Diagnosis and Treatment of Insomnia

Michael H. Bornet, PhD, and Donna L. Arand, PhD

Insomnia is commonly defined as the subjective report of difficulty in initially falling asleep, difficulty in maintaining sleep (long or multiple awakenings during the sleep period), or awakening too early with the inability to go back to sleep. Insomnia may be either an acute or a chronic problem. Acute insomnia, usually defined as poor sleep associated with a specific life event, such as an important examination, resolves when the event passes or within a period of 3 weeks after the event. Chronic insomnia may begin after an acute episode if conditioning factors are involved, and typically presents as a long-standing complaint despite varied attempts at treatment.

Insomnia is an extremely common complaint. In large surveys, about 36% of respondents report at least occasional difficulty with sleep.27-34 Nine percent to 17% of the general population report chronic or severe insomnia.21,27,34 Incidence varies across populations, however. Kuchler et al35 found that older women with multiple health problems and anxiety or depression were most likely to report more serious insomnia. Similarly, incidence is higher in patients with psychiatric disorders35 and chronic medical conditions such as pulmonary disease or arthritis.

There has been some debate concerning the consequences associated with chronic insomnia and whether the consequences outweigh

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From the Dayton Department of Veterans Affairs Sleep Laboratory, and the Wright State University School of Medicine, Dayton (MIH); and the Sleep Disorders Center, Kettering Memorial Hospital, Kettering, Ohio (BLA)
the costs of treatment. Part of the problem in defining the consequences of insomnia is that insomnia is a common symptom of many other conditions, and outcomes may then be confounded or confused based on the nature of the underlying disease. Despite such qualification, it has been known for some time that there is an increased risk of mortality associated with short sleep lengths. In the Alameda County study, the age-adjusted mortality rate for men who typically slept less than 6 hours per night and often had trouble sleeping was 2.5 times the rate for 7- to 8-hour sleepers with no sleep problem. Several studies have now reported that patients with insomnia are likely to have a current psychiatric disorder (40% versus 16% of controls) and that insomnia was also the single best predictor of the future development of depression, even when patients had no other symptoms of depression at initial interview. Insomnia tends to be chronic, particularly in older individuals, and patients with insomnia are at increased risk for developing anxiety, alcohol and drug use disorders, and nicotine dependence.

In older patients, insomnia remains related to depression but is also associated with poor health and decreased activity. Onset of insomnia in older patients is related to decreased survival. Men who died from coronary heart disease were more likely to have had reported sleep lengths of less than 4 hours, although similar findings were reported for other diseases such as cancer. Fatigue was a predictor of myocardial infarction (MI), even after adjustment for the effects of blood pressure, blood sugar, antihypertensive medications, smoking, and age. In patients with documented MI, 39% reported preceding insomnia. Elderly individuals who reported insomnia also reported both angina pectoris and cardiac arrhythmia more than twice as often as individuals without insomnia. In addition, insomnia patients used more cardiovascular medications and were more likely to have cardiac insufficiency.

The most commonly reported causes of disturbed sleep by older patients, however, were worries and nocturia.

Other studies showing increased heart rate and sympathetic nervous system activity in patients with insomnia leave open the possibility that chronic insomnia might increase the risk of development of coronary artery disease, especially because links have also been shown between depression and the development of coronary artery disease. Better knowledge of the relationship between insomnia and the development of other medical conditions depends on the correct differential diagnosis of insomnia followed by long-term treatment or follow-up studies. The following sections are designed to aid in the differential diagnosis of insomnia.

**DIFFERENTIAL DIAGNOSIS OF INSOMNIA**

Insomnia can be an acute or a chronic problem. Acute insomnia is typically linked to specific stressful events, such as loss of a loved one or time-zone shifts, and is defined as lasting for 3 weeks or less. As acute insomnia is directly linked to a known, time-limited situation, short-term treatment with benzodiazepine medication or zolpidem to improve sleep may be warranted. These medications are highly effective in improving sleep. Medications such as triazolam or zolpidem, with short half-lives, are typically prescribed for patients who require maximal daytime alertness, whereas longer-acting medications such as quasupram or estazolam may be used for patients experiencing poor sleep and daytime anxiety. These medications, however, should be prescribed with some care in patients with pulmonary disease or possible underlying sleep apaera.

