

Clinical Update on Epidemiology, Pathogenesis, and Nonpharmacological Treatment of Insomnia / *İnsomninin Epidemiyolojisi, Patogenezi ve Nonfarmakolojik Tedavisi Üzerine Bir Güncelleme*

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ABSTRACT

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Scientific background: Insomnia is by far the most common form of sleep disturbance. Most typically, insomnia has been defined as the symptom of difficulty initiating or maintaining sleep and more rarely as an inability to obtain restorative sleep. Insomnia disorders are most often classified as either primary or secondary to other sleep, psychiatric, or medical conditions, although it is often difficult in practice to determine true causality of insomnia or there may be more than one cause (comorbid conditions). Increasing age, female sex, and psychiatric and medical disorders are consistent risk factors for insomnia. Insomnia is associated with significant social, medical, and financial consequences including impaired social functioning and quality of life, increased risk for psychiatric disorders, and increased health care costs. The clinical assessment of insomnia is based on a careful clinical interview, often supplemented by sleep questionnaires, sleep logs, and psychological testing. Polysomnography is indicated only in selected cases when specific sleep pathologies are suspected.

Assessment: A large proportion of insomnia sufferers go undiagnosed, and therefore untreated, by their doctors, and many of these patients

incur considerable personal, vocational, and health-related consequences as a result. Insomnia can be triggered by a variety of precipitating events, but when it becomes a chronic problem, psychological and behavioral factors are almost always involved in perpetuating or exacerbating sleep disturbances over time. Psychological and behavioral therapies for primary insomnia include sleep restriction, stimulus control therapy, relaxation training, cognitive strategies, and a combination of those methods, referred to as cognitive behavior therapy of insomnia. Results of the controlled clinical trials indicate that 70% to 80% of patients with primary insomnia partially benefit from cognitive behavior therapy. Although only 20% to 30% of patients become completely symptom free and can be called as "good sleepers". The clinical studies showed that sleep improvements are well maintained up to 2 years after treatment completion.

Conclusion: Despite evidence showing psychological and behavioral approaches to be efficacious in the treatment of insomnia, in today's clinical practice, such therapies are not readily available and are not widely used by doctors.

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ÖZET

Bilimsel zemin: Insomnia en sık görülen uyku bozukluğudur. Insomnia, yaygın olarak uykuya başlama zorluğu ya da uyku bütünlüğünün bozulması (uyku devamlılığı zorluğu) olarak tanımlanabilir ise de daha nadir olarak uykunun dinlendirici olmaması olarak da tanımlanmıştır. Insomnia, her ne kadar, çoğunlukla ya primer bir uyku bozukluğu ya da diğer uyku, psikiyatrik, veya tıbbi rahatsızlıklara bağlı sekonder olarak gelişmiş bir uyku bozukluğu olarak sınıflanabilirse de, pratikte insomnia semptomunun altında yatan gerçek nedeni bulmak sıklıkla zordur ya da birden fazla neden olabilir. Yaşlı yetişkinler, bayanlar, ve psikiyatrik veya tıbbi rahatsızlıkları olanlar insomnia risk grubundadır. Insomnia, kişinin sosyal fonksiyonlarında bozulma ve yaşam kalitesinde düşme, psikiyatrik bozukluklar geliştirmeye eğilimi, ve artmış sağlık harcamaları gibi ciddi sosyal, tıbbi, ve finansal sorunlara neden olabilir. Insomnia hastasının klinik değerlendirmesi, (sıklıkla uyku bozuklukları soru formu, uyku çizelgesi, ve psikolojik testler ile desteklenmiş) dikkatlice alınmış bir anamnez üstünde temellendirilir. Polysomnography, sadece belirli uyku bozukluklarından şüphelenilen seçilmiş vakalarda endikedir.

