ABSTRACT

Sleep is an active and highly regulated process, characterized by cycles of rapid eye movement (REM) and non-REM sleep. The number of cycles, their duration, and the proportions of REM and non-REM sleep vary throughout our lifetimes, from infancy to old age. Insomnia (i.e., disturbed sleep despite adequate opportunity and circumstances) is pervasive in American culture, yet is often underdiagnosed and undertreated—in part, because it is under-recognized by the patients, despite symptoms of fatigue and/or drowsiness. Because sleep is a state of altered consciousness, it is difficult to pinpoint problems with sleep, let alone their possible causes. This article discusses the physiology of sleep—the hormonal and neural controls over the sleep-wake cycle and the source of the circadian rhythm—in addition to methods for diagnosing and assessing sleep disorders. Many user-friendly tools are readily available. Because patients often do not recognize their own sleep problems, or if they do, fail to address them with their healthcare practitioner, a review of sleep hygiene should be part of every office visit.

Stage 1, we no longer perceive outside stimuli (eg, noise or light); for example, a person in Stage 1 sleep will not respond if his or her name is whispered. However, it is still considered a period of light sleep. People who are awakened during Stage 2 sleep are more likely to know they are asleep than during later stages. Stages 3 and 4 are called slow-wave (or δ wave) sleep. During these stages of deep sleep, it is difficult to wake the person, whose heart rate and breathing are regular and slow and whose muscles are completely relaxed. Children enter stages 3 and 4 effortlessly, but as we age, we lose that ability to easily reach those stages.

Stages 1 through 4 of non-REM sleep are followed by REM sleep, in which all voluntary muscles become completely paralyzed while brain activity increases greatly. It is during REM sleep that we are dreaming. People who are blind from birth experience REM sleep, but their dreams do not contain visual images.

Each of these cycles (Stages 1 through 4, followed by REM) takes approximately 90 minutes, and we go through 4 to 6 cycles each night, with the proportion of REM sleep increasing with each cycle. During the latter portion of the sleep period, the body temperature is at its lowest (a full 2°F below the peak daytime temperature). Just before awakening, cortisol levels increase to mobilize energy stores and prepare the body for consciousness.

However, before sleep onset is the period of drowsiness, seen on the EEG as α waves. Drowsiness is a calm wakefulness, in which we are relaxing and letting go of the day as our thoughts slow down. During this time, the body temperature declines and melatonin levels rise. Importantly, drowsiness is the last stage before falling asleep, not the first. Once drowsiness sets in, sleep can come instantly. This has important implications for people who experience drowsiness while driving.

How Much Sleep Do We Need?

Most adults need approximately 8 hours of sleep each night, but that can vary from person to person, ranging from 6 to 10 hours. The amount of sleep a person needs is the amount that will keep him or her awake and functioning all day, but allow the person to fall asleep easily at bedtime. The amount of sleep we need also varies throughout our lifetime. Newborns sleep up to 16 to 18 hours per day and spend 60% to 80% of their sleep in REM sleep. Adults require approximately 8 hours of sleep, of which approximately 25% is spent in REM sleep. Older adults often sleep fewer than 8 hours, and only 15% to 20% of their sleep is REM sleep. Elderly people also have very little stage 4 sleep, and their sleep becomes less consolidated (ie, they sleep in segments of a few hours at a time, including daytime naps, rather than 1 long period during a 24-hour day). Teenagers require longer periods of sleep than adults, and understanding teenaged sleep needs has had important implications on education. Some schools in the United States have changed their start times to reflect the differences in teen sleep needs and cycles.

One of our biggest gaps in sleep knowledge is for middle-aged adults, perhaps because they are too busy to participate in sleep studies.

The Physiology of Sleep

The switch that allows us to move from consciousness (awake) to unconsciousness (sleep) is the culmination of opposing forces whose control rises or falls throughout the day, similar to the tide. The 2 forces are referred to as the homeostatic sleep drive and the circadian drive. The “sleep switch” (ie, the all-or-nothing phenomenon in which the sleep-wake circuit is in sleep or wake mode with no intermediate state) is driven by the homeostatic drive during sleep and by the circadian drive during periods of wakefulness. The sleep switch is strongly influenced by the suprachiasmatic nuclei (SCN)—2 small clusters of nerves that sit above the optic nerve. The SCN measure light levels entering the eye and affect hypothalamic control of body temperature, hormone release, and metabolic rate in relation to the sleep-wake cycle.