Patients with chronic insomnia frequently present more difficult diagnosis and treatment problems. The primary factors to be considered in the differential diagnosis of insomnia are as follows:

- **Acute insomnia**
- **Situational stress or emotional factors**
- **Biologic rhythm disorders**
- **Chronic insomnia**
  - **Sleep apnea**
  - **Periodic limb movements, restless legs**
  - **Circadian rhythm disorders**
  - **Use or withdrawal from drugs or medications**
  - **Psychiatric disorders**
  - **Behavioral disorders (inappropriate conditioning)**
  - **Secondary to medical disorders**
  - **Primary insomnia**
    - **Natural short sleep**
    - **True primary insomnia**
    - **Sleep state misperception**

Each of these components is addressed separately in this article.

An analysis of patients referred to sleep disorder centers revealed that about 25% of the referred patients complained of insomnia. Of the insomnia patients, 30% were identified as having insomnia related to psychiatric disorders; 15% were related to primary insomnia; 32% were insomnias related to the use of drugs and alcohol; and 12% were related to periodic limb movements and restless legs. These numbers should be examined with some skepticism because patients who actually are referred to sleep centers form a specialized subgroup of all patients with insomnia. These patient groups are almost certainly biased toward patients with sleep-associated respiratory disorders and periodic limb movements, whereas patients with insomnia primarily associated with depression, for example, are unlikely to be referred to a sleep center and, even if referred, are unlikely to be given a definitive all-night sleep test (often because such tests are not reimbursed unless there is clear evidence of sleep apnea).
Sleep Apnea

Many patients are referred to sleep centers secondary to reported snoring or sleep apnea. A majority of these patients, however, report inability to remain awake during the day more than insomnia at night. In sleep apnea, patients frequently arouse to resume respiration. When these arousals increase in frequency, they reduce the restorative power of sleep, and patients report increasing daytime sleepiness as a primary symptom. About 6% of patients referred to sleep centers with an insomnia complaint, however, are found to have sleep apnea. These patients may report poor sleep, frequent awakenings, or awakening with a sensation of gagging or choking on occasion, and typically have milder apnea than patients reporting daytime sleepiness as a major problem. Patients with sleep apnea at all levels of severity can be identified by the presence of snoring, usually with brief periods of silence corresponding to airflow blockage (see the article by Hudgel and Auckley on sleep apnea in this issue).

Periodic Limb Movements

Stereotypical repetitive limb movements that last from 1 to 5 seconds and occur every 20 to 30 seconds are extremely common during sleep. A proportion of patients with periodic limb movements also report restless legs—discomfort or creeping sensations in the calves that are relieved by movement but that return about 30 seconds after movement cessation. The restless sensations increase in the evening and may interfere with sleep onset. Studies suggest that 12% of patients with insomnia referred to sleep disorder centers have periodic limb movements or restless legs. Population studies suggest that symptomatic periodic limb movements are rare in young individuals but that the incidence is 29% in individuals between 50 and 65 years of age and 44% in individuals over 65 years of age. Patients may be asymptomatic if the limb movements do not actually disturb sleep, but as the number or duration of the movements increase, patients may report difficulty maintaining sleep in addition to restlessness and a tendency to pull out their bed covers each night. Spouses can frequently identify limb movements in affected patients. Treatments for restless legs and periodic limb movements may include the prescription of carbidopa-levodopa, clonazepam, or gabapentin at bedtime.

