

polysomnogram helps rule out other possible causes of sleepiness such as sleep apnea, and the MSLT provides essential, objective evidence of sleepiness plus REM sleep dysregulation. Across the five naps of the MSLT, most patients with narcolepsy will fall asleep in less than 8 min on average, and they will have episodes of REM sleep in at least two of the naps. Abnormal regulation of REM sleep is also manifested by the appearance of REM sleep within 15 min of sleep onset at night, which is rare in healthy individuals sleeping at their habitual bedtime. Stimulants should be stopped 1 week before the MSLT and antidepressants should be stopped 3 weeks prior, because these medications can affect the MSLT. In addition, patients should be encouraged to obtain a fully adequate amount of sleep each night for the week prior to the test to eliminate any effects of insufficient sleep.

## TREATMENT NARCOLEPSY

The treatment of narcolepsy is symptomatic. Most patients with narcolepsy feel more alert after sleep, and they should be encouraged to get adequate sleep each night and to take a 15- to 20-min nap in the afternoon. This nap may be sufficient for some patients with mild narcolepsy, but most also require treatment with wake-promoting medications. Modafinil is used quite often because it has fewer side effects than amphetamines and a relatively long half-life; for most patients, 200–400 mg each morning is very effective. Methylphenidate (10–20 mg bid) or dextroamphetamine (10 mg bid) are often effective, but sympathomimetic side effects, anxiety, and the potential for abuse can be concerns. These medications are available in slow-release formulations, extending their duration of action and allowing easier dosing. Sodium oxybate (gamma hydroxybutyrate) is given twice each night and is often very valuable in improving alertness, but it can produce excessive sedation, nausea, and confusion.

Cataplexy is usually much improved with antidepressants that increase noradrenergic or serotonergic tone because these medications strongly suppress REM sleep and cataplexy. Venlafaxine (37.5–150 mg each morning) and fluoxetine (10–40 mg each morning) are often quite effective. The tricyclic antidepressants, such as protriptyline (10–40 mg/d) or clomipramine (25–50 mg/d) are potent suppressors of cataplexy, but their anticholinergic effects, including sedation and dry mouth, make them less attractive.<sup>1</sup> Sodium oxybate, given at bedtime and 3–4 h later, is also very helpful in reducing cataplexy.

## EVALUATION OF INSOMNIA

Insomnia is the complaint of poor sleep and usually presents as difficulty initiating or maintaining sleep. People with insomnia are dissatisfied with their sleep and feel that it impairs their ability to function well in work, school, and social situations. Affected individuals often experience fatigue, decreased mood, irritability, malaise, and cognitive impairment.

Chronic insomnia, lasting more than 3 months, occurs in about 10% of adults and is more common in women, older adults, people of lower socioeconomic status, and individuals with medical, psychiatric, and substance abuse disorders. Acute or short-term insomnia affects over 30% of adults and is often precipitated by stressful life events such as a major illness or loss, change of occupation, medications, and substance abuse. If the acute insomnia triggers maladaptive behaviors such as increased nocturnal light exposure, frequently checking the clock, or attempting to sleep more by napping, it can lead to chronic insomnia.

Most insomnia begins in adulthood, but many patients may be predisposed and report easily disturbed sleep predating the insomnia, suggesting that their sleep is lighter than usual. Clinical studies and animal models indicate that insomnia is associated with activation

during sleep of brain areas normally active only during wakefulness. The polysomnogram is rarely used in the evaluation of insomnia, as it typically confirms the patient's subjective report of long latency to sleep and numerous awakenings but usually adds little new information. Many patients with insomnia have increased fast (beta) activity in the EEG during sleep; this fast activity is normally present only during wakefulness, which may explain why some patients report feeling awake for much of the night. The MSLT is rarely used in the evaluation of insomnia because, despite their feelings of low energy, most people with insomnia do not easily fall asleep during the day, and on the MSLT, their average sleep latencies are usually longer than normal.

Many factors can contribute to insomnia, and obtaining a careful history is essential so one can select therapies targeting the underlying factors. The assessment should focus on identifying predisposing, precipitating, and perpetuating factors.

**Psychophysiologic Factors** Many patients with insomnia have negative expectations and conditioned arousal that interfere with sleep. These individuals may worry about their insomnia during the day and have increasing anxiety as bedtime approaches if they anticipate a poor night of sleep. While attempting to sleep, they may frequently check the clock, which only heightens anxiety and frustration. They may find it easier to sleep in a new environment rather than their bedroom, as it lacks the negative associations.

**Inadequate Sleep Hygiene** Patients with insomnia sometimes develop counterproductive behaviors that contribute to their insomnia. These can include daytime napping that reduces sleep drive at night; an irregular sleep-wake schedule that disrupts their circadian rhythms; use of wake-promoting substances (e.g., caffeine, tobacco) too close to bedtime; engaging in alerting or stressful activities close to bedtime (e.g., arguing with a partner, work-related emailing and texting while in bed, sleeping with a smartphone or tablet at the bedside); and routinely using the bedroom for activities other than sleep or sex (e.g., TV, work), so the bedroom becomes associated with arousing or stressful feelings.

**Psychiatric Conditions** About 80% of patients with psychiatric disorders have sleep complaints, and about half of all chronic insomnia occurs in association with a psychiatric disorder. Depression is classically associated with early morning awakening, but it can also interfere with the onset and maintenance of sleep. Mania and hypomania can disrupt sleep and often are associated with substantial reductions in the total amount of sleep. Anxiety disorders can lead to racing thoughts and rumination that interfere with sleep and can be very problematic if the patient's mind becomes active midway through the night. Panic attacks can occur during sleep and need to be distinguished from other parasomnias. Insomnia is common in schizophrenia and other psychoses, often resulting in fragmented sleep, less deep NREM sleep, and sometimes reversal of the day-night sleep pattern.

**Medications and Drugs of Abuse** A wide variety of psychoactive drugs can interfere with sleep. Caffeine, which has a half-life of 6–9 h, can disrupt sleep for up to 8–14 h, depending on the dose, variations in metabolism, and an individual's caffeine sensitivity. Insomnia can also result from use of prescription medications too close to bedtime (e.g., theophylline, stimulants, antidepressants, glucocorticoids). Conversely, withdrawal of sedating medications such as alcohol, narcotics, or benzodiazepines can cause insomnia. Alcohol taken just before bed can shorten sleep latency, but it often produces rebound insomnia 2–3 h later as it wears off. This same problem with sleep maintenance can occur with short-acting benzodiazepines such as alprazolam.

**Medical Conditions** A large number of medical conditions disrupt sleep. Pain from rheumatologic disorders or a painful neuropathy commonly disrupts sleep. Some patients may sleep poorly because of respiratory conditions such as asthma, chronic obstructive pulmonary disease, cystic fibrosis, congestive heart failure, or restrictive lung disease, and some of these disorders are worse at night in bed due to circadian variations in airway resistance and postural changes that can result in

<sup>1</sup>No antidepressant has been approved by the U.S. Food and Drug Administration (FDA) for treating narcolepsy.