Değerlendirme: Insomnia hastalarının büyük bir bölümü doktorları tarafından teşhis edilmemiş ve dolayısı ile tedavi edilememiş olduğundan, bu hastaların çoğu hatırı sayılır oranda kişisel, iş yaşamı ve sağlıkla ilgili problemlere maruz kalırlar. Insomnia değişik presipite edici olaylar tarafından tetiklenebilir, ama insomnia bir kere kronik hale geldiğinde, psikolojik ve davranış faktörleri, uyku bozukluğunun zaman içinde daha da müzminleşmesinde ve kötüleşmesinde, hemen her zaman, rol oynar. Primer insomnia'nin psikolojik ve davranış terapileri su yöntemleri kapsar; uyku sınırlama, uyaran control tedavisi, relaxation eğitimi, cognitive stratejiler, ve bu yöntemlerin bir kombinasyonu olabilir, ki bu aynı zamanda insomnia'nin cognitive-behavior tedavisi olarak da adlandırılır. Kontrollü klinik çalışmalarının sonuçları, primary insomnia hastalarının %70-80'inin cognitive-behavior tedavisinden kısmen yararlandığını göstermektedir. Buna rağmen bu hastaların sadece %20-30'u tüm insomnia semptomlarından tamamiyle kurtulup "normal uyku" ya döner.

İzlenimler: Klinik çalışmalar, uykudaki bu düzelmenin, tedavinin bitmesinden iki sene sonraya kadar oldukça iyi muhafaza edildiğini göstermiştir. Kanıtlar, insomnia tedavisinde psikolojik ve davranış yaklaşımlarının etkili olduğunu göstermesine rağmen, bu tedaviler günümüzde kolay ulaşılabilir değildir ve doktorlar tarafından yaygın olarak kullanılmamaktadır.

DEFINITION

Insomnia has been defined by complaints of disturbed sleep in the presence of adequate opportunity and circumstance for sleep. This definition firstly requires elimination of voluntary sleep restriction and sleep deprivation. The sleep disturbance may consist of one or more of these features:¹ difficulty in initiating sleep (sleep onset insomnia);² difficulty in maintaining sleep; waking up frequently during the night and with difficulty returning back to sleep (sleep maintenance insomnia); or³ waking up too early; a final awakening that occurs much earlier than desired (sleep offset insomnia).⁴ A fourth characteristic, nonrestorative or

poor-quality sleep, has frequently been included in the definition. Insomnia patients also complain of associated daytime complaints such as fatigue, mood disturbance, cognitive difficulties, and social or occupational impairment.¹

For research purposes, insomnia is arbitrarily defined as a delay of more than 30 minutes in sleep onset or a sleep efficiency (the ratio of time asleep to time in bed) of less than 85 percent. However, in clinical practice, a patient's subjective judgment of sleep quality and quantity is a more important factor.

When temporal course is considered, transient insomnia can be defined as above symptoms lasting less than one week, and short-term insomnia one to four weeks. Chronic insomnia is defined, more variably, as insomnia lasting more than 1 - 6 months.

Before epidemiology of insomnia, we may briefly review sleep habits of the general population. In a recent article,² to obtain a snapshot of sleep habits around the world, questionnaires from 35,327 adults in 10 countries on four continents were compiled. This was the largest survey ever reported on sleep habits in the general population. People around the world reported going to bed at 11 p.m. and sleeping about 7.5 hours on weeknights on average. The average reported wake-up time was 6 a.m. on weekdays, and 8 a.m. on weekends. About one in four people reported that they took regular naps that lasted about an hour. While the mean sleep latency for all subjects was 25 minutes on weeknights, over 13% of respondents said they took at least an hour to fall asleep. One person in four reported chronic poor sleep. Among self-reported poor sleepers, less than one-third on average sought a physician's help. Less than one-third on average said they took medications to help sleep. Some drank herbal teas or alcohol to improve sleep; some modified their caffeine consumption.

EPIDEMIOLOGY

Prevalance: Insomnia has been named as the most frequent health complaint after pain. Insomnia is by

far the most common form of sleep disturbance. A 1979 US Gallup poll showed that 95% of a randomly selected adult sample reported having experienced insomnia at some time during their lives. However, for most of these people, insomnia represented a transient problem that resolved quickly when the precipitating circumstances abate.