Circadian Rhythm Disorders

 Humans experience 24-hour rhythms in most physiologic functions, including alertness, body temperature, and heart rate. All of these functions begin to decrease near normal bedtime, reach a low point near the end of the usual sleep period, and then begin to increase near the usual time of awakening. These rhythms are maintained by an internal clock that continues approximately 24-hour rhythms, even when individuals are placed in isolation. Because of their approximate 24-hour periodicity, these rhythms are called circadian. Circadian rhythm disorders occur when there is a mismatch between the underlying biologic rhythm of alertness and the choice of time to sleep. For example, when individuals attempt to go to sleep earlier than expected by their circadian controller, their body is still at a high level of arousal and they have difficulty falling asleep. If this is a consistent clinical problem, it is called delayed sleep phase syndrome. On the other hand, if individuals remain awake after their circadian controller has already decreased physiologic function in anticipation of sleep onset, they will become increasingly sleepy, but after they finally do go to bed and fall asleep, they may find that they awaken sooner than they would like. If this is a persistent problem it is called advanced sleep phase syndrome.

Young adults usually have a circadian rhythm that is slightly longer than 24 hours, and this may predispose them to stay up later each night if allowed. Unfortunately, staying up later on Friday and Saturday evening, for example, allows the circadian rhythm to shift so that an attempt to go to bed at a more normal time on Sunday night (an example of delayed sleep phase syndrome) produces difficulty in falling asleep.

Many individuals, including shift workers, retired persons, chronically ill individuals, and students, may have irregular sleep patterns. For example, when individuals retire from work, they may no longer have a standard time to rise each morning and their levels of activity during the day may decrease. They may also start to nap during the day. It has been shown that elderly individuals have a flattened 24-hour temperature curve, that is, their body temperature is lower during the day and higher during the night than that seen in young adults. Such changes in physiologic function may be secondary to decreased activity and sleep during the day and more irregular sleep at night. Treatment for an irregular sleep-wake pattern involves establishing standard bedtimes and wake times, eliminating naps during the period of wakefulness, and increasing levels of activity and bright light exposure during the waking period.

Circadian rhythms are normally maintained by activity, exposure to bright light, and social cues. Therefore rhythm shifting is facilitated by increasing activity, bright light exposure, and social interaction during the new waking period being established. Melatonin is a hormone that helps synchronize circadian rhythms and has been used to shift sleep to a new time after third shift of work or time zone changes. Melatonin is less useful, however, in patients who usually go to bed at
the same time, because they already secrete melatonin at their usual bedtime. Melatonin is also less useful in rotating shift workers, because the time melatonin has helped shift rhythms to a new sleep period, the shift worker has already switched to a different shift time.

Drugs and Medications

Drugs and medications frequently produce acute or chronic insomnia based on dose, time of administration, tolerance level, and age. It is well known that caffeine or other stimulants can produce insomnia. In addition, similar caffeine consumption may only start to produce insomnia in some individuals as they age because decreases in metabolic rate with aging increase the effective half-life of caffeine. Ethanol, which is known more as a CNS depressant, may also produce difficulty in maintaining sleep both because it increases snoring and sleep apnea and because REM sleep and therefore produces increased wakefulness when REM sleep normally occurs. Even the barbiturate sedatives such as secobarbital, which improve sleep during short-term use, produce rapid tolerance that is perceived as a return of insomnia. For this reason, barbiturates are seldom used to treat insomnia.

Insomnia holds a prominent position in the side-effect list of many medications. Classes of medications often associated with insomnia include stimulants, anorectics, β-antagonists, and, in some cases, sedatives or alcohol. The role of stimulants such as caffeine in the production of insomnia is clear. When taken in the evening, however, respiratory stimulants such as theophylline can also cause insomnia. Most appetite-suppressing medications also have central stimulant properties and therefore can produce insomnia. β-Blockers such as propranolol, metoprolol, and pindolol may produce sleep-onset insomnia or increased awakenings and dreams.

Another large list of medications produce drowsiness and may be used in the treatment of situational insomnia. Currently, the most commonly prescribed hypnotics include the benzodiazepines and zolpidem. Other common treatments with less empirical support include sedating antidepressants, such as amitriptyline, and antihistamines. Melatonin, although commonly used over the counter for insomnia, probably has a main effect of shifting circadian rhythms to produce sleep at unusual times rather than improving sleep at the normal time. L-tryptophan marketed as a health food substance until removed from the market by the Food and Drug Administration, probably has limited hypnotic efficacy. Because of manufacturing problems, however, L-tryptophan should only be ingested as a natural component of food (i.e., a glass of milk or a turkey sandwich).

