in establishing a consistent rhythm.

Rule 6. The goals of this rule are to keep patients from disrupting their sleep patterns by irregular napping and to prevent them from losing the advantage of the sleep loss of the previous night for increasing the likelihood of faster sleep onset the following night. A nap that takes place 7 days a week at the same time is permissible. For elderly people who feel that they need to nap, a daily nap of 30–45 minutes or the use of 20–30 minutes of relaxation as a nap substitute is recommended.

Behavioral treatments for insomnia, including stimulus control instructions, are primarily self-management treatments. The treatments are carried out by the patient at home. Consequently, compliance may be a problem. Most compliance problems can be solved by direct discussion. A common problem is the disturbance of the spouse's sleep when the patient gets out of bed. Discussions with spouses are often helpful in ensuring full cooperation. During the winter in cold climates, some patients may be reluctant to leave the warmth of their bed. Suggestions for keeping warm robes near the bed and keeping an additional room warm throughout the night, along with encouragement to try to follow instructions, are usually effective in promoting compliance (Bootzin, Engle-Friedman, & Hazlewood, 1983).

One important measure of compliance is whether patients continue to follow the instructions after treatment ends. In an evaluation of the effects of sleep information, progressive relaxation, and stimulus control instructions, participants were contacted for a 2-year follow-up;

they completed sleep diaries and were asked a number of questions, including whether they had continued to follow the treatment they had received (Engle-Friedman, Bootzin, Hazlewood, & Tsao, 1992). A higher percentage of those who had received stimulus control therapy reported that they continued to use elements of the treatment (72.7%) when compared with those receiving progressive relaxation (53.3%) or sleep information alone (38.5%). In addition, those receiving stimulus control instructions reported the highest quality of sleep and showed more improvement in sleep-onset latency than those who received the other treatments.

Sleep Restriction

Sleep-restriction therapy (Spielman, Saskin, & Thorpy, 1987) is based on the observation that many people with insomnia, especially older adults, spend too much time in bed attempting to sleep and therefore have poor sleep efficiency. The aim of sleep-restriction therapy is to consolidate sleep through the restriction of the amount of time spent in bed. Sleep-restriction therapy prescribes an individualized sleep–wake schedule that limits the person's amount of time in bed to his or her estimated average amount of nighttime sleep. Partial sleep deprivation is thereby induced, with the resultant consolidation of sleep. Daytime sleepiness may be experienced at the beginning of treatment, but as sleep becomes more consolidated (i.e., efficient), the sleep–wake schedule is altered by increasing the amount of time in bed.

In a meta-analysis, sleep restriction and stimulus control instructions were found to be the two most effective treatments (Morin, Culbert, & Schwartz, 1994). Sleep-restriction therapy has been included as a core component of treatment packages for patients with sleep-maintenance insomnia. Significant reduction in time awake after sleep onset, increase in sleep efficiency and total sleep time, and less time in bed have been reported (e.g., Edinger, Hoelscher, Marsh, Lipper, & Ionescou-Pioggia, 1992; Friedman, Bliwise, Yesavage, & Salom, 1991).

Persons spending too much time in bed attempting to sleep can benefit from sleep-restriction therapy. For example, a patient who spends 9 hours in bed but sleeps only 5 hours would be prescribed an initial time in bed of 5 hours. No matter how little a patient reports that he or she is sleeping, the prescribed sleep window is always a minimum of 4.5

hours. In scheduling the timing of the sleep window and the amount of time in bed, a flexible approach by the therapist along with active participation by the patient has been suggested to ensure compliance (Friedman et al., 1991). Issues to consider during therapy include initial daytime sleepiness and tasks and activities to keep busy and stay awake during the afternoon and evening until the scheduled bedtime (Glovinsky & Spielman, 1991).

Relaxation Training and Meditation

A commonly recommended treatment for insomnia is some type of relaxation training. This includes a variety of procedures such as progressive relaxation, diaphragmatic breathing, autogenic training, meditation, yoga, and hypnosis. Progressive relaxation, developed by Edmund Jacobson (1938) and presented in manual form for more abbreviated use by Bernstein and Borkovec (1973), is the most widely researched single nonpharmacological treatment for insomnia (Lichstein & Riedel, 1994). Progressive relaxation involves sequentially tensing and releasing the body's major muscle groups while attending to the changing sensations of tension and relaxation.