The homeostatic sleep drive can be thought of as a “sleep bank” in which deposits are made at night and withdrawals made during the day. However, if it were the only drive affecting the sleep-wake cycle, we would become increasingly sleepy as the day progressed. Instead, the homeostatic drive is countered by the circadian drive, which increases during the day and discharges at night. As shown in Figure 1, the circadian drive has a short lull in late afternoon (which some cultures honor with an afternoon nap), and our “second wind” starts in the late afternoon and continues until bedtime. After the circadian drive decreases as bedtime approaches, a low circadi-
an drive helps maintain sleep throughout the night. Conversely, during the day, the sleep drive is at its lowest, and a high circadian drive maintains wakefulness. Thus, sleep is constantly battling wake. One of the most important principles of sleep is that all wakefulness is sleep deprivation. As soon as we wake up, the meter starts ticking, calculating how many hours of sleep will be needed to pay off the sleep debt later that night. The brain keeps an exact account of how much sleep it is owed, and lost sleep must be paid back in full. People are severely affected by a large sleep debt, which can impair driving, work performance, and mood. Less well known is the fact that a large sleep debt also can exacerbate drug side effects and the effect of alcohol.

**THE CIRCADIAN RHYTHM**

In the normal state, the transition between wakefulness and sleep represents a delicate balance between environmental, circadian, and homeostatic influences. Humans, after thousands of years of being exposed to the earth's 24-hour cycle of day and night, have established a sleep-wake cycle that is responsive to intrinsic and external stimuli. Intrinsically, the timing of the human sleep-wake cycle is controlled by the SCN, which also is influenced by homeostatic mechanisms that represent accumulated sleep debt. Externally, the body's intrinsic sleep-wake cycle is affected by light, which acts as a photic cue. It is the transition from night to day that sets the stage for wakefulness as the 24-hour cycle is reinforced.

The actual circadian cycle is slightly longer than 24 hours (by approximately 10 minutes). Other factors beyond light, such as exercise or the hormone melatonin, can influence the circadian rhythm of the SCN. In humans, the pineal hormone melatonin exhibits a circadian rhythm of secretion regulated by the SCN. Circulating levels of melatonin increase in the evening, peak between 3:00 AM and 4:00 AM, and gradually decrease as dawn approaches. The secretion of melatonin is synchronized with the earth's 24-hour light-dark cycle, and melatonin secretion is potently inhibited by light as the result of photic information detected by the retina and transmitted to the SCN. At night melatonin is secreted in response to stimulatory signals originating in the SCN. Because our bodies are built to follow the natural changes in light during the 24-hour day, our modern lifestyle—with shift work, television, computers, and even the light bulb—is structured such that we are constantly challenging our circadian rhythm.

**UNDERSTANDING INSOMNIA**

Insomnia may be defined as disturbed sleep in the presence of adequate opportunity and circumstance for sleep. Sleep disturbance consists of at least 1 of the following 3 features: (1) difficulty in initiating sleep; (2) difficulty in maintaining sleep; or (3) waking up too early. Glovinsky and Spielman developed a model to understand the causes of insomnia, referred to as the 3 Ps: predisposing, precipitating, and perpetuating factors. Predisposing factors to insomnia include those we are born with, such as hypersensitivity to noise, emotional arousal (eg, being easily worried), and cognitive hyperarousal (eg, making lists or thinking about the things we have to do). These factors are part of our personality and, therefore, usu-
ally are not easily modified, although behavioral therapy can help.

Precipitating factors include medical illness (even an illness as simple as a cold), psychiatric illness (eg, depression or anxiety), which can precipitate and present as insomnia, work stress, relationship stress (one of the most frequent causes of insomnia we see in our practice), legal stress, prescription drugs, nonprescription (legal and illegal) drugs, shift work, and excessive alcohol. The latter is one of the most frequently used drugs to self-medicate insomnia, but it is ultimately detrimental. Alcohol eases the ability to fall asleep more quickly, but it disrupts sleep architecture and can cause rebound insomnia the same night that alcohol is consumed. Perpetuating factors turn acute insomnia into chronic insomnia. They include a sedentary lifestyle, watching television in the bedroom, caffeine consumption late in the day, napping, eating late in the day, erratic sleep/wake times, and clock watching/worry about sleep (ie, watching the clock and worrying as the time passes about how much sleep you are missing). Precipitating factors can often be eliminated or reduced with the appropriate treatment strategy.12