The majority of the people with insomnia is undiagnosed and untreated; a 1991 National Sleep Foundation Survey showed that only 5% of all insomnia sufferers saw their physicians specifically for their sleep problems. Another survey showed that only 30% of all insomnia sufferers ever discussed their sleep problems with their doctors.³

Chronic insomnia (when defined as insomnia lasting more than one month) has a prevalence of 10 to 15 percent and occurs more frequently in women, older adults, and patients with chronic medical and psychiatric disorders.^{4,5}

Genetics: Yves et al. found that 48.8% of 256 insomnia sufferers reported that one or more first-degree relatives had insomnia, whereas only 23.5% of 90 individuals without insomnia reported insomnia in a first-degree relative.⁶

Morbidity: Compared to those without sleep/wake complaints, those with persistent insomnia report a greater sense of daytime fatigue, more anxiety or stress, poorer mood, greater coping difficulties, decreased memory and concentration, impaired psychomotor performance, less ability to complete tasks, decreased productivity, occupational difficulties, greater tendency to suffer work-related or traffic accidents, greater impairment of family or social functioning (irritability and problems with interpersonal relationships), poorer quality of life, and greater health care utilization. Insomnia sufferers make significantly more visits to practitioners in both the medical and psychiatric clinics than do those without any sleep complaints.

Several studies have shown that the presence of insomnia at the time of an initial interview enhances

risk for a major depressive disorder occurring between 1 and 3.5 years after that interview.^{7,8} Other than depression; additionally, longitudinal data have shown that insomnia sufferers are at risk for anxiety/panic disorders and substance abuse problems as well.⁸ One recent population-based study found that the symptom of insomnia appeared before the onset of a first episode of mood disorder in 41% of cases and before the onset of an anxiety disorder in 18% of cases.⁹

Risk Factors for insomnia:¹ Age; as high as 50% of older adults may complain from insomnia.² Sex; women are more prone to present with insomnia complaints than are men (twice as common in women as in men).³ Marital status; most studies suggest that divorced/separated or widowed individuals are at increased risk for insomnia compared with married individuals.⁴ Income; several studies have suggested that those who are unemployed are significantly more likely to report insomnia than are those who are employed.⁵ Other risk factors include; a stressful lifestyle, physical inactivity, irregular bedtimes, alcohol dependence, and heavy caffeine use. Cigarette smoking also appears to be a risk factor for insomnia symptoms, and smoking cessation has been shown to predict insomnia remission.¹⁰

ETIOLOGY AND PATHOPHYSIOLOGY OF INSOMNIA

The pathogenesis of primary insomnia is unknown, but available evidence suggests a state of hyperarousal; that is, the patient has a level of arousal that is incompatible with the initiation or maintenance of sleep. Physiologic, cognitive, and cortical arousal each play a role in the etiology of insomnia. There are few general models of the etiology and pathophysiology of insomnia. We will briefly review physiologic and behavioral models.

Physiologic model of insomnia: Studies evaluating physiologic arousal in insomnia have used a variety of techniques, including basic psychophysiological measures; heart rate, respiration

rate, skin and core body temperature, muscle tone, skin conductance and resistance, and peripheral blood flow or vasoconstriction. These studies showed that poor sleepers exhibit increased physiologic arousal.

Patients with primary insomnia exhibited significantly higher whole-body metabolic rate (using a measure of oxygen consumption) than good-sleeper controls across the 24-hour day and during the sleep interval.¹¹

Neuroendocrine measures, and hypothalamic-pituitary-adrenal HPA axis and sympathetic nervous system activity is associated with insomnia. Patients with insomnia had significantly higher mean levels of ACTH and cortisol over the course of the 24-hour day, with the largest group differences observed in the evening and first half of the night.¹²

Functional neuroimaging studies: Patients with insomnia exhibited increased global cerebral glucose metabolism during wakefulness and NREM sleep.¹³

Other researches showed that insomnia patients may have increased beta activity and decreased theta and delta activity on electroencephalography during sleep.

Behaviour models of insomnia: Spielman, in his 1987 article, described a behavioral insomnia model that is widely accepted.¹⁴ This is also alternatively referred to as the three-factor model or the three-P model (predisposing, precipitating, and perpetuating factors.)

In brief, this model postulates that insomnia occurs acutely in relation to both traits (predisposing factors) and major life stresses (precipitating factors or "triggers") and that the chronic form of the disorder is maintained by maladaptive coping strategies (perpetuating factors). To explain this in more detail: A person may be prone to insomnia due to trait characteristics (genetic predisposition or a certain degree of inherited hyperarousal state). This person may experience acute episodes because

of precipitating factors (new onset medical or psychiatric illness or major life stresses such as loss of a loved one), and later may suffer from a chronic form of the disorder because of behavioral factors (perpetuating factors), which will be described below.