There are a number of studies that indicate that relaxation procedures can produce improved sleep (e.g., Espie, Lindsay, Brooks, Hood, & Turvey, 1989; Lick & Heffler, 1977; Nicassio & Bootzin, 1974). In one of the few studies using polysomnographic (PSG) measurement, progressive relaxation training was found to be effective in improving sleep-onset latency in patients with either psychophysiological or subjective insomnia (Borkovec, Grayson, O'Brien, & Weerts, 1979). *Psychophysiological insomnia* refers to sleep disturbances that can be verified by objective measures (e.g., PSG) and are not associated with either extrinsic determinants (e.g., noise or drugs) or other disorders (e.g., major depression). In *subjective insomnia,* the complaint of insomnia cannot be verified by objective measures. This disorder has also been called *disorder of initiating or maintaining sleep without objective findings* and *sleep-state misperception*. Borkovec and colleagues (1979) found improvement in subjective insomnia on daily sleep diaries and on subjective estimates while in the sleep laboratory. Because these patients did not exhibit a problem on the PSG, no improvement was observed on that measure. Improvement for psychophysiological insomnia, on the other hand, was found on both sleep diaries and polysomnography.

The different types of relaxation and meditation procedures have all been found to be about equally effective as treatments for insomnia in controlled studies (Van Oot, Lane, & Borkovec, 1984). All of these procedures are based on the same premise: If people can learn to be relaxed at bedtime, they will fall asleep faster. Studies investigating the relationship between physiological arousal and sleep-onset latency have produced both positive (Bonnet & Arand, 1995; Freedman & Sattler, 1982; Monroe, 1967) and negative (Good, 1975; Haynes, Follingstad, & McGowan, 1974) results. Thus, not all patients with insomnia are hyperaroused, but those that are are likely to be aroused both during the day and at night. Bonnet and Arand (1995) found that people with insomnia had an increased 24-hour metabolic rate when compared with age-matched controls. The people with insomnia also had more disturbed sleep and took longer to fall asleep during the day as measured by the Multiple Sleep Latency Test (MSLT).

Because many people who have insomnia are aroused and anxious during the day, relaxation training may provide a double benefit: first, as a general coping skill to reduce daytime arousal and allow the patient to deal more effectively with the stresses of the day and, second, as a means of helping to induce sleep (Bootzin & Nicassio, 1978). Regardless of the type of relaxation method used, it is important to teach relaxation as a "portable" skill. The reliance on tapes may foster dependence on the machine and make it less likely that the patient will be able to use relaxation as a general coping skill for other life stressors.

Biofeedback

Three types of biofeedback have been found to be effective in studies of the treatment of insomnia: electromyography (EMG), theta electroencephalography (EEG) and sensorimotor rhythm (SMR) EEG. Studies involving EMG biofeedback have been the most prevalent.

EMG biofeedback is best characterized as a relaxation procedure. In addition to undergoing biofeedback-assisted relaxation training in the laboratory, patients are usually taught a relaxation procedure to use at home when biofeedback is not available. The EMG biofeedback literature suggests that this can be an effective treatment for insomnia. Groups receiving EMG biofeedback have shown significant decreases in sleep latency (Freedman & Papsdorf, 1976; Haynes, Sides, & Lockwood, 1977; Nicassio, Boylan, & McCabe, 1982; Sanavio, 1988;

Sanavio, Vidotto, Bettinardi, Rolletto, & Zorzi, 1990; VanderPlate & Eno, 1983), number of nighttime awakenings (Haynes et al., 1977), and time awake after sleep onset (Sanavio et al., 1990), as well as increased total sleep time (Sanavio, 1988).