DIAGNOSING INSOMNIA

Although sleep disorders appear to be pervasive in American culture, people with sleep problems often go unnoticed in the healthcare system. A Sleep in America: 2005 survey revealed that of the 664 adults who reported difficulty sleeping, 70% (n = 465) had never discussed their problem with a physician. Whereas 24% (n = 159) reported having discussed their problem with a physician, it had been a secondary reason for the consultation. Only 6% (n = 40) reported that they had sought the help of a physician for their sleep difficulty as the primary reason for their consultation.13 Some patients with insomnia may not even realize that their fatigue, low mood, or decreased work performance/attention may be the result of poor sleep. Yet, poor sleep is one of the most common reasons for poor work performance. Because it is pervasive yet hidden, it is up to the healthcare practitioner (HCP) to be proactive in addressing sleep hygiene as part of each office visit. Unfortunately, 70% of respondents to the 2005 National Sleep Foundation survey reported that their physician had never asked them about their sleep.13

There are 2 sets of diagnostic criteria for insomnia: the International Classification of Sleep Disorders, Second Edition (ICSD-2) and the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR).14,15 The DSM-IV-TR divides insomnia into primary and secondary, the latter being related to another mental disorder, a general medical condition (eg, chronic back pain, Alzheimer’s disease, or gastroesophageal reflux disease), or being substance-induced.15 However, the DSM-IV-TR criteria are considered to be less useful than the ICSD-2 criteria, because classification as secondary insomnia may promote undertreatment by focusing on the primary complaint. The term “comorbid insomnia” is preferred.11

The ICSD-214 categorizes sleep disorders as follows: insomnia, sleep-related breathing disorders,
hypersomnia (eg, narcolepsy), circadian rhythm sleep disorders, parasomnias, sleep-related movement disorders, and other sleep disorders. Insomnia is further divided into adjustment insomnia, psychophysiological insomnia, paradoxical insomnia, insomnia due to a mental disorder, inadequate sleep hygiene, behavioral insomnia of childhood, insomnia due to a drug or substance, insomnia due to a medical condition, and miscellaneous categories.

Table 1 lists some of the medical and neurologic conditions associated with insomnia. Most people have secondary insomnia, and it is often assumed that treating the underlying medical condition will eliminate or reduce the sleep problems. However, this often is not the case, and the patient should understand this.

Most patients with insomnia do not have a psychiatric disorder, but if they do, it is most often anxiety or depression (Figure 2). Insomnia is a very common symptom during the course of a mood or anxiety disorder and is more likely to emerge before, rather than during or after, a mood disorder first episode or recurrence. Insomnia is associated with higher rates of lifetime and current major depressive disorder (MDD), and its presence and persistence predict future MDD. Insomnia also predicts poorer outcomes in MDD (ie, persistence, chronicity, and suicidality) and may be related to impaired daytime function seen in MDD.

By contrast, insomnia usually follows a new-onset or recurrent anxiety disorder.

Ironically, several of the medications used to treat depression or anxiety can cause or aggravate insomnia—notably selective serotonin reuptake inhibitors, venlafaxine, duloxetine, and bupropion. Table 2 lists medications commonly associated with insomnia.

**Factors Influencing Insomnia**

Insomnia may be caused by extrinsic or intrinsic factors. Extrinsic factors include inadequate sleep hygiene, which is at least 1 month of activities that precipitate or perpetuate insomnia (eg, working or paying bills in bed, caffeine intake, or sleeping with pets). Intrinsic causes include circadian rhythm disorders, restless legs syndrome (described as feeling like worms are crawling on the skin, tingling, or an uncontrollable urge to move during sleep), and breathing-related sleep disorders (ie, central, obstructive, or hypventilation syndromes). Obstructive sleep apnea (OSA), which is becoming more widely recognized, is characterized by periods of sleep during which airflow decreases or ceases. These periods can extend to 10 to 30 seconds or longer, after which the lower saturated oxygen and elevated carbon dioxide levels prompt the patient to awaken. When patients with OSA resume breathing, they are technically awake (based on EEG recordings), even if they do not remember being awakened. This cycle can repeat several hundred times per night, with a dramatic effect the next day on wakefulness, concentration, and mood. The risk factors for OSA are varied and include obesity, a large neck, alcohol consumption, older age, Asian ethnicity, and children with tonsils. When children become excessively tired, they enter a more hyperactive state. As a result, insomnia in...
children as a result of OSA is often misdiagnosed as attention-deficit/hyperactivity disorder, underscor-
ing the importance of reviewing sleep hygiene with
patients of all ages as part of the general office visit.

