Dedication

To Scott and Caelie, who are the center of my universe

J A M

To Thad, Evan, and Grace for their ongoing love and patience; and to my colleagues at Children’s National Medical Center, for their support and friendship

J A O
Preface to the Third Edition

Sleep profoundly impacts virtually every aspect of a child's physical and mental health, daily functioning, and well-being. Thus, it is not surprising that insufficient, disrupted, poor-quality, and, at times, elusive sleep constitutes one of the most common complaints raised by parents to pediatric and family medicine practitioners, as well as to child mental health providers. Approximately 25% of children overall experience some type of sleep problem, ranging from difficulty falling asleep and nightwakings to more serious primary sleep disorders, such as sleep apnea or narcolepsy; more than one-third of elementary school-aged children and 40% of adolescents have significant sleep complaints. Although many sleep problems in infants and children are transient and self-limited, the common wisdom that children “grow out of” sleep problems is not necessarily accurate, and a host of intrinsic and extrinsic risk factors ranging from demographic variables such as age, gender, and race/ethnicity to the presence of chronic medical, neurodevelopmental, and psychiatric comorbidities to important ancillary issues such as difficult temperament, maternal depression, and family stress may predispose a given child to develop a more chronic sleep disturbance.

 Furthermore, there are few pediatric health issues that have a more significant impact on health and well-being than childhood sleep disorders. Sleep affects every aspect of a child's physical, emotional, cognitive, and social development, and the consequences of sleep disorders in children range from significant cognitive, mood and behavioral concerns, academic failure, health and safety issues (e.g., obesity, hypertension, type 2 diabetes), and an increased risk of accidental injuries and car crashes. Sleep problems in children also have a major impact on the family, often resulting in significant caregiver stress, fatigue, mood disturbances, and a decreased level of effective parenting. In addition, the coexistence of sleep problems exacerbates virtually every medical, psychiatric, developmental, and psychosocial problem in childhood. Given that sleep disorders are highly treatable with effective medical and behavioral interventions, sleep disorders in children and adolescents are particularly important for the primary care healthcare provider to recognize and diagnose.

Sleep also represents an important health education issue. Not only are sleep problems treatable, but many are preventable. Thus, the pediatric health encounter provides an ideal opportunity to educate parents about normal sleep in children and to introduce strategies to prevent sleep problems from either developing in the first place (primary prevention) or becoming chronic when problems already exist (secondary prevention). Primary care providers are in the optimal position to identify sleep concerns because of their regular access to children and families during well-child encounters, especially prior to school entry, and because of the inherently biopsychosocial orientation of primary care practice. However, while pediatric providers have become more vigilant in monitoring and providing education about virtually every aspect of children's health and well-being, survey studies suggest that sleep issues in both primary care and mental health clinical settings are commonly inadequately addressed. Unfortunately, given the expanding number of competing demands for increasingly smaller amounts of time and the information overload that the average busy pediatric practitioner faces on a daily basis, sleep issues in clinical practice may not always receive the attention that they deserve.

The purpose of this updated and expanded edition of *A Clinical Guide to Pediatric Sleep: Diagnosis and Management of Sleep Problems in Children and Adolescents*, therefore, is threefold:

1. To provide the practitioner with an appreciation for the pervasive effect that sleep problems have on children's and families' lives and the multiple ways in which they impact clinical pediatric practice.

2. To synthesize new state-of-the-art information about the etiology, clinical assessment tools, and management of specific sleep disorders in children and adolescents in a format that provides a comprehensive and accessible resource for practitioners.
To provide the basic knowledge and practical tools with which pediatric healthcare providers can recognize, evaluate, and treat sleep issues in children and adolescents in primary care, as well as mental health settings.


*A Clinical Guide to Pediatric Sleep* is divided into four sections, with extensive appendices of handouts for healthcare practitioners to give parents, now available online: Please see inside cover of your book for online access information.

**Section I** addresses basic sleep issues in pediatric practice, including an overview of sleep regulation and core features of sleep physiology relevant to clinical practice. This section also presents information on developmental aspects of sleep, including normal sleep parameters and commonly experienced sleep problems for each age group, as well as updated data regarding epidemiology of pediatric sleep disorders. Finally, basic approaches to evaluation of sleep problems, including the use and interpretation of sleep diagnostic tools, as well as a new chapter on polysomnography, are presented.

**Section II** provides extensively updated, comprehensive information on the etiology, evaluation, treatment, management, and prognosis for each of the most common pediatric sleep disorders. Section II also contains symptom-based algorithms based on the three most common presentations of sleep problems in the clinical setting (i.e., difficulty falling asleep, nightwakings, and daytime sleepiness). Since there are multiple sleep problems that can account for each of these presenting symptoms, the algorithms enable the healthcare provider to evaluate sleep complaints in a stepwise fashion with the goal of developing the most appropriate treatment plan.

**Section III** discusses sleep pharmacology, including sedatives and hypnotics in children and medication effects on sleep. New hypnotics, use of over-the-counter medications in pediatrics, especially melatonin, and misuse of stimulants (e.g., medications such as methylphenidate, caffeine) to manage fatigue in the adolescent population are also covered.

**Section IV** presents updated and expanded information on sleep problems in special populations, including children with neurodevelopmental disorders and pediatric patients with a comorbid medical or psychiatric problem.

Furthermore, an updated national listing of clinical resources and other informational resources (e.g., foundations, Internet sites) is included. Finally, the updated online **Appendices** (see inside cover of your book for online information) provide comprehensive resources for pediatric providers, including an expanded list of intake and screening questionnaires, as well as parent education handouts for each age group and each sleep disorder. All parent education handouts are now provided in English and Spanish.

Our goal is to make it possible for all children and families to get the quantity and quality of sleep they need and to identify and treat disorders that may impact and thus compromise sleep, in order to optimize the health and well-being of children and adolescents. It is our hope is that this book will provide primary care and mental health providers with a state-of-the-art, comprehensive, accessible, and user-friendly resource on pediatric sleep to help us move closer to achieving that goal.
Acknowledgments

No project of this magnitude is ever developed without the enthusiasm and support of others. We would like to extend our thanks to the many individuals who supported this work with their advice and feedback, to the clinicians with whom we have had the privilege of working, and to the families who shared their experiences (and sleepless nights!) to help further our knowledge of pediatric sleep.

We would also like to acknowledge the incredible work of the nonprofit organization Sleeping Children Around the World, whose mission is to provide children in the poorest nations around the globe with the comfort of a good night's sleep, as well as other nonprofits whose goals are to support sleep for youths and their families, including Beds for Kids (Philadelphia, PA) and SweetDreamzz (Detroit, MI).

Finally, our deepest appreciation to those who have supported this project: particularly Jamie Elfrank, Acquisitions Editor of Lippincott Williams & Wilkins, and Ashley Fischer, Product Development Editor, who shepherded this book through the publishing process. We are also grateful to our many colleagues in sleep medicine, past, present, and future, who continue to inspire and educate us.

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Sleep is the primary activity of the brain during early development. It is estimated that by the age of 2 years the average child has spent about 9,500 hours (or a total of 13 months) sleeping, in contrast to 8,000 hours for all waking activities combined. Between the ages of 2 and 5 years, children spend equal amounts of time awake and asleep. And throughout childhood and adolescence, sleep continues to account for about 40% of a child's average day.

The organization and regulation of sleep and wakefulness are complex, highly active physiologic processes that involve the interaction of multiple central nervous system components. A detailed description of the neuroanatomy and neurophysiology of sleep, which may be found in a number of excellent reviews (see Suggested Readings), is beyond the scope of this book. However, some understanding of the function of sleep, the impact of insufficient sleep, the basic structure or architecture of sleep, and familiarity with the basic neural mechanisms that regulate sleep and wakefulness is necessary in order for the primary care practitioner to fully appreciate the causes and effects of both sleep disturbances and insufficient sleep in children and adolescents.

**Definition of Sleep**

Sleep may be defined as a behavioral state characterized by the following:

- Reduced motor activity
- Decreased interaction with and responsivity to the environment
- Specific postures (e.g., lying down, eyes closed)
- Easy reversibility

Sleep is a reversible behavioral state of perceptual disengagement from and unresponsiveness to the environment. It is also true that sleep is a complex amalgam of physiological and behavioral processes.