Perpetuating factors refer to the maladaptive coping strategies that the patient adopts to compensate for sleep loss. Though, against the patient's will, these maladaptive strategies may actually worsen insomnia. Research and treatment have focused on two kinds of perpetuating factors: the practice of staying in bed while awake and the tendency to extend sleep opportunity (going to bed earlier or by getting up later, or both.) They may extend sleep opportunity but not necessarily sleep ability and the patient ends up staying in bed awake for hours. This may cause a negative conditioning with the patient's sleep environment.

Insomnia can be triggered by a variety of precipitating events, but when it becomes a persistent problem, psychological and behavioral factors are almost always involved in perpetuating or exacerbating sleep disturbances over time. When insomnia becomes chronic, then the person worries about the inability to fall sleep and the consequences of sleep loss. These worries may prevent the person from relaxing and falling asleep and also contributes to hyperarousal state. Are these behavioral models valid? Probably yes, because the treatments based on this model's principles are usually successful.

ASSESSMENT

A careful clinical interview is the foremost component of an insomnia evaluation. If possible, interviewing the patient's bed partner about the patient's sleep pattern and habits can reveal important diagnostic information such as symptoms of other sleep disorders (snoring, witnessed apneas, PLM) and to confirm the patient's sleep pattern. Because insomnia is a subjective complaint, the use of subjective assessment methods such as self-

report questionnaires and sleep logs is needed. Medical history and physical examination are important in establishing the presence of comorbid syndromes. Other than self-report questionnaires and sleep logs, we can also use more objective methodologies such as actigraphy and polysomnography. Polysomnogram may play an important role in diagnostic decisions in selected patients only. Actigraphy and Polysomnogram are not recommended for the routine diagnostic evaluation of insomnia. On the other hand, sleep log is recommended in the recent practice parameters for insomnia evaluation.

Clinical Interview: Components of a clinical interview for insomnia include detailed sleep history and assessment; nature of complaint (onset, course, pattern, and severity), physiologic or cognitive arousal at bedtime (trouble unwinding, ruminating thoughts), daytime symptoms, symptoms of other sleep disorders, factors that exacerbate insomnia or improve sleep pattern, sleep environment, sleep-incompatible behaviors, bedtime routines, daily activity and exercise pattern, patient's beliefs about the importance of sleep and cause of insomnia, treatment expectations, and previous treatment history (self-treatment attempts, coping strategies, and response to treatments). During clinical interview, also medication and substance use and past medical and psychiatric history should be reviewed.

Self-Report Questionnaires: Sleep questionnaires may be helpful, particularly when combined with other assessment methods such as sleep logs or actigraphy. These questionnaires should review the following insomnia-related factors: Sleep hygiene behaviors, sleep quality, satisfaction with sleep, daytime sleepiness, functional impairment, sleep-related beliefs and attitudes, and insomnia severity.

Sleep Log: Insomnia assessment should routinely include a sleep log completed for 1 to 2 weeks. Sleep log is simple and inexpensive. The sleep log or diary provides a subjective record of an individual's perceived sleep pattern and has become an essential

tool in the assessment of insomnia. On a typical sleep log; bedtime, falling asleep time, nighttime awakenings, time to get back to sleep, waking up time, getting out of bed time, and naps are marked. It allows the physician to easily calculate sleep onset latency, total wake and sleep time, number of awakenings, time in bed, and therefore sleep efficiency. Additional information such as sleep quality, stimulant use (coffee, caffeinated soft drinks, tea, and chocolate), alcohol consumption, and sleep medication use can be easily documented by the patient in sleep log. Compared to a patient's retrospective report during the clinic visit, sleep log, which has been compiled daily, provides more accurate information about sleep-wake patterns and insomnia severity. Sleep log may reveal circadian changes or insufficient sleep. Sleep logs can also be used over long time periods to estimate treatment effects.

Psychological Testing: Psychological screening tests are often useful to rule out depression and anxiety as causes of insomnia. Because of the high prevalence of mood and anxiety symptoms among patients with insomnia, routine psychological screening is recommended. Brief psychological questionnaires such as the Beck Depression Inventory (BDI) can easily administered.^{15, 16} The BDI is a self-administered 21 item self-report scale measuring supposed manifestations of depression. The BDI takes approximately 10 minutes to complete. The original version of the BDI was introduced in 1961 and was revised in 1971.