Many physiologic processes are influenced by the
body's circadian clock, most notably sleep. Circadian
rhythm disorders result from a problem with the
internal biological clock; they include time zone
change syndrome (or jet lag), shift work sleep disor-
der, delayed sleep phase syndrome (DSPS), advanced
sleep phase syndrome (ASPS), and non–24-hour
sleep/wake syndrome.9 Most people are familiar with
jet lag and shift work disorder, even if they have not
experienced them. ASPS describes a sleep pattern in
which the night phase is moved forward by several
hours. The patient will go to bed earlier than usual
(eg, 8:00 PM) and awaken earlier than usual (eg, 4:00
AM). It also is known as “farmer’s sleep”.

By contrast, individuals experiencing DSPS are
known as “night owls”; this occurs during the
teenaged years but also occurs with regularity in some
adults. The most well-known example is Sunday-
evening insomnia. (Because most people sleep later
than normal on Saturday and Sunday mornings, their
sleep phase cycle is pushed back by Sunday evening.)
Individuals experiencing non–24-hour sleep/wake
syndrome are most frequently blind people, whose
internal clock is out of synch with a 24-hour day,
because they do not register light and, therefore, have
no photic cues for the circadian rhythm.9

**CLINICAL EVALUATION FOR ASLEEP DISORDER**

Diagnosing insomnia involves quantifying the
parameters of sleep: initiation, duration, consolidation,
quality, and daytime impairment. These can be
elucidated with specific questions, such as, “When
did it start?” “How long has it been going on?” “Is

**Table 2. Medications Associated with Insomnia**

| Antidepressants | Selective serotonin reuptake inhibitors  
| Bupropion  
| Monoamine oxidase inhibitors  
| Venlafaxine  
| Antihypertensives | Clonidine  
| β-blockers  
| • Propranolol  
| • Atenolol  
| • Pindolol  
| Methyl dopa  
| Reserpine  
| Antineoplastics | Medroxyprogesterone  
| Leuprolide acetate  
| Goserelin acetate  
| Pentostatin  
| Daunorubicin  
| Interferon α  
| Anticholinergics | Ipratropium bromide  
| Sympathomimetic amines | Bronchodilators  
| • Terbutaline  
| • Albuterol  
| • Salmeterol  
| • Metaproterenol  
| Xanthine derivatives  
| • Theophylline  
| Decongestants  
| • Phenylpropanolamine  
| • Pseudoephedrine  
| Hormones | Oral contraceptives  
| Thyroid preparations  
| Cortisone  
| Progesterone  
| Neurologic | Phenytoin  
| Topiramate  
| Methylphenidate  
| Lamotrigine  
| Levodopa  
| Miscellaneous | Quinidine  
| Opiates  
| Nicotine  
| Caffeine  
| Anacin (acetylsalicylic acid and caffeine)  
| Excedrin (acetaminophen, acetylsalicylic acid, and caffeine)  
| Empirin  
| Cough/cold preparations  


**Table 3. Sleep Assessment Scales**

- Epworth Sleepiness Scale  
- Pittsburgh Sleep Quality Index  
- Berlin Questionnaire  
- Lundt Sleep Questionnaire
the sleep consolidated or broken up?”, or “Does it affect daytime performance?”

The 3 Ps model also provides a good basis for the clinical evaluation. The patient history should include the age of onset of the sleep disorder, the severity, and any functional impact. There are several user-friendly assessment tools, listed in Table 3. We have developed our own sleep assessment form (the Lundt Sleep Questionnaire), an example of which is shown in Figure 3. A sleep diary is also extremely useful for the patient and HCP to help identify risk factors and assess the type of insomnia. An example of the sleep diary we use is provided in Figure 4.

Referral to a sleep specialist would be prudent in cases of excessive daytime sleepiness (ie, impairing functionality or ability to drive safely), suspicion of OSA, dangerous nighttime behavior (eg, sleep eating, walking, or punching), or a lack of response to treatment. In some cases, a polysomnograph (or sleep study) may be required, but these can be difficult to obtain depending

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### Table 3: The Lundt Sleep Questionnaire

**INSTRUCTIONS:** Please answer the questions, based on the past month. Select only 1 answer by filling in the appropriate circle.