(Carskadon & Dement, 2011)

**FUNCTIONS OF SLEEP**

Despite the virtual explosion of knowledge in the past half-century regarding the structure, neuroanatomy, and neurophysiology of sleep (e.g., the discovery of rapid eye movement [REM] sleep occurred in the 1950s), the basic function of sleep largely remains a mystery. Most of what we understand about the function of sleep has evolved from studies that have examined the impact of experimentally induced sleep loss or pathologic sleep conditions on a host of physiologic and neurobehavioral systems in both animal models and humans. What we do know is that adequate sleep is a biologic imperative that appears necessary for sustaining life, as well as for optimal functioning. For example, slow-wave sleep (SWS; also termed “deep sleep,” delta sleep, and stage N3 sleep) appears to be the most “restorative” form of sleep and is relatively preserved in the face of insufficient total sleep. The increased amount of SWS in children and the high arousal threshold characteristic of SWS may also be protective in terms of neurodevelopment. REM sleep (see below, Sleep Architecture) appears not only to be involved in vital cognitive functions, such as the consolidation of memory, but also to be an integral component in the growth and development of the central nervous system. Recent studies indicate that adequate amounts of both of these sleep stages are necessary for optimal learning. In addition, the release of growth hormone during SWS clearly links sleep to the regulation of somatic growth as well as many other...
neuroendocrine functions. Sleep may also functionally reflect a neurobiologic need to limit time in the awake state, thereby protecting the individual, especially the developing organism, from being bombarded by information and environmental stimulation that cannot be adequately processed. Finally, studies have clearly demonstrated that “rest” is not a substitute for sleep and, furthermore, that wakefulness-promoting agents (e.g., psychostimulants, caffeine) used to combat sleepiness induced by insufficient sleep do not restore the physiologic benefits of sleep itself.

**Insufficient Sleep**
Insufficient sleep is the most common cause of excessive daytime sleepiness. The resulting chronic sleep loss impacts daytime functioning, including mood disturbances, daytime behavior problems, cognitive impairment, and increased risk-taking behavior.

**INSUFFICIENT SLEEP**
A basic principle of sleep physiology relates to the consequences of the failure to meet basic sleep needs, termed insufficient, inadequate sleep, or “sleep loss.” Many of the early studies conducted examined the effects of total sleep loss (sleep deprivation) rather than the more “real-world” scenario of partial sleep loss (sleep restriction). However, more recent studies have demonstrated that partial sleep loss on a chronic basis accumulates into what is termed a “sleep debt” and produces deficits equivalent to those seen under conditions of total sleep deprivation. If the sleep debt becomes large enough and is not voluntarily paid back (by obtaining adequate recovery sleep), the body may respond by overriding voluntary control of wakefulness, resulting in periods of decreased alertness, dozing off, and napping that is excessive daytime sleepiness. In addition, the sleep-deprived individual may experience very brief (several seconds) repeated daytime microsleeps of which he or she may be completely unaware, but which nonetheless may result in significant lapses in attention and vigilance. There also appears to be a relationship between the amount of sleep restriction and performance, with decreased performance correlating with decreased sleep. Furthermore, subjective perception of sleepiness and the degree of associated performance impairment tend to be poorly correlated with actual impairment, commonly leading individuals to overestimate their ability to function adequately in the face of sleep loss.

**What is Sleepiness?**
Sleepiness is defined as a state of decreased ability to maintain wakefulness or an increased propensity to fall asleep; this is in contrast to fatigue, which typically does not include this sleep tendency. Sleepiness, like hunger or thirst, is a normal biologic need; sleep is to sleepiness as food is to hunger. Furthermore, the only thing that replaces sleep is sleep (i.e., not “rest”). Excessive sleepiness is a symptom characterized by difficulty maintaining wakefulness and an increased propensity to fall asleep, even in inappropriate circumstances and situations, that interferes with activities of daily living.

Chronic sleep loss impacts daytime functioning and causes excessive daytime sleepiness, which can manifest in a number of ways in children and adolescents (e.g., dozing off while engaged in activities, hyperactivity, behavioral problems). Falling asleep at an unintended time, especially in school, is a common manifestation, as are unplanned naps and planned naps past an age when napping is appropriate. Functional neuroimaging studies in both adults and children have suggested that chronic sleep loss selectively impacts the following brain regions: the prefrontal cortex (PFC), which houses complex cognitive or “executive” functions (e.g., time management, decision making, organization, selective attention, judgment, motivation, monitoring and modifying behavior, predicting outcomes); the amygdala, which regulates emotional responses; and the
striatum, which controls reward-related behaviors. For example, in one study in which sleep-restricted volunteers viewed emotional images, there was an increased response of the “emotional brain” (amygdala) combined with a weaker connection between the amygdala and the PFC, implying a heightened emotional response with less emotional control. Studies also suggest that insufficient sleep is linked to changes in reward-related decision making, and so these individuals tend to take greater risks and are less concerned about the potential negative consequences of their behavior. In addition, it should be noted that both the PFC and the striatum are undergoing important structural and functional changes during adolescence, a period during which the risk of chronic sleep loss is especially high.

### Sleep Deprivation/Prolonged Wakefulness Affects the Brain

On a basic neurobiologic level, sleep loss (and its parallel, extended wakefulness) profoundly impacts brain function:

- Neuronal functions
- Neuronal “plasticity”: ability of the brain to change structure/function in response to the environment
- Downscaling of synapses to compensate for net increase in synapse formation and strength during the wake state
- Gene activation/expression
- Neurogenesis
- Brain cell protection/repair from stress
- Neurotransmitters (e.g., serotonin, dopamine)
- Melatonin production/circadian biology
- Cellular metabolism, neurogenesis, brain/eye development
- Highest susceptibility during critical developmental periods
- Increases in stress response and stress hormones

The relationship between sleep and memory has also been actively explored. Both SWS and REM sleep play an active and important role in regard to memory, particularly memory consolidation (as opposed to encoding of memories during wakefulness). For example, postlearning reactivation and reorganization of memory representation occurs during sleep, with potential impact on long-term subsequent memory performance. REM sleep may be particularly important for emotional memory processing.

These effects on brain function account for many of the observed behavioral and cognitive sequelae of chronic sleep loss. Insufficient sleep, however, can also manifest in the following manner:

- **Fatigue and daytime lethargy**, including increased somatic complaints (e.g., headaches, muscle aches)
- **Mood disturbance**, including complaints of moodiness, irritability, emotional lability, increased negative emotions, depression, and anger
- **Cognitive impairment**, including problems with memory, attention, concentration, decision making, and problem solving
- **Daytime behavioral disinhibition**, including hyperactivity, oppositional, defiant, and aggressive behavior, impulsivity, and noncompliance
- **Poor impulse control and increased risk-taking behaviors**, especially in adolescents
- **Academic problems**, including chronic tardiness related to insufficient sleep and school failure resulting from
chronic daytime sleepiness

- **Use of stimulants**, and other alertness enhancers such as caffeine and nicotine, to artificially maintain wakefulness and combat daytime fatigue

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**Functions of Sleep: Cognition**

Sleep is needed to:

- Remember what we learned
- Organize our thoughts, predict outcomes and avoid consequences, be goal-directed (“executive functions”)
- React quickly
- Work accurately and efficiently
- Think abstractly
- Be creative

There are multiple causes of insufficient sleep, including that resulting from a primary sleep disorder (e.g., insomnia, obstructive sleep apnea); however, the predominant reasons are environmental:

- Academic and extracurricular demands can result in delayed bedtimes.
- Social activities, such as late-night socializing (including on the Internet), often delay bedtime in older children and adolescents.
- Electronic media use such as television viewing, smartphone use, and playing computer/video games, particularly if the electronic devices are readily available in the child's bedroom, may take priority over bedtime in some families.
- Part-time employment, especially if over 20 hours per week, is associated with insufficient sleep in teenagers.
- Early school start times (particularly before 8:00 a.m.) are a major risk factor for insufficient sleep in middle- and high-school students and adolescents. Given the need for 8.5 to 9.5 hours of sleep in adolescents, coupled with a biologically based circadian phase delay occurring in conjunction with puberty, early school start times do not enable most adolescents to obtain sufficient sleep on school nights.

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**SLEEP ARCHITECTURE**

**Basic Sleep Terminology**

- **Sleep architecture**: structure/stages of sleep: REM, non-REM (NREM; stages 1-3), wake
- **Ultradian rhythms**: the nocturnal cycle of sleep stages
- **Circadian rhythms**: the 24-hour rhythm of sleep/wakefulness and many physiologic systems (e.g., body temperature, hormones)
- **Sleep regulation**: determinants of sleepiness and alertness levels
- **Sleep patterns**: a combination of biology, learning, maturation, culture, and environment

The framework or architecture of sleep is based on the recognition of three distinct states: *wake, NREM sleep,* and *REM sleep* (or “dream” sleep). These stages are defined by distinct polysomnographic features of
electroencephalographic (EEG) patterns, eye movements, and muscle tone. Note that a summary of normal sleep architecture parameters in children and adolescents can be found in Chapter 4 (Tables 4.4 and 4.5).