Studies comparing EMG biofeedback with other types of relaxation procedures, however, have found no differences on any sleep parameter (Coursey, Frankel, Gaarder, & Mott, 1980; Freedman & Papsdorf, 1976; Haynes et al., 1977; Hughes & Hughes, 1978; Nicassio, Boylan, & McCabe, 1982). In addition, in studies in which biofeedback has led to improvements in sleep, the reduction in muscle tension during treatment has not been related to amount of improvement in sleep (Freedman & Papsdorf, 1976; Haynes et al., 1977; Hughes & Hughes, 1978; Nicassio et al., 1982). Therefore, although EMG biofeedback has proved to be more effective than no treatment at relieving symptoms of insomnia, it has not proved to be superior to relaxation training or other behavioral treatments.

In contrast to EMG biofeedback, which attempts to improve sleep by reducing physiological arousal, EEG biofeedback has the promise of inducing sleep directly. Theta EEG rhythm (4–7 Hz) is characteristic of Stage 1 sleep in humans and is also seen in combination with alpha rhythm during presleep drowsy periods. There has been some success in using a combination of EMG, alpha, and theta EEG biofeedback in the treatment of insomnia (Budzynski, 1973) or in using theta EEG biofeedback alone (Bell, 1979; Hauri, Percy, Hellekson, Hartmann, & Russ, 1982). Bell (1979) reported a case study in which previous relaxation training did not yield any significant benefits, but eleven 20-minute sessions of theta biofeedback led to a decrease in sleep latency of approximately 30 minutes at a 3-month follow-up.

SMR biofeedback is used to strengthen a 12- to 14-Hz EEG rhythm from the sensorimotor cortex. Sleep spindles, which are present during Stage 2 sleep and are thought to be part of an inhibitory process that deepens sleep, are bursts of 12- to 14-Hz EEG activity. Sterman, Howe, and Macdonald (1970) trained cats through operant conditioning to produce more of this rhythm while awake and found that these cats showed more SMR rhythm not only during wakefulness but also during sleep. They also found that the trained cats had more spindle-burst activity and longer periods of undisturbed sleep when compared with a control group. A subsequent study (Feinstein, Sterman, & Macdonald, 1974) compared SMR biofeedback with other frequencies (occipital, 10 Hz; central, 10 Hz; and central, 15 Hz) in humans. The

SMR biofeedback group showed an increase in the number and duration of sleep spindles, decreases in sleep latency and number of movements during sleep, and an increase in percentage of REM time. The other forms of biofeedback yielded none of these benefits.

Jordan, Hauri, and Phelps (1976) measured waking SMR in a group of patients with insomnia and discovered that they had significantly less waking SMR than a control group of "good sleepers." They also found that waking SMR correlated positively (.59) with sleep efficiency and negatively (.64) with sleep latency as measured by the PSG. Hauri and colleagues then conducted two investigations designed to evaluate the effects of SMR biofeedback training on the sleep of persons with insomnia. The first of these studies (Hauri, 1981) compared groups who received EMG biofeedback, combined EMG and theta EEG biofeedback, and SMR EEG biofeedback, and a no-treatment control group. The results indicated that amount of feedback learning was related positively to sleep improvement only in the SMR group. None of the treatment groups, however, improved significantly more than the control group on any sleep parameter at a 9-month follow-up.

An internal analysis of the results indicated that there was an interaction between treatments and type of insomnia. Patients in each of the three treatment groups were rank ordered according to initial tension levels. The more tense a patient was initially, the more he or she benefited from EMG biofeedback. A similar nonsignificant pattern of results was obtained in the EMG–theta group. In the SMR biofeedback group, participants who were least tense initially received the most benefit. These results suggested that tailoring biofeedback treatment on the basis of initial tension levels might be beneficial in the treatment of insomnia. To replicate this finding, Hauri and colleagues (1982) randomly assigned 16 patients with psychophysiological insomnia to treatment with either theta biofeedback or SMR biofeedback. Although both theta and SMR biofeedback yielded significant improvements on sleep diaries, the PSG results indicated that patients with low levels of presleep tension benefited only from SMR biofeedback and those with high tension levels benefited only from theta biofeedback. However, many of the correlations were nonsignificant trends, which the authors attributed to the small sample size. Hauri and his colleagues (1982) concluded that either type of biofeedback leads to improvements on subjective measures of sleep but that only an appropriate form of biofeedback, chosen on the basis of initial tension levels, is likely to lead to the PSG improvements in sleep. Despite these encouraging findings, particularly with regard to SMR biofeedback, we have been unable to find any subsequent, published evaluations of SMR biofeedback for insomnia.