Psychiatric Disorders

In a survey of clinic patients reporting severe insomnia, 83% had abnormal results on the Minnesota Multiphasic Personality Inventory (MMPI). The clinically elevated scales were depression, hysteria, and hypochondriasis; this means that many depressed individuals express their depression as a physical illness, and that those patients focusing on poor sleep are referred to the sleep center. In general, 57% of individuals reporting insomnia have a psychiatric disorder or will develop one within a year. These patients have poor sleep, and their poor sleep tends to be related to the degree of stress or depression during the day. In many sleep disorder centers, all patients are screened with a personality inventory, primarily because the incidence of depression is so high. When insomnia is a symptom associated with depression, the appropriate initial referral is to psychiatry. As the depression improves, sleep also recovers.

Inappropriate Conditioning

Some patients who have normal psychological status during the day find that they become tense only when they try to fall asleep. Difficulty falling asleep is disturbing, and these patients worry about whether they will be able to fall asleep and the consequences of not falling asleep. Such thoughts may produce a vicious cycle in which increasing arousal limits the ability to fall asleep, resulting in increasing arousal. Eventually just going to bed can initiate the worry-arousal cycle. Other patients may begin to plan their next day or think about problems when they go to bed. Eventually, the bedroom sleep setting comes to be seen as the place to plan or worry rather than the place to sleep. These examples of psychophysiologic insomnia typically are treated behaviorally.

Other Medical Disorders

Many medical disorders produce discomfort, pain, stress, or depression. Any of these can limit the ability to fall asleep or maintain sleep. A few common medical conditions that predispose patients to insomnia are briefly reviewed.

Pulmonary disease patients complain of poor sleep, and several studies have objectively verified the poor sleep. It is known that arterial oxygen saturation falls and that arterial carbon dioxide increases during sleep in patients with chronic obstructive pulmonary disease (COPD). These changes are greatest during REM sleep. When a patient with COPD lies down, the work of breathing is increased by the need to
move the chest in direct opposition to gravity. Any secretions tend to pool in the airways, causing coughing. Either of these effects predispose COPD patients to increased awakenings during the night as they attempt to clear their airway or maintain sufficient airflow. Patients may benefit from sleeping on their side or with their head and chest elevated enough to allow movement of secretions and to decrease some of the direct force of gravity on their chest. Appropriate treatment of COPD will improve sleep unless the medications used also produce poor sleep.

Asthma patients have also been documented to have increased wakefulness during the night. Patients with asthma have significant nocturnal bronchoconstriction that results in coughing or wheezing. When patients are treated with short-acting bronchodilators, the medication may lose effectiveness in the middle of the night, resulting in awakenings due to the return of asthma symptoms. Treatment with longer-acting agents may help to resolve this problem.

Pain is a frequent cause of poor sleep. Pain may be secondary to a number of factors, including inflammation, as in rheumatoid arthritis; headache; fibrositis; or after surgery or various injuries. Reduction in pain by treatment of the underlying medical condition usually results in improved sleep. For example, rheumatoid arthritis is characterized by chronic joint inflammation, which results in chronic pain and stiffness. Sleep disturbance and early morning stiffness are typically reported. When treated with nonsteroidal anti-inflammatory agents to reduce inflammation, patients frequently report decreased pain and some improvement in sleep. Other common treatments for pain may include low doses of amitriptyline, other antidepressants, or the judicious use of benzodiazepines.

Primary Insomnia

In about 15% of insomnia patients referred to sleep disorder centers, all of the preceding factors can be ruled out as a cause of the reported poor sleep. These patients may be divided into short sleepers (no sleep pathology), patients with true primary insomnia, and patients who sleep well despite their report of insomnia (sleep state misperception).