**NREM sleep** may be viewed as a period of relatively low brain activity during which the regulatory capacity of the brain continues to be active and in which body movements are preserved. Recent evidence suggests that the sleeping brain is still able to process external information and assess its salience. Respiratory and cardiovascular parameters are regular in NREM sleep. After about the age of 6 months, NREM may be further divided as follows:

- **Stage 1 (N1)** sleep occurs at the sleep-wake transition. Initial stage 1 typically lasts from 30 seconds to 5 minutes. Recall of fragmented visual imagery (hypnogogic hallucinations) as well as brief involuntary muscle contractions (hypnic jerks) may occur, both of which are considered to be normal phenomena in most cases.

- **Stage 2 (N2)** sleep is usually considered the initiation of “true” sleep. It is characterized by bursts of rhythmic rapid EEG activity called sleep spindles (fluctuating episodes of fast activity, occurring after 4 weeks of age) and high-amplitude slow-wave spikes called K complexes (first occurring at 6 months). The initial stage 2 period lasts from 5 to 25 minutes.

- **Stage 3 (N3)** is also known as “deep” sleep, slow-wave sleep (SWS), or delta sleep (note that SWS is no longer divided into stages 3 and 4). This stage of sleep is dominated by delta waves, characterized by high-voltage, low-frequency activity. Respiration is slowest and most regular during SWS, and parasympathetic activity is high. The highest arousal threshold (the period during which it is most difficult to awaken) also occurs during SWS; the lowest arousal threshold (the period during which it is easiest to awaken) is in stage 1. The initial SWS period is about 30 to 45 minutes and is followed by a brief arousal (i.e., transition to wakefulness or to a lighter-sleep stage).

- **REM sleep** is characterized by desynchronized cortical activity (low-voltage, high-frequency EEG) and the highest brain metabolic rate, dreaming, absence of skeletal muscle tone (except for diaphragm, middle ear, and erectile muscles), lack of normal thermoregulation, and episodic bursts of phasic eye movements that are the hallmark of REM sleep. Several of these characteristics of REM sleep (e.g., muscle atonia, dream mentation) have direct clinical relevance to symptoms of REM-associated sleep disorders such as narcolepsy (e.g., cataplexy, hypnogogic hallucinations). In young infants, REM sleep is termed “active sleep” and is characterized by frequent muscle twitches and grimaces that parents may interpret as abnormal. Until age 3 months, infants also enter sleep through REM. REM-associated alterations in autonomic parameters (increased sympathetic tone) and changes in control of breathing not only result in irregular respiration and heart rate, but also typically increase the severity of sleep-disordered breathing during this stage of sleep. The first REM sleep period occurs about 70 to 100 minutes after sleep onset (REM onset latency) and lasts for about 5 minutes.

**Sleep Cycles**

NREM and REM sleep alternate throughout the night in cycles (**ultradian** cycles or rhythm) of about 90 to 110 minutes (50 minutes in infancy and gradually lengthening to adult levels at about school age); this typical pattern of nocturnal sleep is illustrated in a hypnogram (**Figure 1.1**). Brief arousals normally followed by a rapid return to sleep often occur at the end of each sleep cycle (4-6 times per night); this pattern of normal arousals plays an important role in the etiology of problematic nightwakings in infancy and childhood (see **Chapter 7**). The relative proportion of REM and NREM sleep per cycle changes across the night, such that SWS predominates in the first third of the night and REM sleep in the last third. That is, REM sleep percentage increases and SWS percentage
declines over the course of the night. The potential clinical significance of this timing of sleep stages is important. For example, partial arousal parasomnias that occur during NREM sleep primarily occur at the beginning of the night. In addition, the amount of REM sleep is selectively compromised with forced early-morning waking, such as occurs when an adolescent has to awaken for an early school start time.

The amount and timing of each sleep stage are also affected by a multitude of extrinsic factors. For example, the amount and depth of SWS is determined by prior sleep loss, as well as the time of sleep onset and the length of prior wakefulness. SWS is relatively “protected” by its appearance early on in the nocturnal sleep period and is also preserved at the expense of other sleep stages when total sleep amounts are restricted. There is also typically a marked increase in SWS (“rebound”) during nights of recovery sleep following sleep restriction. REM sleep also demonstrates this rebound phenomenon in recovery sleep, further highlighting the physiologic importance of these two sleep stages. Increased arousals, such as those occurring as in relation to obstructive sleep apnea or periodic limb movements, result in sleep fragmentation and reduced amounts of SWS and REM sleep. With an increase of REM sleep, there may be reports of increased and/or more vivid dreams. REM sleep is also intimately linked to circadian core body temperature rhythms. The relative percentage of sleep stages on a given night is also a reflection of other factors, such as circadian rhythm disruption associated with shift work or jet lag (causes disruption of timing of REM sleep), stress (may increase REM sleep need), and medications, which have both direct effects and withdrawal effects, especially on SWS and REM sleep (see Chapter 20).
Sleep also changes as a function of age (see Table 1.1). Both the emergence of defined sleep stages and the proportion of sleep that each occupies have a distinct ontogenic and developmental pattern. Although a more detailed discussion of the development of sleep patterns and behaviors across infancy and childhood is found in Chapter 2, several general trends in normal sleep patterns across childhood are listed below; these may be summarized as the gradual assumption of more adult sleep patterns as children mature. These trends reflect the physiologic and chronobiologic, developmental, and social and environmental changes that are occurring across childhood and include:

- **A decline in the average 24-hour sleep duration** from infancy through adolescence, which involves a decrease in both diurnal and nocturnal sleep amounts. In particular, there is a gradual decline in daytime sleep (scheduled napping) over the first 5 years of life, with a less marked and more gradual continued decrease in nocturnal sleep amounts into late adolescence.

- **A marked decrease in the proportion of REM sleep** from birth (50% of sleep) through early childhood into
peaks in early childhood, drops off abruptly after puberty (40%-60% decline), and then further decreases over the life span. This SWS preponderance in early life has clinical significance; for example, the high prevalence of partial arousal parasomnias (sleepwalking and sleep terrors) in preschool and early school-aged children is related to the relative increased proportion of SWS in this age group.

**TABLE 1.1. Normal Developmental Changes in Children's Sleep Architecture**

<table>
<thead>
<tr>
<th>Age Category</th>
<th>Sleep Physiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborns</td>
<td>Three sleep states: active (&quot;REM-like,&quot; 50% of sleep), quiet (&quot;non-REM-like&quot;), and indeterminate Enter sleep through active state</td>
</tr>
<tr>
<td>Infants (0-1 y)</td>
<td>Three amount of active/REM sleep declines Development of 4 stages of non-REM sleep Sleep cycles every 50 min Enter sleep through non-REM</td>
</tr>
<tr>
<td>Toddlers (1-3 y)</td>
<td>REM sleep amounts continue to decline</td>
</tr>
<tr>
<td>Preschool (3-6 y)</td>
<td>REM sleep amounts continue to decline Sleep cycles every 90 min High levels of SWS</td>
</tr>
<tr>
<td>Middle childhood</td>
<td>Latency from sleep onset to REM sleep increases High sleep efficiency (time asleep/time in bed)</td>
</tr>
<tr>
<td>(6-12 y)</td>
<td></td>
</tr>
<tr>
<td>Adolescence (&gt;12 y)</td>
<td>40% decline in SWS REM sleep at adult levels (25%-30%)</td>
</tr>
</tbody>
</table>

- A concomitant decrease in the number of end-of-cycle arousals across the nocturnal sleep period due to the lengthening of the ultradian sleep cycle.
- A gradual shift to a later bedtime and sleep onset time that begins in middle childhood and accelerates in early- to mid-adolescence.
- Irregularity of sleep-wake patterns characterized by increasingly larger discrepancies between school night and non-school night bedtimes and wake times and increased weekend oversleep that typically begins in middle childhood and peaks in adolescence.

**NEUROANATOMY AND PHYSIOLOGY OF SLEEP**

Although it shares many features with the awake state, sleep is not merely the absence of wakefulness and vice versa. There are many highly complex neural networks and related processes that actively control the three distinct states of wakefulness, NREM sleep, and REM sleep. Furthermore, sleep is an active process during...
which many physiologic, metabolic, and neurobehavioral functions continue to occur.

Wakefulness is promoted by ascending projections that originate in neurons located in the brain stem (reticular formation) as well as hypothalamic pathways (Figure 1.2). These largely excitatory neurons relay sensory input to the thalamus, hypothalamus, and basal forebrain and activate vast areas of the cortex to increase wakefulness; their activity is suppressed during sleep. Cholinergic neurons of the dorsal midbrain and pons (pedunculopontine [PPT] nucleus and the laterodorsal tegmental [LDT] nucleus) also demonstrate increased activity during wakefulness and REM sleep and decreased activity during NREM sleep; they send excitatory projections to the thalamus, where they then regulate cortical activity and allow the flow of information through the thalamus to and from the cortex (thalamocortical activation). Cholinergic neurons located in the basal forebrain also send projections throughout the cortex, hippocampus, and amygdala; their activity is high during wakefulness and REM sleep and low during NREM sleep.