Although there have been few studies in recent years investigating the efficacy of biofeedback as a treatment for insomnia, there is a growing literature that suggests that EEG biofeedback procedures are effective interventions for other problems. SMR biofeedback has been employed as a treatment for patients with epileptic seizures that have not responded to medication (e.g., Lantz & Sterman, 1988). In addition, in the past few years, alpha–theta biofeedback has been evaluated as a promising treatment for alcoholism and posttraumatic stress disorder (e.g., Fahrion, Walters, Coyne, & Allen, 1992; Peniston & Kulkosky, 1989, 1991).

Cognitive Therapy

There are a number of cognitive symptoms that contribute to insomnia, such as worry, cognitive intrusions, and dysfunctional beliefs about sleep and its consequences. Among the cognitive interventions are paradoxical intention, cognitive restructuring, thought stopping, and articulatory suppression.

Paradoxical Intention

A cognitive intervention that has been frequently evaluated is paradoxical intention (PI). Many persons with insomnia exacerbate their problem by worrying about whether they will be able to fall asleep. To reduce the anticipatory anxiety associated with "trying" to fall asleep, patients are instructed to get into bed and stay awake rather than to try to fall asleep. Because this intention presumably reduces the anxiety associated with trying to fall asleep, patients should become more relaxed and fall asleep faster than they would otherwise. Paradoxical instructions tend to be most effective with patients who are resistant and reactant to therapeutic suggestions (Shoham, Bootzin, Rohrbaugh, & Urry, 1995). The rationale provided by the therapist for the paradoxical instruction may be a crucial component of its effectiveness. In a meta-analysis of the application of PI to a number of different problems, Shoham-Salomon and Rosenthal (1987) found that rationales that emphasize a positive benefit or the positive qualities of the person having the problem are more effective than rationales that are neutral or that emphasize negative aspects of the problem.

Cognitive Restructuring

Patients with insomnia often subscribe to a number of irrational beliefs about sleep. Examples of these beliefs are that an individual must get at least 8 hours of sleep to feel refreshed and function well the next day; that if an individual goes for one or two nights without sleep he or she will have a nervous breakdown; or that the individual should avoid or cancel social, family, and work obligations after a poor night's sleep (Morin, 1993). Cognitive therapy directed at changing maladaptive attitudes and beliefs is called *cognitive restructuring*. Five types of dysfunctional cognitions are identified. These are (a) misconceptions about the causes of insomnia, (b) misattributions or amplifications of the consequences of poor sleep, (c) unrealistic sleep expectations, (d) diminished perceptions of control and predictability of sleep, and (e) faulty beliefs about sleep-promotion practices (Morin, 1993). Treatment involves providing accurate information and having the patient identify and rehearse alternative belief statements. These techniques have been effective as part of a multicomponent treatment in increasing sleep efficiency and reducing sleep latency, time awake after sleep onset, and early-morning awakening (Morin, Kowatch, Berry, & Walton, 1993).

Thought Stopping and Articulatory Suppression

Most persons with insomnia complain of cognitive intrusions when trying to sleep. Two techniques that have been used to help patients suppress cognitive intrusions are thought stopping and articulatory suppression. In *thought stopping*, the patient says "stop!" forcefully, every time obsessive rumination occurs (Wolpe, 1973). This briefly disrupts the chain of thought, and repetitions decrease the frequency of subsequent cognitive intrusions. A related procedure is *articulatory suppression* (Levey, Aldaz, Watts, & Coyle, 1991). In this procedure, patients are instructed to repeat a word, such as *the*, subvocally three to four times a second until sleep occurs. This technique is based on cognitive research that indicates that articulatory suppression interferes with short-term memory. It has been effectively used in insomnia treatments to reduce cognitive intrusions that interfere with sleep.