Short Sleepers

One group of patients who may report difficulty falling asleep or a problem with awakening too early in the morning, without any daytime consequences, may simply have a short sleep requirement. Such patients may report poor sleep because they actually spend too much time in bed for their sleep requirement and therefore spend excessive time awake in bed. Daily sleep requirement changes rapidly in childhood, but some reduction in daily sleep requirement may continue in later years, such that some older individuals may require 6 or fewer hours of sleep per 24 hours. In addition, aging is accompanied by lighter sleep, increased awakenings, and a tendency to awaken early in the morning. If these changes have occurred without the development of daytime consequences, they may be a function of normal aging. In such patients, spending less time in bed, along with some reassurance that sleep requirements differ from individual to individual, usually resolves the problem.

True Primary Insomnia

On the other hand, patients with true primary insomnia complain of poor sleep as well as other symptoms, including subjective fatigue and increased stress, anxiety, or depression. Sleep laboratory evaluations of these patients have also documented long latencies or inability to fall asleep on the Multiple Sleep Latency Test (MSLT), as well as psychopathology as measured by the MMPI, increased physiologic activation as indexed by measures such as increased body temperature, whole-body metabolic rate, or heart rate, and consistent overestimation of sleep latency and time spent awake during the night. Because primary insomnia patients typically display both mood alteration and evidence of physiologic arousal, identification of underlying causal factors has been difficult. One study, however, examined the effects of increasing physiologic arousal in the production of insomnia through the administration of caffeine 400 mg three times a day for 1 week in normal sleepers. This chronic use of caffeine increased arousal level, as measured by whole-body metabolic rate, and sleep efficiency declined significantly. During the initial days of caffeine use, subjects had elevated MSLT scores (i.e., took longer to fall asleep) and reported increased subjective vigor. By the end of the week of caffeine use, however, significantly increased daytime fatigue was reported despite MSLT scores that still remained significantly elevated as compared with baseline. In addition, anxiety, as measured by the anxiety scale of the MMPI, moved significantly toward psychopathology. The finding from this study that chronically elevated levels of physiologic arousal would paradoxically lead to reports of increased fatigue even while patients were less sleepy on the MSLT was a strong indication that physiologic activation by itself could be responsible for the same paradoxical reports in patients with insomnia. The increase in the anxiety scale of the MMPI, a nontransparent trait measure of personality, offered evidence that cognitive and personality components frequently seen in patients with insomnia could be influenced significantly by a chronically increased level of CNS arousal.
Two recent studies have examined this issue by questioning the extent to which insomnia patients' complaints of poor sleep and daytime dysphoria are related to their sleep parameters. In a yoked-control experiment, normal sleepers were given the same sleep pattern as a matched insomnia; a technician awakened the normal sleepers when the insomniacs (aroused or awake) for a week to determine if the poor sleep pattern per se would produce the daytime symptoms that insomniacs typically experience. It was hypothesized that if poor sleep by itself was responsible for the secondary symptoms reported by patients with insomnia, then normal sleepers given a similar sleep pattern would also begin to display the secondary symptoms. The study results, however, indicated that normal sleepers given such poor sleep suffered from mild sleep deprivation but did not develop the changes in mood, personality, or physiologic activation typically seen in insomnia patients.

In a similar study, it was hypothesized that if nocturnal sleep parameters produced the daytime dysphoria reported by patients with insomnia then sleep maintenance insomnia patients who were kept awake even longer than usual during the night should have increased dysphoria during the following day. To test this hypothesis, patients with sleep maintenance insomnia were allowed only 80% of their already reduced total sleep each night for seven consecutive nights. This sleep reduction was accomplished by waking patients up at the end of each quarter of the night if they accumulated more than 80% of their baseline sleep for that quarter of the night (while holding time in bed for the entire night at the baseline level). This paradigm produced very poor sleep (total sleep of 4.2 hours on each night for the week). The reduction of total sleep time by experimental awakenings resulted in a significant decrease in daytime MSLT values. After seven nights of 4.2 hours of sleep, MSLT values had decreased from 15.6 to 11.1 minutes, still within the normal range for the test despite apparent significant sleep loss. The results indicated that when the sleep of insomniacs was experimentally reduced, they displayed increased sleepiness during the day in agreement with the expectancy in normal sleepers but not in typical insomnia patients. Despite the large reduction in total sleep, the insomniacs did not become pathologically sleepy on the MSLT, and this probably indicated the degree to which their hyperarousal was successful in masking their sleep tendency. Of equal interest, patients did not report significant decreases in their sleep quality or show changes in their personality or physiologic parameters consistent with more severe insomnia when their wake time during the night was increased by 2 hours. One conclusion from such data is that the reports of poor sleep quality and daytime dysphoria from insomniacs are not directly related to EEG sleep at all but rather to level of arousal. In the real world, degree of hyperarousal would vary from night to night. On nights with less hyperarousal, insomniacs would have improved EEG sleep and would also report a good night of sleep, but the change in arousal level could be the cause of both events. For example, Chambers and Kim reported a significant negative correlation between state anxiety at bedtime and reports of feeling rested on the next day in insomniacs, but neither anxiety nor reports of feeling rested were significantly correlated with sleep values.