Actigraphy: Actigraphy is a recording device that looks like a wristwatch. It measures the body movements and sleep estimates are based on these measurements. There would be much less wrist movements during sleep and inactivity periods in an actigraphy printout and these periods are assumed as probable sleep. It is inexpensive and sleep-wake data can be collected over extended periods, usually 1-3 weeks, in the home environment. Current standards of practice recommend a minimum recording period of 3 days with concurrent use of a sleep log. Like polysomnography, actigraphy is not

recommended for the routine diagnostic evaluation of insomnia. However, if we need better and more objective documentation of sleep pattern, or for certain patients, which could not reliably complete a detailed sleep log, then actigraphy may be considered. This information, like in sleep log, can be used to assist in diagnosis, to document severity of an insomnia problem, to measure treatment outcome, and to monitor the patient's compliance to treatment recommendations.

Polysomnography: Polysomnography is not usually necessary or indicated for the routine evaluation of chronic insomnia unless there exists a valid indication and clear rationale. Polysomnography should be considered only in cases with symptoms of other sleep pathologies (e.g., sleep-disordered breathing, periodic limb movements, or parasomnias), pathologic levels of daytime sleepiness, or failure to respond to insomnia treatment.¹⁰

DIFFERENTIAL DIAGNOSIS

Insomnia disorders are most often classified as either primary or secondary to other disorders, although it is often difficult in practice to determine true causality. Because insomnia can arise from varied causes (e.g., psychiatric, medical i.e. chronic pain, substance use, cognitive, or behavioral) and is often secondary to some other disease process, consideration of differential diagnoses is a critical element in successful treatment planning since the treatment of each insomnia subtype will be different.

When a patient presents with fatigue and tiredness. First, we have to rule out insufficient sleep syndrome (voluntary sleep restriction), since it is the most common reason for sleep complaints in today's demanding world. Various sleep pathologies also can present clinically as an insomnia complaint. Primary sleep disorders such as sleep-related breathing disorders (obstructive sleep apnea syndrome, central sleep apnea syndrome), restless legs syndrome, and periodic limb movement disorder can cause sleep onset difficulties, sleep

fragmentation, and nocturnal awakenings that lead to insomnia complaints. Insomnia is also a prominent symptom of most circadian rhythm disorders (jet lag disorder, shift work disorder).

Approximately half of individuals with chronic insomnia have a current psychiatric diagnosis or past psychiatric history.⁹

Psychiatric disorders that may have comorbid insomnia include: Mood disorders; 40-90% may have sleep disturbance. Generalized anxiety disorder and panic disorder; usually presents with sleep-onset or sleep maintenance insomnia due to sudden awakenings from sleep with intense fear. Post-traumatic stress disorder. Substance use disorders and alcohol abuse and dependency.

Some examples of the medical disorders that may have comorbid insomnia include: Neurologic disorders; dementia, epilepsy, and Parkinsonism. Respiratory; COPD and asthma. Gastrointestinal; sleep-related GERD and PUD. Cardiac; nocturnal cardiac ischemia and heart failure. Pain syndromes; fibromyalgia and arthritis. Hyperthyroidism and menopause.

Common drugs or substances that can cause insomnia include: Corticosteroids, thyroid hormones, decongestants, bronchodilators, beta blockers, calcium channel blockers, some antidepressants (SSRI), some anticonvulsants, alcohol, smoking or nicotine patch, excessive consumption of caffeine and chocolate.

NON PHARMACOLOGIC (PSYCHOLOGICAL AND BEHAVIORAL) TREATMENT OF INSOMNIA

Despite its high prevalence and negative impact, insomnia often goes unrecognized and remains untreated, with less than 15% of those with severe insomnia receiving any treatment.¹⁷

Most patients who initiate treatment do so without professional consultation, self treatment, with herbal, or dietary supplements or over the counter medications

like some cold medications or antihistaminics. When insomnia is brought to professional attention, typically to a primary care physician, treatment is often limited to medication. Although hypnotic medications are clinically indicated and useful in selected situations, psychological and behavioral factors are almost always involved in perpetuating sleep disturbances in chronic insomnia, and these factors should be addressed in the management of insomnia. In current practice, psychological and behavioral management of insomnia (cognitive-behavioral therapy) is not widely available and remain underutilized by health care practitioners.