A combination of cognitive interventions (PI, thought stopping, and cognitive restructuring) has been found to be effective. In fact, it has been shown to produce as much improvement in sleep as EMG biofeedback or the combination of stimulus control and progressive muscle relaxation training (Sanavio et al., 1990).

Multicomponent Treatments

A commonly used multicomponent package includes stimulus control instructions, relaxation training, and sleep education. In one evaluation, this combination of stimulus control instructions and relaxation training produced more overall improvement than stimulus control instructions alone, although the difference was not statistically significant (Jacobs et al., 1993). This combination has also been evaluated as a self-help treatment delivered by audiotape (Morawetz, 1989). The audiotape treatment produced as much improvement in sleep as did the same treatments with a live therapist for patients who were not taking hypnotics. For those who were taking hypnotics, the live therapist produced more improvement than the tapes.

A multicomponent treatment consisting of stimulus control instructions, sleep restriction, cognitive restructuring, and sleep education has been found to be effective for the sleep-maintenance problems of older adults (Edinger et al., 1992; Morin et al., 1993). Epstein (1994) used this combination of interventions with 22 older patients with insomnia in a 6-week treatment program organized in a small-group format. After treatment, there was significant improvement in waking after sleep onset, sleep efficiency, and total sleep time. These gains were maintained at a 3-month follow-up.

This same combination of interventions (stimulus control instructions, sleep restriction, cognitive restructuring, and sleep education) also lends itself well to clinical settings in which adults of all ages are seen. Chambers and Alexander (1992) reported on the results of treatment for 103 patients who were referred to a sleep disorders clinic. Patients were excluded who were referred for polysomnography for suspected organic sleep disorders (e.g., sleep apnea, periodic limb movements) as were patients for whom treatment required a change in medications. The final sample consisted of 34 men and 69 women with an average age of 39.9 years. The evaluation was conducted on the basis of questionnaires completed before and 6 months after treatment.

Overall, 77.5% of the patients reported improved sleep and 73% reported that the general quality of their lives had improved as a result of treatment.

Similarly, Morin, Stone, McDonald, and Jones (1994) reported on a clinical replication series of 100 patients using the same combination of interventions, with the addition of a separate medication withdrawal component for patients who needed it. The final sample consisted of 36 men and 64 women with an average age of 45.1 years. The patients attended a median of six sessions. Outcome measures were taken from sleep diaries the patients had completed daily during a baseline period and throughout treatment. Patients showed significant improvement on all sleep variables, including sleep-onset latency, time awake after sleep onset, frequency of awakenings, total sleep time, and sleep efficiency. Improvement ranged from 42–50% on the main target symptoms of sleep-onset latency, time awake after sleep onset, and early-morning awakening. These results are comparable to those found in controlled outcome studies. There was a statistically significant but modest gain in total sleep time (from 325 min at baseline to 350 min after therapy). However, there was a substantial gain in sleep efficiency (68% at baseline to 80% after therapy). Therefore, this treatment appears to be more successful at reducing wakefulness and consolidating sleep than increasing the amount of sleep obtained.

Because of the multifaceted etiology of insomnia, the tailoring of treatments to patient needs would seem to be appealing. Each causal mechanism, however, must be tested and evaluated. For example, one might assume that stress-managment techniques should be recommended for a patient who is in a stressful situation and, therefore, experiences tension. Nevertheless, relaxation treatments have not been found to be significantly more effective than other behavioral treatments, even for those who are tense and under stress (Espie, Brooks, & Lindsay, 1989). There is no advantage to tailoring treatments if more effective treatments are omitted and less effective ones are included.