FIG 1.2. Ascending arousal systems in the brain stem and posterior hypothalamus. PPT/LDT, pedunculopontine and laterodorsal tegmental areas; LC, locus coeruleus; TMN, tuberomammillary nucleus; SN/VTA, substantia nigra and ventral tegmental area; BF, basal forebrain; ACh, acetylcholine; HA, histamine; DA, dopamine; 5-HT, serotonin; NE, norepinephrine. From España RA, Scammell TE. Sleep neurobiology for the clinician. Sleep 2004;27(4):811-820.

A number of neurotransmitters and neuropeptides actively modulate and influence wakefulness promotion. These include acetylcholine (increased in wakefulness and REM sleep) and a number of aminergic neurotransmitters (increased in wakefulness and very low in REM; see Figure 1.2), including histamine in the
tuberomammillary nucleus (posterior hypothalamus); dopamine in the ventral tegmental area, substantia nigra, posterior hypothalamus, and brain stem; serotonin in the median and dorsal raphe; and norepinephrine (NE) in the locus coeruleus (midbrain). While coordinated activity across these arousal systems is necessary for complete and sustained awake states, each aminergic pathway may mediate different functions of wakefulness. For example, histamine appears to be the major arousal-promoting neurotransmitter at wake onset; NE increases cortical activation, particularly under conditions of stress and in the presence of novel stimuli; and dopamine may be more likely to promote wakefulness under conditions of motivation or physical activity. Finally, neurons in the lateral or posterior hypothalamus that produce hypocretin (also called orexin) are also active during the wake state (Figure 1.3); a deficiency of hypocretin/orexin has been shown to be the primary etiology of both the excessive daytime sleepiness and cataplexy in narcolepsy. Hypocretin/orexin function also appears to be linked to control of feeding behaviors, locomotion, and autonomic functions.

The ventrolateral preoptic area (VLPO) in the anterior hypothalamus is a major sleep-promoting area of the brain (Figure 1.4); most likely distinct subregions of the VLPO control NREM and REM sleep. During sleep, especially SWS, VLPO neurons are active and exhibit high firing rates. VLPO neurons send projections to all major wake-promoting regions, including the tuberomammillary nucleus, locus ceruleus, and LDT and PPT. These inhibitory neurons are believed to induce sleep by coordinating the inhibition of all the wake-promoting cholinergic and aminergic regions. Most VLPO neurons release the inhibitory neurotransmitter g-aminobutyric acid (GABA) at their sites of projection, while some utilize the inhibitory neurotransmitter galanin. The control of REM sleep involves the interaction of brain stem cholinergic and aminergic neurons in a complex feedback loop; neurons releasing acetylcholine (LDT/PPT region) are disinhibited by the suppression of aminergic neurons (e.g., NE, histamine) during REM (Figure 1.5). REM-associated muscle atonia is linked to inhibition or loss of excitation of motor neurons in the brain stem and spinal cord via the medulla; these pathways originate in the LDT and PPT and involve neurotransmitters, including acetylcholine, glutamate, and glycine.

These complex relationships help to explain the powerful sleep effects of drugs that alter the balance in these complex and interrelated systems. For example, sleep-promoting benzodiazepines and antihistamines enhance GABA signaling and block histamine receptors, respectively. Tricyclic antidepressants and serotonin reuptake inhibitors act as REM suppressants because of their enhancement of aminergic signals and the resultant inhibition of REM-promoting neurons. Psychostimulants promote wakefulness by increasing dopamine and NE signals (see also Chapter 20).

**SLEEP REGULATION**

On a basic level, sleep and wakefulness are usually described as being regulated by two highly coupled processes operating simultaneously (the “two process” sleep system [Figure 1.6]):

- **Homeostatic process (“process S”):** This process primarily regulates the length and depth of sleep. The homeostatic drive may be related to the accumulation of adenosine and other sleep-promoting chemicals (“somnogens”), such as cytokines, during prolonged periods of wakefulness; caffeine most likely exerts its wake-promoting effect by blocking adenosine receptors. This sleep pressure appears to build up more quickly in infants and young children, thus limiting the duration of sustained wakefulness during the day and necessitating short periods of daytime sleep (i.e., naps).

- **Endogenous circadian rhythm (“process C”):** This process influences the internal organization of sleep and timing and the duration of daily sleep-wake cycles. It also governs predictable patterns of alertness.
throughout the 24-hour day. The “master circadian clock” that controls sleep-wake patterns is located in the suprachiasmatic nucleus (SCN) in the ventral hypothalamus; however, we now know that “circadian clocks” are present in virtually every cell in the body and help to govern the timing of various other physiologic systems in the body (e.g., cardiovascular reactivity, hormone levels, renal and pulmonary functions). Circadian rhythms are generated by the expression of specific “clock genes.” The circadian timing system develops rapidly in the first 6 months of life as a result of the combined influence of neurodevelopmental maturation and social and environmental cues (especially light-dark cycles).

![Diagram of sleep stages]

**FIG 1.6.** Normal distribution of sleep stages in healthy children, adults, and the elderly. From Dement WC, Vaughan C. *The promise of sleep: a pioneer in sleep medicine explores the vital connection between health, happiness, and a good night's sleep.* New York: Delacorte Press, 1999.

The relative level of sleepiness (sleep propensity) or alertness existing at any given time during a 24-hour period is partially determined by the duration and quality of previous sleep as well as time awake since the last sleep period (the homeostatic drive or “sleep drive”). Interacting with this “sleep homeostat” is the 24-hour cyclic pattern or rhythm characterized by clock-dependent periods of maximum sleepiness (“circadian troughs”) and maximum alertness “circadian peaks”. There are two periods of maximum sleepiness—one in the late afternoon (3:00-5:00 p.m.) and one toward the end of the night (3:00-5:00 a.m.)—and two periods of maximum alertness—one in mid-morning and one in the evening, just prior to sleep onset (the so-called second wind). In addition, relative sleepiness and wakefulness are influenced by many other variables, including individual factors (e.g., age and individual variations in sleep needs and tolerance to the effects of inadequate sleep), the nature of the task being performed (e.g., type, length, complexity), and environmental and physiologic factors (e.g., noise, light, ambient temperature, satiety) that may “unmask” but do not cause sleepiness. Finally, a phenomenon known as “sleep inertia,” defined as a period of incomplete arousal characterized by confusion, disorientation,
cognitive slowing, and irritability occurring immediately upon waking in the morning or after a nap, especially from SWS, may further compromise alertness levels.

Because the human circadian clock is actually slightly longer than 24 hours, intrinsic circadian rhythms are synchronized or “entrained” to the 24-hour-day cycle by environmental cues called “zeitgebers.” In the absence of zeitgebers, circadian rhythms are desynchronized or “uncoupled” from one another (what is termed the “free-running state”). The most powerful of these zeitgebers is the light-dark cycle; light signals are transmitted to the suprachiasmatic nucleus via the circadian photoreceptor system within the retina (functionally and anatomically separate from the visual system), which switch the body’s production of the hormone melatonin off (light) or on (dark) by the pineal gland. The evening pattern of melatonin secretion can be assessed by sequential measures of melatonin in saliva under dim light conditions (dim light melatonin onset, or DLMO); although currently largely a research tool, kits are expected to become commercially available in the future. The biomarker core body temperature, which falls just prior to sleep onset, and hormone levels (e.g., cortisol) are also linked to circadian timing.

Circadian rhythms are also synchronized by other external time cues, such as timing of meals and alarm clocks. Thus, daytime schedules that are not consistently regulated may further exacerbate circadian disturbances such as delayed sleep-phase disorder. Finally, research also supports an important role for genetics in determining intrinsic circadian clock periodicities and, thus, influencing individual circadian preference for sleep-wake cycle timing or chronotype (“night owl” versus “morning lark”). Moreover, a strong circadian “eveningness” preference or chronotype, compared to “morningness” and intermediate types, has been shown to be independently associated with a number of adverse health, behavioral, and performance outcomes, such as lower physical activity, depression, impulsivity, aggressive and antisocial behavior, substance and alcohol use, lower academic achievement, and increased perceived stress level in adolescents.

The Importance of Morning Light Exposure

Morning light exposure is one of the most powerful influences on a person’s sleep-wake schedule. Early-morning light exposure basically sets a person’s internal clock for the day. For example, research has shown that morning light influences sleep consolidation in newborns and infants, and it is critical for keeping an adolescent's schedule on track. So families should be encouraged to expose their children to bright light in the morning, whether heading out for a walk or a trip to the playground, eating breakfast in the sunniest place in the house, or having adolescents open their bedroom shades and avoid wearing sunglasses on their way to school.