Evidence from controlled clinical trials indicates that 70% to 80% of patients with primary insomnia benefit from cognitive-behavioral therapy; they achieve significant symptom reductions on sleep onset latency and on wake after sleep onset, with the absolute values of those parameters returning to below or near the 30-minute cutoff criterion typically used to define insomnia. With the aid of cognitive-behavioral therapy, total sleep time is increased by a modest 30 minutes to, in some studies, 45 minutes. Treatment also increases sleep satisfaction and reduces psychological symptoms and hypnotic usage. Effect is long lasting compared to medication therapy alone.¹

Treatment options for insomnia include basic sleep hygiene education, psychological and behavioral interventions, pharmacotherapy, and a variety of complementary and alternative therapies (acupuncture, hypnosis). In this review article, the first two will be discussed in detail. Psychological and behavioral interventions for primary insomnia include; stimulus control therapy, sleep restriction, relaxation-based interventions (relaxation training), cognitive strategies, and a combination of those methods, referred to as cognitive behavior therapy.

Sleep Hygiene Education: Inadequate sleep hygiene is rarely the primary cause of insomnia, though it may potentiate sleep difficulties caused by other factors, or it may interfere with treatment progress, so it should definitely be addressed. In general, these are common sense rules for better

sleep:

- Maintain a regular sleep schedule (including the weekend days). Resist naps, especially if you have trouble falling asleep. No naps after 3-4 PM and no naps more than 30-40 minutes long.
- If you must snack before bedtime, prefer carbohydrates (crackers, bread) or dairy products. No heavy meals close to bedtime.
- Allow at least a 1-hour period to unwind before bedtime; do not go directly from work to bed (allowing enough time to relax before bedtime). Do something relaxing in the half-hour before bedtime (bedtime rituals). Reading, meditation, etc are all appropriate activities.
- Exercise regularly; especially in late afternoon or early evening. A low point in energy occurs a few hours after exercise; sleep will then come more easily. Though exercise should not be too close to the bed time, which may increase alertness.
- Avoid stimulants (e.g., caffeine, nicotine) for several hours before bedtime. Caffeine may have a variable half life (as long as 5-6 hours) in different individuals. Sensitive persons may not use caffeinated drinks after noontime.
- Drink little or no fluids after 7-8 PM, so that sleep is not disturbed by the need to urinate.
- Avoid alcohol around bedtime, as it fragments sleep. Alcohol decreases sleep onset latency, though in 2-4 hours when the alcohol is eliminated, then the person may awaken and experience difficulty getting back to sleep.
- Take a hot bath about an hour and a half to two hours before bedtime. This alters the body's core temperature rhythm and helps people fall asleep more easily and more continuously. (Taking a bath shortly before bed may increase alertness.)
- Keep the bedroom environment dark and quiet, may need to use earplugs if necessary or may consider using a white noise machine or a fan to drown out other sounds. Use a comfortable mattress and pillow. Keep the bedroom well ventilated. Adjust the room temperature (sleep is better in a cool room, around 18 – 20°C)

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- Do not fixate on the bedside clock. Don't let yourself repeatedly check the time while trying to fall asleep! Obsessing over time will just make it more difficult to sleep. Can turn the clock around or put it under the bed.
 - If you get up during the night to use the bathroom, use minimum light.

Sleep hygiene education may be helpful for mild insomnia, though it is rarely sufficient (as the sole treatment) for more severe and chronic forms.

Psychological and behavioral interventions

Comparative studies of monotherapies have shown that stimulus control and sleep restriction therapies are slightly more effective than relaxation or paradoxical intention. On the other hand, sleep hygiene education produces little impact on sleep when used as the only intervention.

Stimulus Control Therapy: Stimulus control is one of the most widely used interventions for insomnia. The rationale of stimulus control therapy is that repeated and unsuccessful sleep attempts (such as spending 1-1.5 hour in bed awake and struggling to fall asleep) eventually may lead to conditioning; a negative association between the bedroom environment and sleep. This conditioning process may take place over several weeks or months and may be unrecognized by the patient. Over time, (after many unsuccessful sleep attempts over the weeks), the pre-sleep rituals like eating or TV watching in bed become cues or stimuli for arousal rather than for relaxation and sleep.

Stimulus control therapy consists of a set of three instructions. The goal is to re-associate bed and bedroom with rapid sleep onset and to reestablish a consistent sleep-wake schedule.