<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. How would you rate the quality of your sleep?</td>
<td>Very good, Fairly good, Fairly poor, Very poor</td>
</tr>
<tr>
<td>2. Approximately how many minutes do you take to fall asleep once you decide to go to sleep?</td>
<td>0–15 min, 16–30 min, 31–60 min, Over 60 min</td>
</tr>
<tr>
<td>3. Approximately how many times do you awaken each night?</td>
<td>0, 2–3, 5–6, &gt;5</td>
</tr>
<tr>
<td>4. Approximately how many hours of sleep do you actually get each night?</td>
<td>7–8 hrs, 6–7 hrs, 5–6 hrs, &lt;5</td>
</tr>
<tr>
<td>5. How much do the hours of your sleep change from night to night?</td>
<td>&lt;1, 1–2 hrs, 2–4 hrs, &gt;4</td>
</tr>
<tr>
<td>6. Approximately how much time do you typically spend awake in bed each night?</td>
<td>&lt;30 min, 31–60 min, 1–2 hrs, &gt;2 hrs</td>
</tr>
<tr>
<td>7. How many cups of caffeinated coffee, or other caffeinated beverages, do you drink in an average day?</td>
<td>0–1, 2–3, 4–5, &gt;5</td>
</tr>
<tr>
<td>8. How many alcoholic beverages do you drink each week?</td>
<td>0–1, 2–5, 6–12, &gt;12</td>
</tr>
<tr>
<td>9. How many prescription or nonprescription medications do you take? (for any purpose)</td>
<td>0–1, 2–3, 4–5, &gt;5</td>
</tr>
<tr>
<td>10. In general, how would you rate your health?</td>
<td>Excellent, Very good, Fair, Poor</td>
</tr>
<tr>
<td>11. Has a poor night’s sleep interfered with your activities the next day?</td>
<td>Very unlikely, Somewhat unlikely, Somewhat likely, Very likely</td>
</tr>
<tr>
<td>12. In the past 12 months, have you been late or missed work because of a poor night’s sleep?</td>
<td>No, Yes, &lt;1 time/mo, 2–5 times/mo, &gt;5 times/mo</td>
</tr>
<tr>
<td>13. How much do you worry about sleep or not being able to sleep when you decide to go to sleep?</td>
<td>Not at all, A little bit, Quite a bit, All the time</td>
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<tr>
<td>14. How relaxed do you feel at bedtime?</td>
<td>Very much, Quite a bit, A little bit, Not at all</td>
</tr>
<tr>
<td>15. Over the past month, were you aware or told that you snore?</td>
<td>No, Yes, a little bit, Quite a bit, All the time</td>
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</table>

on the patient’s location, and they are expensive (which may be prohibitive in the absence of insurance coverage). Nonetheless, as will be reviewed in the next article, there are several treatment approaches that can and should be tried before a polysomnograph is warranted.

CONCLUSIONS

Although it is pervasive in our culture, sleep disorders—and in particular insomnia—are underdiagnosed and undertreated, in part because they are under-recognized by the patients themselves. Chronic insomnia eventually affects virtually every qualitative facet of life and measure of functionality (and even our response to alcohol or drugs). Yet, it is easily diagnosed with a more proactive effort by the HCP. With so many diagnostic and assessment tools available, addressing sleep hygiene should be part of every office visit.

REFERENCES


Figure 4. The Lundt Sleep Diary

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
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</thead>
<tbody>
<tr>
<td>1. What time did you first go to bed last night?</td>
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<td>2. About how long did it take you to fall asleep?</td>
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<td>3. About how many times, if any, did you awaken during the night?</td>
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<td>4. Overall, about how many hours did you sleep?</td>
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<td>5. At what time did you wake up (for the last time) this morning?</td>
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<td>6. In general, how did you feel when you woke up?</td>
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<tr>
<td><strong>Answer these last 3 questions each night:</strong></td>
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<tr>
<td>7. How much time, if any, did you spend napping during the day?</td>
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<td>8. Did you consume any of these substances during the day? (eg, caffeine or alcohol)</td>
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<tr>
<td>9. On a scale of 1 to 5, how would you rate your overall functioning during the day?</td>
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