SLEEP AS AN INTRINSIC BIOLOGIC PROCESS AND AS A LEARNED BEHAVIOR

Despite the seemingly contradictory nature of this statement, it is important for the pediatric practitioner to understand the implications of both of these concepts in approaching sleep and sleep problems in children. Few clinical paradigms in pediatrics are better examples of the need for a biopsychosocial approach than sleep. While our scientific understanding of the structure, organization, regulation, and development of sleep has advanced significantly in the past decades and has elucidated much regarding the genetics and neurobiology of sleep, it is clear that sleep is also impacted by multiple psychosocial factors, ranging from exposure to environmental toxins (such as lead), to cultural variables (such as co-sleeping practices), and to community standards (such as school start times). While much of the chronobiologic “hard-wiring” of sleep is immutable, the plasticity of the developing brain makes it likely that sleep and its relationship to other neural systems in the brain are also highly susceptible to environmental influences. Furthermore, the construct that much of sleep behavior is learned behavior not only
underscores the importance of developing health-promoting sleep behaviors early in childhood but also emphasizes the role that parents and healthcare providers may play in shaping and modifying sleep behaviors in children.
GENERAL CONSIDERATIONS
The evaluation of pediatric sleep problems first and foremost requires a basic understanding of what constitutes “normal” sleep in infants, children, and adolescents. Sleep disturbances, as well as many characteristics of sleep itself, have some distinctly different features in children from those in adults. As outlined in Chapter 1, normal sleep architecture changes significantly over the first two decades of life. In addition, sleep patterns and sleep behaviors also evolve and are modified by both “intrinsic” (e.g., normal development) and “extrinsic” (e.g., environment, parenting practices) processes as children progress from infancy to middle childhood through adolescence.

Appreciation and understanding of these changes are needed to provide parents with anticipatory guidance regarding normal sleep and sleep patterns at different developmental stages. For example, two important developmental “milestones” are normally achieved during the first 6 months of life; these are known as sleep consolidation and sleep regulation. Sleep consolidation is generally described as an infant's ability to sleep for a continuous period of time that is concentrated during the nocturnal hours, augmented by shorter periods of daytime sleep (naps). Because this developmental progression occurs over the first few months of life, parents should be counseled not to expect their infant to “sleep through the night” until about 10 to 12 weeks of age. Sleep regulation refers to the infant's ability to be able to control internal states of arousal (e.g., “self-soothe”) in order to both fall asleep at bedtime without parental intervention or assistance, and fall back to sleep following normal brief arousals during the night. Thus, anticipatory guidance counseling to put a baby to sleep “drowsy but awake” is most appropriately initiated at the 2-month visit, so that parents can begin to establish the conditions under which their baby can more readily learn to self-soothe. Studies indicate that sleep at 3 months is an important predictor of future sleep habits, as the ability to fall asleep independently at this age is associated with fewer nightwakings at 6 and 12 months. Thus, preventive education is essential.

The relative prevalence and the various types of sleep problems that occur throughout childhood must also be understood in the context of normal physical and cognitive/emotional phenomena that are occurring at different developmental stages. For example, normal separation anxiety in older infants may be associated with increased bedtime problems; development of normal fears in toddlers may result in nighttime fears and problematic nightwakings; and the prevalence of obstructive sleep apnea in preschoolers is linked to the relative prominence of lymphoid tissue (adenotonsillar hypertrophy) in early childhood. Parental identification and reporting of sleep problems in children also varies across childhood, with parents of infants and toddlers more likely to be aware of sleep concerns than those of school-aged children and adolescents. For example, a poll by the National Sleep Foundation (NSF) found that just 7% of parents thought that their adolescent had a sleep problem, whereas 16% of adolescents themselves thought so. One-third of these adolescents had not told anyone about their sleep issues. Finally, the very definition of a “sleep problem” by parents is often highly subjective and is commonly determined by the amount of disruption caused to parents’ sleep.

In addition to considering sleep disturbances in a developmental context, it is important for the clinician to recognize that a number of other important child, parental, and environmental variables affect the type, relative prevalence, chronicity, and severity of sleep problems.

Child variables that may significantly impact sleep include temperament and behavioral style, individual variations in circadian preference, medical problems, delays in development, and acute and chronic stress.
Parental variables include parenting styles, parents’ educational level and knowledge of child development, mental health issues, family stress, quality and quantity of parents’ sleep, and fatigue level. Environmental variables include the physical environment (space, sleeping arrangements), family composition (number, ages, and health status of siblings and extended family members), lifestyle issues (working parents, regularity of daily schedule), and cultural issues and family values (importance and meaning of sleep, acceptance of co-sleeping). Finally, research including over 40,000 children ages 0 to 3 years indicates great variability in sleep amounts and sleep behaviors across cultures, with children in different countries and regions obtaining significantly more or less overall sleep at night. The findings of cross-cultural studies, however, suggest that there may be relatively less variability across cultures in young children in regard to night waking and the factors that contribute to sleep disruption. Interestingly, there are almost no differences in daytime sleep, indicating that napping likely has a strong biological context, whereas nighttime sleep is more influenced by environmental and cultural factors, as well as potentially by biologic and genetic factors. Other studies also indicate that the amount of daytime sleep is strongly negatively correlated with age and that the influence of environment is relatively limited.

Nature versus Nurture
Sleep habits, in general, may be viewed as learned behaviors superimposed on fundamental processes of circadian and sleep biology that, in turn, may be modified by genetic factors and developmental changes.

A description of normal sleep patterns, developmental issues, common parental sleep concerns, prevalence and types of common sleep problems, and other sleep issues in different age groups is provided below, followed by general sleep hygiene tips. Suggested specific anticipatory guidance points to emphasize during well-child visits are provided in Table 2.1.

What is “Enough” Sleep for Children?
The not-so-simple answer to this question has been the subject of some controversy over the past several years, with some authors challenging the notion that currently “recommended” sleep amounts for children of different ages are based on valid empirical data. While recent research in adults and some preliminary data in children suggest that there may be some individual genetically based variability in both sleep need and tolerance to not meeting those needs, there is as of yet no means of objectively assessing these differences in the clinical setting. Individual variability in sleep times appears to be highest in the first 3 years, especially in the first 12 months, but whether this variability represents “nature” versus “nurture” (or both), or is a by-product of inherent methodological limitations in collecting epidemiological data in large samples (e.g., “sleep duration” is actually parent-reported time in bed), remains to be seen.

The bottom line is that current sleep duration recommendations include a range of what is considered “typical” for age; moreover, because they are necessarily approximations for the “average” child, they should be viewed by parents and practitioners as such and not as a “prescription” for a specific sleep amount for a given individual child. These recommendations should also always be framed in the context of the question “what is the amount of sleep that this child needs to feel well-rested?” Thus, additional parameters that need to be considered include the amount of sleep a child typically gets when allowed to sleep “ad lib”; for example, if a child sleeps longer on weekends, that means he or she needs more sleep on weekdays.

Waking spontaneously in the morning at the desired time (e.g., without alarm clocks, multiple parental reminders) is also a good indicator of sufficient sleep. Thus, monitoring sleep amounts and mood, while allowing a child to sleep until he or she awakens spontaneously in the morning for at least 3 days (e.g.,
During vacation, may help in sorting out this issue. Finally, it is important for parents to recognize that end-of-the-day behaviors that they may associate with being “overtired” (whininess, moodiness, having a “short fuse,” hyperactivity) may be evidence of chronically insufficient sleep.

### TABLE 2.1. Anticipatory Guidance for Sleep Issues in Infants, Children, and Adolescents

**Prenatal Visit**
- Discuss normal newborn sleep patterns (day/night reversal, sleep amounts per 24 h, average sleep-wake periods).
- Discuss plans for sleeping arrangements.

**Newborn Visit**
- Discuss normal newborn sleep patterns (day/night reversal, sleep amounts per 24 h, average sleep-wake periods, irregularity).
- Discuss specific safe sleep practices (sleeping position, surface, environment).
- Breast-fed baby: Discuss shorter sleep periods, avoidance of caffeine.
- Highlight importance of parental sleep needs.

**2-wk Visit**
- Review normal newborn sleep patterns (day/night reversal, normal range of total sleep per 24 h, average sleep-wake periods, irregularity).
- Review safe sleep practices (sleeping position, surface, environment).
- Breast-fed baby: Discuss shorter sleep periods, avoidance of caffeine.
- Highlight importance of parental sleep needs.

**2-mo Visit**
- Discuss normal development of infant sleep patterns (normal range of total sleep per 24 h, “sleeping through the night,” self-soothing, appropriate sleep-onset associations).
- Encourage parents to put their baby to sleep *drowsy but awake* starting at about 10-12 wk of age to avoid the development of inappropriate sleep associations.
- Encourage parents to make the transition to final sleeping arrangements (e.g., bassinet to crib; parents' room to baby's room) by 10-12 wk.
- Explain to parents that periodic, brief arousals during the night are part of a baby's normal sleep pattern, which may help to avoid unnecessary parental intervention and subsequent reinforcement of nightwaking.
- Review safe sleep practices (sleeping position, surface, environment).
- Encourage establishment of a bedtime routine.
- Breast-fed baby: Discuss shorter sleep periods, avoidance of caffeine.
- Highlight importance of parental sleep needs.