These instructions are:

(1) Go to bed only when sleepy (not just fatigued, but sleepy). Get out of bed when unable to sleep

(e.g., after 15-20 min of trial), go to another room and involve in activities (such as reading a book in a dim light) that will not promote wakefulness. Return to bed only when sleep is imminent. (This may need to be modified in older adults with difficulty ambulating.)

(2) Use the bed/bedroom for sleep (and sex) only. No sleep-incompatible activities in bed such as; eating, TV watching, radio listening, planning, or problem solving.

(3) Arise at the same time every morning (including weekends) regardless of the amount of sleep the night before. Avoid daytime napping.

Sleep Restriction: People with insomnia tend to increase the amount of time spent in bed in an effort to provide more opportunity for sleep; a strategy that is more likely to result in fragmented and poor-quality sleep. Sleep restriction therapy consists of limiting the amount of time spent in bed to the actual amount of time asleep. For example, if a person reports sleeping an average of 6 hours per night out of 8 hours spent in bed, the initial prescribed sleep window (i.e., from initial bedtime to final arising time) would be 6 hours. This 6 hour of sleep window is subsequently increased by about 15 to 20 minutes per week when sleep efficiency (total sleep time divided by time in bed) exceeds 85%. Or sleep window is decreased by the same amount of time when sleep efficiency (SE) is lower than 80%. Sleep window will be kept stable when SE falls between 80% and 85%. These adjustments are made weekly until optimal sleep duration is achieved. Initially this may create some mild sleep deprivation, though sleep will be more consolidated and more efficient. To prevent excessive daytime sleepiness, time in bed should not be reduced to less than 5 hours per night. The rules may be a bit flexible for older adults and may allow 30 minute naps.

Relaxation-Based Interventions: The goal of this treatment is to reduce arousal at bedtime. This can be achieved in several ways. Progressive muscle relaxation focus primarily on reducing somatic arousal. There are many commercial audiotapes available. Passive relaxation is an alternative to

progressive muscle relaxation (especially in the older adults with joint and muscle pain); its procedures omit the sequential muscle tensing of progressive relaxation and employ passive body focusing.

On the other hand, attention-focusing procedures (e.g., imagery training, meditation, thought stopping) target mental arousal in the form of worries, intrusive thoughts, or a racing mind. Or the patient can be instructed to engage in any activities that he or she finds relaxing shortly before bed or while in bed. This can include listening to a relaxation tape, soothing music, or a pleasant image.

Cognitive Therapy: Insomnia is often exacerbated by excessive preoccupation with sleep and by apprehensions and monitoring of the next-day consequences. Actually these thoughts can heighten arousal and interfere with sleep. For example, when a person is unable to sleep at night and begins thinking about the possible consequences of sleep loss on the next day's performance, then this feeds into a vicious cycle of insomnia, emotional distress, and more sleep disturbance. Cognitive therapy is designed to break this vicious cycle. Treatment targets include unrealistic expectations ("I must sleep 8 hours every night"), and amplification of the consequences of insomnia ("Insomnia may have serious effect on my health" or "I can't have a normal day after a sleepless night"). Certainly, the following day of sleepless night will be a more difficult day, though still sleep should not be used as a scapegoat and insomnia should not be attributed as the only cause of all impairments. The main therapeutic message to patients: Do not blame insomnia for all daytime impairments; do not give too much importance to sleep; never try so hard to sleep; and keep realistic expectations.

One of the cognitive strategies is paradoxical intention. This is designed to eliminate performance anxiety. Some people, perhaps the more perfectionist ones, may take the relaxation attempt (prior to sleep) so seriously and may have a paradoxical response and actually become more anxious when trying to relax.

Any excessive attempt to induce sleep voluntarily (trying so hard to fall asleep) is likely to generate a performance anxiety and to delay sleep onset in some patients. With paradoxical intention, the patient is instructed to remain passively awake and to give up any effort or intention to fall asleep.

To minimize mental activity at bedtime and in the bedroom surroundings, we may ask the patients to set aside a time and a place (other than bedtime and the bedroom) to write down thoughts or worries of the day and plans for the next day.

Multicomponent therapy (or Cognitive Behaviour Therapy): This approach typically includes an educational component (sleep hygiene), a behavioral component (stimulus control, sleep restriction, and, sometimes, relaxation based interventions), and a cognitive component. For typical patients with primary insomnia, direct consultation time varies between 4 and 6 hours per patient, which is usually spread over a treatment period of 6 to 8 weeks.