The literature indicates that there is a significant positive correlation between inability to fall asleep at night and inability to fall asleep on the MSLT in insomniacs. Such a relationship makes sense if CNS arousal level masks sleepiness (i.e., the greater the CNS arousal level, the less likely sleep should occur at any time). If the daytime symptoms of insomnia were secondary to the nocturnal EEG values, the worsening of EEG sleep parameters in the laboratory should have made the secondary symptoms worse. That did not happen. On the other hand, if hyperarousal caused both poor sleep and the secondary symptoms, the EEG laboratory poor sleep would have had no effect on the secondary symptoms that were related to hyperarousal. These studies indicate that the daytime dysphoria of a patient with primary insomnia is not due to simply having worse sleep at night. The daytime symptoms are produced by hyperarousal.

Sleep State Misperception

Another group of patients who would be classified as having primary insomnia based on their history actually have normal sleep in the laboratory. Despite the normal sleep pattern, the patients continue to report that they have difficulty falling asleep or remaining asleep. These patients are currently given the diagnosis of Sleep State Misperception. Insomnia. A recent study, however, has shown that these patients, even while having a normal night of sleep in terms of EEG parameters, did have elevated metabolic rate throughout the night and day as compared with matched controls. It is likely that these patients actually suffer from a disorder of hyperarousal and that the hyperarousal is responsible for the insomnia symptoms that they report.

Summary

Insomnia is an extremely common complaint. It frequently occurs secondary to a number of medical and psychiatric conditions that directly affect the ability to initiate or maintain sleep. Insomnia may also occur secondary to behavioral factors, such as shift work, in which patients do not follow or control the dictates of their internal circadian rhythm.
or develop inappropriate conditioned responses to their sleep surround. Finally, insomnia may be a primary and sometimes lifelong complaint. Patients with primary insomnia probably have a real physiologic problem that has not been clearly identified as the basis for their subjective complaint.

**TREATMENT**

**Secondary Insomnia**

Treatment options for patients with secondary insomnia must be directed toward alleviating the underlying cause of the insomnia. In practice, of course, treatments for some of the underlying disorders are less than perfect and poor sleep may remain. For example, chronic pain causes insomnia and is often difficult to control. In some cases, individual decisions need to be made concerning the consequences of continued pain and poor sleep as compared with the consequences of protracted treatment with benzodiazepine medications to help maintain sleep.

**Primary Insomnia**

Most guidelines for the treatment of insomnia specify that hypnotic medications are indicated only for short-term use in patients with situational insomnia. A number of behavioral treatment approaches are available for use in the treatment of primary insomnia or insomnia secondary to circadian rhythm dysynchrony or inappropriate conditioning. In general, all of these patients can benefit from improved sleep hygiene: that is, having regular times of going to bed and arising; sleeping in a consistently quiet, dark room at a comfortable temperature, avoidance of caffeine and alcohol, and development of a consistent period of sleep preparation. In addition, several behavioral treatments are available to help patients relax and avoid inappropriate conditioning.