**4-mo Visit**
- Discuss normal development of infant sleep patterns.
- Encourage parents to put their baby to sleep *drowsy but awake*.
- Discuss importance of a daily schedule with consistent feeding times and sleep times.
- Suggest that during-the-night feedings should be discouraged after about 6 mo, as they may contribute to inappropriate learned sleep behaviors and are no longer physiologically necessary for most healthy babies.
- Encourage establishment of a bedtime routine.
- Review safe sleep practices (sleeping position, surface, environment).

**6-mo Visit**
• Discuss normal development of infant sleep and napping patterns.
• Encourage parents to put their baby to sleep drowsy but awake.
• Suggest that during-the-night feedings should be discouraged after about 6 mo, as they may contribute to inappropriate learned sleep behaviors and are no longer physiologically necessary for most healthy babies.
• Encourage establishment of a bedtime routine and consistent sleep schedule.
• Anticipate possible temporary regression in sleep behaviors with developmental milestones, illness, and changes in routine.
• Invite discussion of co-sleeping.

9-mo Visit
• Discuss normal development of infant sleep and napping patterns.
• Anticipate recurrence of nightwakings at 9-12 mo; discourage parental reinforcement of nightwakings.
• Encourage establishment of a bedtime routine and consistent sleep schedule.
• Invite discussion of co-sleeping.

1-y Visit
• Discuss normal development of toddler sleep and napping patterns.
• Encourage use of transitional objects.
• Discuss transition to single daily nap period.
• Encourage bedtime routine and regular bedtime.
• Discourage bedtime bottles.

15-mo Visit
• Discuss normal development of toddler sleep and napping patterns.
• Encourage use of transitional objects.
• Discuss transition to single daily nap period.
• Discourage bedtime bottles.

18-mo Visit
• Discuss normal development of toddler sleep and napping patterns.
• Encourage transitional objects.
• Discourage bedtime bottles.
• Discuss nighttime developmental fears (especially separation).
• Discourage electronic use (including television, e-readers) at bedtime.

2-y Visit
• Discuss normal development of toddler sleep and napping patterns.
• Discuss effects of insufficient sleep on daytime behavior.
• Discuss transition from crib to bed.
• Discuss parental limit setting.
• Discourage electronic use (including television, e-readers) at bedtime.

3-5-y Visits
• Discuss normal development of sleep and napping patterns in preschoolers.
• Discuss effects of insufficient sleep on daytime behavior and review signs of sleepiness.
• Discuss development of good sleep habits.
• Discuss parental limit setting.
• Discourage electronic use (including television, e-readers) at bedtime and encourage no televisions or other electronics in the child's bedroom.

6-12-y Visits
• Discuss normal range sleep amounts in school-aged children.
• Encourage obtaining sufficient sleep and appropriate bedtime.
• Discuss impact of insufficient sleep on school performance.
Discuss development of good sleep habits.
Discourage electronic use (including television, e-readers) at bedtime and encourage no televisions or other electronics in the child's bedroom.
Discourage caffeine use.

**Adolescent Visits**

Discuss average sleep needs (9-9½ h) in teenagers.
Encourage obtaining sufficient sleep and appropriate bedtime.
Discuss avoiding discrepant weekday and weekend schedules.
Review pubertal influences on sleep (phase delay).
Review healthy sleep habits, including regular bedtimes and waketimes, avoidance of caffeine, avoidance of electronics (e.g., televisions, smartphones, video games, laptops/computers) in bedroom.
Discuss dangers of drowsy driving.

There are a number of other considerations that should be included in any assessment of sleep duration in children. First, many studies do not differentiate between weekday and weekend (or nonschool day) sleep, especially in younger children. However, this weekday-weekend discrepancy, which is usually characterized by longer “catch-up” sleep on Friday and Saturday, is no longer a phenomenon exclusive to adolescents, but is observable in many school-aged and younger children as well. Second, while information regarding daytime naps is typically solicited in younger children, this may not be queried in older children and adolescents, who nonetheless may occasionally or even habitually supplement insufficient nocturnal sleep with scheduled naps. In addition, parental report of sleep duration may or may not subtract sleep-onset latency or time awake after sleep onset and thus may more closely approximate “time in bed” rather than actual time asleep. Finally, school start times (see below) have been shown to have a profound impact on sleep duration not only in high school students but also in early adolescents (i.e., middle school) as well (see below).

**Average 24-hour Sleep Duration in Children**

The numbers given below for average sleep duration at different ages represent the best data that are currently available.¹

<table>
<thead>
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<th>Age</th>
<th>Mean (h)/24 h</th>
<th>Range (h)</th>
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<td>9.3-20.0</td>
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<td>13.6</td>
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<td>8.9</td>
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</table>

*Galland et al. (2012)*

**NEWBORNS (0-2 MONTHS)**

**Normal Sleeping Patterns**

- **Hours of sleep:**
  - Total sleep: 9 to 18 hours per 24 hours (average = 14½ hours), may be higher in premature babies.
  - Bottle-fed babies generally sleep for longer periods (2- to 5-hour bouts) than breast-fed babies (1-3 hours).
  - Sleep periods are separated by 1 to 2 hours awake.
No established nocturnal or diurnal pattern in the first few weeks; sleep is evenly distributed throughout the day and night, averaging 8½ hours at night and $5\frac{1}{4}$ hours during the day.

**Developmental Issues and Sleep**

- Sleep-wake cycles are largely dependent on hunger and satiety; circadian rhythms and environmental cues play a much smaller role in newborns than in older infants. Immaturity of the circadian sleep-wake system in the first 10 to 12 weeks of life results in limited rhythmicity and predictability of sleep.

**Common Sleep Issues**

- **Day/night “reversal”:** Day/night reversal is common in the first few weeks; increasing the infant's activity during the day and promoting dim lights at night will help align sleep with nighttime. Studies indicate that newborns often have limited bright light exposure; however, increased natural light exposure, especially in the morning, may be associated with improved sleep at night.

- **Irregular sleep patterns:** Regular rhythm of periods of sleepiness and alertness will emerge by 2 to 3 months.

- **Active versus quiet sleep:** The smiling, grimacing, sucking, snuffling, and body movements such as twitches and jerks that are typical of "active" sleep (equivalent to REM sleep) in infants may be misinterpreted by parents as "restless" or disturbed sleep.

- **Sleeping environment:** Options include a bassinet or crib in a sibling's or baby's own room or a bassinet or crib in the parents' room. Sleeping in the parental bed may pose a risk of accidental suffocation or strangulation, particularly under unsafe sleeping conditions (e.g., maternal smoking, obesity, or use of alcohol or sedating medications; sleeping on a couch or with multiple family members). The American Academy of Pediatrics (AAP) issued a formal recommendation in 2005 advocating against bed-sharing in the first year of life, instead encouraging proximate but separate sleeping surfaces for mother and infant. The infant's bedroom and surrounding environment (e.g., lighting) should be the same at bedtime as it will be throughout the night in order to avoid the development of inappropriate sleep-onset associations.

- **Sleeping surface:** Safety and prevention of accidental suffocation and strangulation are key considerations. Crib mattresses should provide a firm sleeping surface and fit tightly in the crib, and no pillows or comforters should be used. The distance between crib slats should be no greater than 2⅜ inches.

- **Sleeping position:** Empirical studies have clearly demonstrated that sleeping in the prone position ("back to sleep") significantly reduces the risk of sudden infant death syndrome (SIDS).

- **Parental sleep:** Parental sleep also needs to be a priority; sleep deprivation is associated with maternal mood changes and is a risk factor for postpartum depression.

### Safe Sleep Practices for Infants

- Place the baby on his or her back to sleep at night and during naptimes.
- Place the baby on a firm mattress with a well-fitting sheet in a safety-approved crib.
- Do not use pillows or comforters.
- Cribs should not have corner posts over $1\frac{1}{16}$ inch high or decorative cut-outs.
- Make sure the baby's face and head stay uncovered and clear of blankets and other coverings during
**Prevalence of Sleep Problems**

Most sleep issues that are perceived as problematic at this stage represent a discrepancy between parental expectations and developmentally appropriate sleep behaviors. Newborns who are noted by parents to be extremely fussy and persistently difficult to console are more likely to have underlying medical issues, such as colic, gastroesophageal reflux, and formula intolerance.

**Other Important Concepts**

- Parents should be encouraged to learn to recognize both their newborn's intrinsic sleep-wake rhythms and signs of drowsiness (e.g., eye rubbing, irritability, yawning); delaying the opportunity to sleep when these signs occur may result in more difficulty settling.