Findings from more than 50 clinical trials (more than 2000 patients) conducted in the 1980s and early 1990s and evaluating nonpharmacologic interventions for insomnia have been summarized in at least three meta-analyses^{18,19,20} and also in a review/practice parameters paper commissioned by the American Academy of Sleep Medicine.⁵

Evidence from these different sources shows that psychological and behavioral treatments produce reliable changes in several sleep parameters; sleep onset latency, number of awakenings, duration of awakenings, total sleep time, and sleep quality ratings. These data indicate that approximately 70% to 80% of patients with insomnia benefit from psychological and behavioral treatments. In terms of absolute changes, treatment reduces subjective sleep-onset latency from an average of 60 to 65 minutes at baseline to about 35 minutes after treatment. The duration of awakenings is decreased from an average of 70 minutes at baseline to about 38 minutes after treatment. Total sleep time is increased by a modest 30 minutes, from 6 hours to

6.5 hours after treatment. Thus, for the average patient with insomnia, with cognitive behavioral treatment, sleep onset latency and wake after sleep onset may decrease by an average of about 50% and it brings the absolute values of those sleep parameters below or near the 30-minute cutoff criterion typically used to define insomnia. Most studies indicate that these improvements in sleep latency and wake after sleep onset continue to be observed long after treatment and are well maintained up to 12, 24, and even 36 months later. Although the majority (70-80%) of patients benefit from treatment, only a small proportion (20% to 30%) of them achieve full remission. In other words, the majority may continue to experience residual sleep disturbances.

Suitability in Clinical Practice: Most of the insomnia patients are typically seen by family physicians. An important question; are these cognitive behavior treatments (CBT) also effective and feasible in purely primary care clinical settings? In a clinical effectiveness trial^{21, 22}, nurse practitioners were trained to provide group CBT to patients with insomnia seen in primary care practices. The results showed an average reduction of sleep onset latency from 61 minutes to 28 minutes after treatment, with similar improvements on time awake after sleep onset. In addition, 84% of patients initially using hypnotics remained drug free at a 1-year follow-up.²²

Self-help approaches using printed materials, brief, 15 min, phone consultations, internet-based interventions, or videotapes have also been shown to be useful in the treatment of insomnia.^{23, 24} These are cost-effective ways to enhance treatment access and facilitate its implementation in daily clinical practice.

In conclusion; the patient should be informed about the nature of behavioral treatment, its rationale, and the importance of the patient's involvement in and compliance with the treatment. The patient's expectations should be discussed before treatment onset and the patient should be provided with realistic outcome expectations and with a

conceptual framework (predisposing, precipitating, and perpetuating factors) to explain how insomnia may have evolved into a persistent problem over time. The patient should be encouraged to continue daily sleep diary monitoring throughout treatment; review of these data enables physician to evaluate insomnia severity, to monitor treatment compliance and progress, and to involve patient in the treatment. Periodic follow-up visits should be scheduled to address compliance issues and to monitor treatment progress. The success of psychological and behavioral approaches depends largely on the patients' willingness to comply with the recommended self-management procedures.

The reader is highly recommended to review two additional sources:

(1) Buscemi N, Vandemeer B, Friesen C, Bialy L, Tubman M, Ospina M, Klassen TP, Witmans M. Manifestations and Management of Chronic Insomnia in Adults. Summary, Evidence Report/Technology Assessment No. 125. (Prepared by the University of Alberta Evidence-based Practice Center, under Contract No. C40000021.) AHRQ Publication No. 05-E021-1. Rockville, MD: Agency for Healthcare Research and Quality. June 2005. For summary of this publication, please visit: <http://www.ahrq.gov/clinic/epcsums/insomrsum.htm> For full report; a very comprehensive 313 pages of literature review (also including pharmacological management of insomnia): <http://www.ahrq.gov/downloads/pub/evidence/pdf/insomnia/insomnia.pdf>

(2) National Institute of Mental Health (NIMH) convened a conference in 2005 (previous insomnia conference was in 1984) to assess current knowledge of insomnia and its treatments. Final statement of this conference is available on the internet: NIH State-of-the-Science Conference Statement on Manifestations and Management of Chronic Insomnia in Adults. National Institutes of Health State-of-the-Science Conference. <http://consensus.nih.gov/2005/2005InsomniaSOS026html.htm>

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