Combining Behavioral and Pharmacological Treatments

Benzodiazepine hypnotics continue to be widely prescribed for insomnia (Shader, Greenblatt, & Balter, 1991). Many patients who request treatment at sleep disorders centers are reluctant to withdraw from hypnotics for fear that their sleep will become substantially worse. This fear is not entirely unfounded; benzodiazepine withdrawal symptoms have been well documented and may last as long as 4 or 5 weeks following abrupt discontinuation (Espie, Lindsay, & Brooks, 1988; Noyes, Garvey, Cook, & Perry, 1988; Rickels, Schweitzer, Case, & Greenblatt, 1990). Withdrawal effects appear to be related to the size of the dose, the length of time the hypnotics are taken, and the pharmacology of the hypnotic (e.g., there are stronger withdrawal effects with short-half-life hypnotics).

In 1993, a new hypnotic, zolpidem (Ambien), was introduced that attaches to one of the two benzodiazepine receptors. However, zolpidem does not have the same side-effect profile as the benzodiazepines and does not appear to produce the same degree of withdrawal symptoms. Consequently, zolpidem may be a better candidate than benzodiazepine hypnotics when pharmacological and behavioral treatments are combined. Caution is recommended, however, because studies involving zolpidem and cognitive–behavioral treatment have not yet been conducted.

Behavioral treatment has been shown to be effective when patients either withdraw from hypnotics or maintain a consistent dose of the hypnotic throughout the behavioral treatment (Espie et al., 1989). There is also an accumulating literature to indicate that behavioral treatments can be used to help patients both withdraw from hypnotics and improve their sleep. Davies (1991) assigned 20 consecutively referred patients to multitreatment cognitive–behavioral group therapy. The treatments consisted of self-monitoring by means of daily diaries, sleep education, progressive relaxation, and stimulus control instructions. Patients were seen in groups of five for 11–13 weekly sessions. The composition of the groups was heterogeneous for gender, age, and whether or not patients were currently taking hypnotics. Twelve of the 20 patients were taking hypnotics at the beginning of the study, and 10 of those 12 took hypnotics daily. At posttreatment, 9 patients had stopped taking hypnotics altogether, 2 were taking hypnotics once a week, and 1 was taking a reduced dose six nights a week. This is a substantial reduction in the use of medication for sleep. In addition, the patients' sleep had improved. The 12 patients who had been taking hypnotics increased their total sleep by more than an hour and a half and reduced the number of nights they had difficulty falling asleep from more than four nights to less than two nights a week. This degree of improved sleep was comparable to that achieved by those receiving the behavioral treatment who had not been taking hypnotics.

Relaxation training as a primary intervention has also been shown to help reduce hypnotic use in community elderly (Lichstein & Johnson, 1993) and in residents of aged-care facilities (Gilbert, Innes, Owen, & Sansom, 1993). Although sleep did not get worse as participants reduced their use of hypnotics, it did not show the same degree of improvement as reported by Davies (1991). It is possible that relaxation is not as powerful a treatment for sleep disturbance as the multitreatment approach used by Davies (1991).

In recent years, a number of researchers have compared pharmacological and cognitive–behavioral therapies for the treatment of depression and panic disorders. A general conclusion from that literature is that both types of treatments reduce symptoms, with the pharmacological therapies having an earlier effect than the cognitive–behavioral therapies. The cognitive–behavioral treatments, however, appear to have an advantage in the maintenance of treatment gains (Hollon, Shelton, & Loosen, 1991; Michelson & Marchione, 1991).

In the insomnia literature, there have been few controlled studies that compare a hypnotic with a nonpharmacological treatment. In a comparison of triazolam with a combination of stimulus control instructions and relaxation training, triazolam had an immediate effect, whereas the nonpharmacological treatment took 3 weeks to have an equivalent effect (McClusky, Milby, Switzer, Williams, & Wooten, 1991). At a 1-month follow-up, however, the nonpharmacological treatment was found to be more effective than triazolam at maintaining improvement. The differential course and effects of pharmacological and behavioral treatments for insomnia suggested that it might be possible to use them together. To examine this hypothesis, the investigators assigned 15 patients to treatment with either triazolam with behavioral therapy or triazolam with sleep-related information (Milby et al., 1993). At follow-up, triazolam plus behavioral therapy produced greater improvement in total sleep and restedness in the morning than did triazolam plus sleep-related information. These results suggest that hypnotics and behavioral therapy might be effectively combined.