**INFANTS (2-12 MONTHS)**

**Normal Sleeping Patterns**

- **Hours of sleep:**
  - Total sleep: average is 12 to 13 hours (note that there is great individual variability in sleep times during infancy)
  - Naps: average is 3 to 4 hours
  - **Naps:** Decrease from 4 to 1, with fewer as infants get older, each lasting from 30 minutes to 2 hours. Although most infants move to a longer morning/afternoon nap schedule by 6 months of age, many infants continue to take several short naps of 30- to 45-minute duration until the age of even 9 or 10 months, with naps typically following 1/2 to 2 hours of awake time.

**Developmental Issues and Sleep**

- Issues of attachment and social interaction are felt to play an important role in shaping sleep behaviors. For example, infants who are insecurely attached have been reported to have more sleep problems, although sleep problems do not necessarily imply attachment concerns. On the other hand, studies have shown that infants who are more socially engaged with their caretaker may be more reluctant to interrupt social interactions at bedtime for sleep.

- Acquisition of gross motor developmental milestones (e.g., rolling over, pulling to standing, crawling) and other such events may temporarily disrupt sleep, with one study indicating sleep disruption occurring in the weeks prior to walking.

- As object permanence develops in the second half of the first year, separation anxiety may result in increased
Sleep as a Social Behavior

Sleep behavior in infancy, in particular, must be understood in the context of the relationship and interaction between child and caregiver, which impacts greatly on the quality and quantity of sleep.

Common Sleep Issues

- **Sleep regulation** or “self-soothing” involves the infant's ability to negotiate the sleep-wake transition both at sleep onset and following normal awakenings throughout the night. The capacity to “self-soothe” begins to develop in the first 10 to 12 weeks of life, and is a reflection of both neurodevelopmental maturation and learning.

- **Sleep consolidation** or “sleeping through the night,” although operationally defined by some researchers as a period of sleep without waking from 12:00 a.m. to 5:00 a.m., is typically defined by parents as a continuous sleep episode without the need for parental intervention (e.g., feeding, soothing) from the child's bedtime through the early morning. Infants develop the ability to consolidate sleep between 6 weeks and 3 months, and studies suggest that about 50% to 80% of infants sleep through the night on a regular basis by 9 months.

- **Sleep-onset associations** are those conditions that are present at the time of sleep onset and to which the infant becomes conditioned in order to fall asleep; these may include being rocked or fed or sucking on fingers or a pacifier. These same conditions may be required again during the night in order for the infant to fall back asleep following normal nighttime arousals; “inappropriate” or problematic sleep-onset associations are typically those that require parental intervention (see Chapter 8).

- **Nighttime arousals** are a consequence of the normal ultradian rhythm of 50 (in infants) to 90-to 120-minute (children and adolescents) sleep cycle. Most infants and children arouse briefly on average 2 to 6 times throughout the night. Characterization of these nighttime arousals include the following considerations:

  - **“Self-soothers” versus “non-self-soothers”:** Babies who soothe themselves back to sleep generally experience brief arousals rather than prolonged nightwakings. This capacity to self-soothe is associated with the practice of being put to bed while drowsy but still awake and without sleep-onset associations that will be unavailable during the night. It may be beneficial to suggest to parents that they help their infant develop appropriate sleep-onset associations by 3 to 6 months that will be readily available to the infant during the night without the need for parental intervention (e.g., falling asleep independently in crib, thumbsucking, use of a transitional object).

  - **“Signalers” versus “nonsignalers”:** During nighttime arousals or awakenings, infants may alert (signal) parents by crying or may return to sleep without disrupting parental sleep (nonsignalers). Not surprisingly, signalers are more likely to be labeled by parents as having problematic sleep. It has been estimated that 20% to 30% of 1-year-olds are considered to be signalers.

  - **Parental intervention versus nonintervention:** Parents can choose whether or not to respond, how quickly to respond, and the type of response (e.g., verbal soothing, rocking, or feeding) to a signaled or nonsignaled nighttime arousal or awakening. The practice of parents responding immediately to signaled nightwakings, particularly if the social or feeding interaction becomes reinforcing for the infant, may result in a “trained night crier.”

  - **Night-to-night variability:** It should be noted that there is often considerable night-to-night and week-to-
week variability in all of these behaviors, and neither infants nor parents are always consistent in the way they behave and interact. However, it is clear that a pattern of persistent difficulty in self-soothing is associated with frequent and problematic night-wakings in infants.

- **Transitional objects**, such as a pacifier or a blanket, become important during infancy. Providing the same object at naptime may strengthen attachment to a transitional object. A television set is not a transitional object and should not be in the room where the baby sleeps or play a role in the regular bedtime routine. Some infants respond positively to the use of a mother's well-worn, knotted (to prevent accidental suffocation or strangulation) T-shirt with her scent as a comfort object.

- **Night feedings** do not improve the quality or quantity of sleep and, in most healthy infants, are not physiologically necessary after 6 months of age. If the bedtime transition to sleep onset routinely occurs in the context of feeding (breast or bottle), night feeding may become a necessary component of the infant's returning to sleep following night-wakings. The need for feedings during the night may also become a learned behavior (“learned hunger”), which leads to more frequent and prolonged night-wakings. Frequent night feedings may also disrupt sleep by resulting in bladder distention and discomfort from soaked diapers. Finally, being put down to sleep with a nighttime bottle may increase the risk of dental (“baby bottle”) caries and otitis media. Breast-fed babies are likely to have more nighttime awakenings than bottle-fed babies. In younger infants, this may be related to hunger, but in older infants it is likely related to the development of the habit of feeding to sleep.

Parents should be encouraged to wean their infant from middle-of-the-night feedings by 6 months and to avoid putting him or her to sleep with a bottle.

- **Pacifiers**

Pacifier use during infancy is somewhat controversial. Concerns have been raised regarding its potential negative impact on establishment of breastfeeding, the possible risk of it becoming a prolonged habit, and the need for parental intervention to replace the pacifier during the night in young infants, thus creating an inappropriate sleep-onset association (after the age of 6-8 months, most infants are physically capable of finding a displaced pacifier during the night; some parents scatter several in one corner of the crib to increase this likelihood). Other studies have, however, found no differences across infancy in sleep outcomes (e.g., night-wakings) based on pacifier use. Furthermore, based on a number of epidemiologic studies of SIDS risk and preventive factors, the AAP now recommends the use of pacifiers at bedtime to reduce the risk of SIDS in infants (2005).

**Prevalence of Sleep Problems**

Many sleep “problems” that occur before the age of 6 months are defined as such because of a discrepancy between unrealistic parental expectations in regard to meeting developmental sleep milestones (e.g., “sleeping through the night,” settling at bedtime) and normal maturation of the infant; thus, these “problems” are likely to be transient in nature. Others are the result of a mismatch between infant behavior and parental expectation. Temporary sleep disturbances that occur in conjunction with developmental milestones, as well as acute illnesses, also tend to be self-limited if parents avoid engaging in behaviors that reinforce difficulty settling or night-wakings, such as resorting to rocking to sleep. However, not all infant sleep problems are transient, and certain factors appear to be associated with an increased risk of chronicity. These include inability to self-soothe, difficult temperament, maternal depression, and bed-sharing.

It is estimated that about 25% to 50% of 6- to 12-month-olds and 30% of 1-year-olds have problematic
nightwakings and about 50% have sleep-onset or settling difficulties at 12 months.

Common Sleep Disorders

- Nightwakings (see Chapter 8)
- Sleep related rhythmic movements (head banging, body rocking, body rolling) commonly present in the first year of life (see Chapter 12)

Other Important Concepts

- Most infants benefit from a set sleep schedule with regular naptimes and bedtime that reflects the infant's natural preferences for sleep and waking patterns as well as family lifestyle issues.
- At the 2-month visit, parents should be encouraged to start putting the infant to bed drowsy but awake within the next 4 to 8 weeks.
- A consistent and enjoyable bedtime routine leading up to sleep onset should be established by the age of 3 months. Studies indicate that a bedtime routine results in reduced sleep-onset difficulties and nightwakings. The bedtime routine may include any of the following: feeding, bath, story, music, massage, and rocking.
- Parents should be encouraged to take the opportunity to nap or rest when the infant is sleeping.
- It is important to discuss the possibility with parents that their previously “great” sleeper may develop transitory problems with nightwakings and bedtime resistance during times of stress or in association with achievement of developmental milestones. Avoidance of parental reinforcement (e.g., attention, night feedings) often prevents a small, temporary sleep problem from becoming a large and chronic one.

To Co-sleep or Not to Co-sleep

Co-sleeping, which is broadly defined as room-sharing and bed-sharing typically with siblings or adult caregivers, is a topic that has generated a considerable amount of controversy and discussion among pediatric practitioners, parent advocacy organizations, and the general public. The issues that have been raised in defending or decrying the practice of co-sleeping are myriad, ranging from the effects on the health of the co-sleeping infant (the AAP recommends against the practice in the first year of life to prevent accidental suffocation, while breastfeeding advocates cite the association between co-sleeping and reinforcement of nursing practices) to both positive and negative psychosocial and developmental effects (individuation versus intimacy, sexual attitudes, attachment) to sociologic and anthropologic considerations as viewed in a family context (cultural and ethnic values).