Several forms of relaxation therapy can be of benefit for patients who complain of muscle tension when trying to fall asleep. For example, in progressive muscle relaxation therapy, patients are taught to tense and relax each muscle group in a systematic fashion. Sometimes biofeedback of muscle activity is used to inform patients of their success in muscle relaxation.

Patients who primarily suffer from racing thoughts or who worry can sometimes be treated with cognitive behavioral therapies. Patients may be taught meditation techniques or given guided imagery or thought-stopping techniques. In addition to providing techniques to refocus thoughts, patients are typically taught stimulus control. Stimulus control therapy holds that when people worry in bed, the bed itself eventually becomes a conditioned stimulus that produces worrying. In stimulus control, patients are told that if they are unable to fall asleep in a reasonable period of time or if they notice that they have begun to worry while lying in bed, then they should get out of bed and go into another room to relax and resolve the issue that bothers them. They are then instructed to return to bed when they begin to feel relaxed and sleepy again so that they can begin to associate their bed with relaxation and sleepiness rather than tension and worry.

If it is suspected that the patient is a short sleeper or spends too much time in bed (resulting in long periods of wakefulness), sleep restriction therapy may be appropriate. In sleep restriction therapy, the amount of time that the patient is allowed to be in bed each 24 hours is systematically reduced and then adjusted over time so that the patient spends less time in bed and sleeps for a much higher proportion of the time actually spent in bed.

Behavioral therapies may be quite demanding in terms of time and effort, both on the part of patients and clinicians. Studies suggest that behavioral therapies do improve sleep and help patients deal with their insomnia problem. A majority of patients, however, continue to have at least occasional problems with their sleep.

Treatment with medication may be appropriate in another group of patients. As indicated, many patients with chronic insomnia have stressful lives, low levels of chronic pain, or subclinical depression as a factor in their insomnia. Some of these patients may be treated effectively with low doses of tricyclic antidepressants at bedtime. Other patients use hypnotics on an as-needed basis in response to particularly stressful days. Unfortunately, outcome studies that provide specific risk and benefit ratios for patients with primary insomnia treated with long-term behavioral therapy versus no therapy versus pharmacologic therapy have not been done. This means that a realistic estimate of long-term benefits or risks of these therapies is not available.

**CONCLUSIONS**

Insomnia is an extremely common problem that is most frequently related to specific acute situational factors. Chronic insomnia is frequently a symptom of another sleep disorder, such as sleep apnea; a symptom of an underlying medical problem such as chronic pain; or a symptom of a psychiatric disorder, such as depression. In these cases, effective treatment first requires a correct differential diagnosis and then attention to the underlying problem.
Clinical and Laboratory Evaluations of Excessive Daytime Sleepiness

Philip R. Westbrook, MD

The state of unwanted sleepiness, the tendency to doze off when sleep is neither desired nor safe, is both very common and potentially disastrous. It is also a symptom, not unlike snoring, that is all too often treated as an object of derision and humor both by the public and the medical profession. Sleepiness is one of the most frequent complaints recorded by sleep medicine practitioners, and it is almost surely under-recorded by most primary care physicians. This article on the evaluation of the patient who complains of (or is suspected of having) excessive sleepiness is intended as an introduction to the topic for those who are relatively new to the field of sleep disorders medicine.

WHAT IS EXCESSIVE SLEEPINESS?

We need to begin by trying to define excessive sleepiness, also known as hypersomnia or excessive daytime sleepiness (EDS). This turns out to be not as easy as one would like. The International Classification of Sleep Disorders defines sleepiness as "difficulty in maintaining alert wakefulness so that the person falls asleep if not actively kept aroused. This is not simply a feeling of physical tiredness or listlessness. When sleepiness occurs in inappropriate circumstances, it is considered excessive sleepiness." Another definition of sleepiness is that it represents our attempt to resist turning the CNS over to sleep. Hypersomnia is inappropriate sleepiness, that is, the demonstrable propensity to doze off or fall asleep quickly in situations that are sedentary but in which sleep is not expected or desired. No one falls asleep while being

From Pacific Sleep Medicine Services, Inc., Redlands, California