The possibility of using hypnotics and cognitive–behavioral treatment together was also evaluated in a large, controlled study by Morin, Colecchi, Stone, Sood, and Brink (1995). Seventy-eight community-resident older adults (average age of 64.5 years) with chronic insomnia were randomly assigned to temazepam pharmacotherapy, cognitive–behavioral therapy, combined pharmacotherapy and cognitive–behavioral therapy, and a medication placebo group. All treatments lasted 8 weeks. Cognitive–behavioral therapy consisted of "cognitive restructuring aimed at altering dysfunctional beliefs and attitudes about sleep and combined stimulus control and sleep restriction aimed at regulating sleep schedules, curtailing sleep-incompatible behaviors, and consolidating sleep over shorter periods of time spent in bed" (p. 303).

The results at posttest were that patients in all three active treatment groups had improved sleep when compared with those receiving the placebo treatment. The combined treatment showed a nonsignificant trend to produce more improvement than the cognitive–behavioral treatment, which was itself nonsignificantly superior to the pharmacotherapy. The pattern of results was similar for both sleep diary and polysomnography measures.

Even though cognitive–behavioral and pharmacological therapies, either alone or in combination, were about equally effective in the short-term treatment of insomnia, patients reported greater subjective improvement and more satisfaction with the cognitive–behavioral and combined treatment than with the pharmacotherapy or placebo condition. Furthermore, preliminary results at the 12-month and 24-month follow-ups suggested that only patients who had received cognitive–behavioral treatment alone maintained their gains over time. Patients who had received either pharmacotherapy or the combined treatment showed more relapse. It is possible, therefore, that pharmacotherapy works in the short run but undermines the capacity of cognitive– behavioral treatment to maintain improvement over the long term. Perhaps some patients in the combined treatment group attributed their improvement to the medication rather than to the skills acquired through cognitive–behavioral therapy. This perception might result in their becoming more vulnerable to relapse (Davison, Tsujimoto, & Glaros, 1973).

It is too early to conclude whether or not pharmacotherapy and behavioral therapies can be effectively combined in the treatment of insomnia. More studies of the quality of that by Morin et al. (1995) are needed. However, caution in the prescription of hypnotics and other substances for sleep would be prudent. Cognitive–behavioral treatments have typically been found to be most effective with patients who are not currently taking hypnotics (Murtagh & Greenwood, 1995).

Conclusion

In recent years, there have been many controlled studies that examined the efficacy and durability of psychological treatments for chronic insomnia. Two separate meta-analyses have been published (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995). Both meta-analyses concluded that psychological treatments have been found to improve the sleep of patients with insomnia, when compared with control conditions, and improvements were maintained at 6-month follow-ups. Overall, treatment reduced the average sleep-onset latency from 64.3 to 36.6 minutes and reduced the average time awake after sleep onset from 70.3 to 37.6 minutes (Morin, Culbert, & Schwartz, 1994).

Murtagh and Greenwood (1995) concluded that there are few differences in efficacy among the active treatments, although stimulus control instructions had the strongest effects. Morin, Culbert, and Schwartz (1994) concluded that both stimulus control instructions and sleep restriction, as single-component treatments, had the strongest effects. Multiple-component treatments were often just as effective, but they did not exceed stimulus control or sleep restriction in effectiveness. PSG measures were included in only about 15% of the studies reviewed. Consequently, the results of the meta-analyses were based on information recorded in daily sleep diaries. However, the results from studies using PSG were similar in pattern, although with smaller effects, to those found in the larger group of studies (Morin, Culbert, & Schwartz, 1994).

Stronger treatment effects were seen in those who were not regular users of hypnotics and in clinically referred patients compared with volunteers (Murtagh & Greenwood, 1995). It is often difficult to withdraw patients with chronic insomnia from hypnotics; therefore, it is generally a wise policy not to prescribe hypnotics for sleep problems to begin with. Neither age nor sex was related to outcome in either meta-analysis. Because many patients with insomnia are older adults, it is encouraging that cognitive–behavioral treatments can be used effectively for them as well.

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