The decision to co-sleep is made by families for a variety of reasons, which are important to explore in addressing this issue in the healthcare setting. It is important to differentiate between “lifestyle” or “cultural” co-sleeping, in which caregivers make a conscious decision to have a “family bed,” and “reactive” co-sleeping, which is typically initiated by caregivers in response to the child's sleep difficulties (e.g., bedtime resistance or nightwakings). Potential safety concerns and other issues, such as parental sexual activity or the birth of a new sibling, are appropriate to discuss with caregivers in the former situation, whereas working with parents to develop workable solutions for sleep problems that do not involve bed-sharing is more likely to be helpful for the latter circumstance.

Parents should be encouraged to share their practices and feelings; all too often, parents are reluctant to be honest about the practice of co-sleeping for fear of disapproval from the medical profession. It may be more useful to consider the strengths and vulnerabilities of the individual child, parent, and parent-child
dyad in assessing the potential risks of co-sleeping in a given situation rather than adopting a “one-size-fits-all” approach. Counseling parents on this issue requires a sensitive, open, and nondogmatic approach, the goal of which is to more effectively guide parents in making informed and safe choices that are in their child’s and family’s best interest.

TODDLERS (12 MONTHS TO 3 YEARS)

Normal Sleep Patterns

- **Hours of sleep:**
  - Total sleep: average is 11 to 13 hours (note that there is individual variability in sleep times for toddlers)
  - Naps: average is 2 to 3 hours

- **Naps** decrease from two naps to one at an average age of 18 months. Overall, almost 100% of 1-year-olds and 81% of 2-year-olds take a nap, decreasing to just over half of 3-year-olds.

- **Circadian preference or “chronotype”** is a construct reflecting individual differences in relative “morningness” versus “eveningness” with respect to wake time, sleep onset, and periods of alertness and sleepiness throughout the day. Experimental studies have now demonstrated that sleep-wake timing in toddlers parallels biological markers of circadian phase (i.e., melatonin secretion), as it does in adolescents and adults. While there are large intraindividual differences, empirical evidence now supports the clinical observation that toddlers typically display a morningness circadian preference.

Developmental Issues and Sleep

**Gross Motor**

- Independent locomotion and increased mobility makes it easier for the child to engage in limit-testing behaviors (e.g., getting out of bed, coming into the parents’ bedroom) at bedtime and during the night.

**Cognitive**

- Drive to learn and rapid acquisition of skills may result in difficulty settling at bedtime.
- Comprehension of cause and effect enables the child to respond to simple behavioral interventions.
- Understanding of the symbolic meaning of objects leads to increased interest in and reliance on transitional objects.
- Development of imagination and fantasy may result in increased nighttime fears.

**Language**

- Expressive language development lags behind receptive, which may limit verbal expression of nighttime fears and concerns.

**Social/Emotional**

- Links between sleep and the development of self-regulation processes (e.g., the ability to control one’s behavior, attention, and emotions when challenged) in early childhood have been proposed. Clinical observation and experimental studies suggest that after losing daytime sleep, toddlers are less able to engage
effectively in a difficult task and revert to lessmature self-regulation strategies compared to the well-rested state. Sleep restriction also appears to result in a reduction in positive emotion responses, as well as an increase in negative emotional responses. Over time, chronic sleep loss may impair young children's self-regulation abilities, resulting in an increased risk for social-emotional, behavioral, and school problems.

- The emerging drive for autonomy and independence may lead to increased bedtime resistance.
- Separation anxiety is often associated with bedtime difficulties and increased nightwakings.
- Regression in behavior is a common stress response and may increase the likelihood of co-sleeping.

Common Sleep Issues

- **Naps:** There are many components that can raise concerns with naps, including number, duration, timing, location, giving up, and need for naps. Restricting naps will not help a child sleep better at night; however, placement of naps too close to the scheduled bedtime may interfere with sleep onset.

- **Transition from crib to bed:** This step is usually taken between 2 and 3 years of age, or when safety concerns related to falling while climbing out of the crib become an issue. In the latter situation, a mesh crib tent is recommended to alleviate safety concerns while maintaining the child in a crib. The transition to a bed at too young an age can often result in the development of sleep problems because young children often do not have the cognitive development or behavioral control to stay within the essentially imaginary boundaries of a bed. Waiting until closer to age 3 to make the change is recommended. Similar to recommendations regarding premature attempts at toilet training before the child is developmentally ready, parents should be encouraged to temporarily switch back to a crib if the transition does not go smoothly, and to wait and make the transition at a later age.

- **Bedtime bottles and night feedings:** As noted in the section on infants, the bedtime transition to sleep onset ideally occurs without a feeding in order to facilitate independent settling. The same issues as described in infants in terms of learned hunger, arousals related to bladder distention, dental caries, and increased risk of otitis media associated with nighttime bottles apply to toddlers as well.

- **Bedtime routines:** These are an important daily ritual for parents to develop and maintain in order to help ease the transition from high levels of activity during the day to sleep onset. The events of the bedtime routine should be consistent, occur in the same temporal order, and be of an increasingly relaxing nature for that particular child as bedtime nears. As with infants, a study found that the institution of a consistent bedtime routine in toddlers reduced latency to sleep onset and improved nighttime sleep.

- **Transitional objects:** Introducing these, such as blankets, dolls, and stuffed animals, at bedtime and naptime becomes increasingly important at this stage and helps to foster independent settling and self-soothing. The practice of thumbsucking and use of pacifiers, especially if limited to nighttime, is unlikely to result in significant orthodontic problems before age 4 years, and should not be discouraged. Thumbsucking is found to result in better sleep outcomes, with no differences in sleep between infants who use pacifiers and infants who do not engage in any nonnutritive sucking.

Prevalence of Sleep Problems

Sleep problems in toddlers are very common, occurring in 25% to 30% of this age group. Bedtime resistance and stalling at bedtime, found in 25% to 30% of toddlers, and nightwakings in up to 50%, are the two most common concerns of parents, followed by nighttime fears and nightmares. Sleep problems may also be persistent, especially in children with daytime behavior problems. Conversely, daytime behavior problems are more common in poor sleepers (45%).
Common Sleep Disorders
- Bedtime problems (see Chapter 7)
- Nightwakings (see Chapter 8)
- Sleep related rhythmic movements (head banging, body rocking, body rolling) may continue into the preschool years (see Chapter 12)

Other Important Concepts
- Secondary prevention involves avoiding reinforcement of transitory sleep problems and preventing a sleep “disturbance” from becoming a sleep “disorder.”
- Sleep disturbances are often manifested in daytime behavior as a result of the bidirectional effects of sleep on behavior.
- It should be noted that in children with nightwaking problems, there may be a “golden” window of opportunity to initiate a behavioral intervention, such as a graduated extinction program (see Chapter 8) before the child is transitioned to a bed and is, thus, easily able to come into the parents' bedroom during the night.

Cultural and Family Context of Sleep
It is important to always consider the cultural and family context within which sleep problems in children occur. How we sleep, where we sleep, with whom, and for how long we sleep are all molded by culture and customs. For example, co-sleeping of infants and parents is a common and accepted practice in many ethnic groups, including African American, Hispanic, and Asian families. Therefore, the goal of independent self-soothing in young infants may not be shared by these families. The relative importance of sleep as a health behavior, “normal” sleep practices such as napping patterns and bedtime rituals, the sleeping environment, and the relative acceptability of various treatment strategies for sleep problems (e.g., the “cry-it-out” approach) are just a few additional examples of sleep issues that are influenced by cultural and family values and practices. Furthermore, not only is the range of sleep behaviors that may be considered “normal” or “pathologic” wide, but the very definition of what constitutes a “problem” is often quite subjective and highly dependent on culturally determined factors. These factors include parents' awareness of, expectations for, and tolerance of sleep behaviors and the perceived societal impact of sleep problems and insufficient sleep on children's health, behavior, and learning. Figure 2.1 summarizes the myriad influences that help to determine “optimal” sleep in children.

PRESCHOOL-AGED CHILDREN (3-5 YEARS)
Normal Sleep Patterns
- Hours of sleep:
  - Total sleep: average is 11.5 to 12 hours
  - Longitudinal epidemiologic studies suggest that there may be distinct identifiable sleep duration patterns in young children (e.g., typical sleepers, initially short sleepers, poor sleepers, and persistent short sleepers), and that these sleep “trajectories” may have different implications for physical, emotional, and social health.
Naps decrease from 1 nap to no nap. Overall, 26% of 4-year-olds and just 15% of 5-year-olds nap. However, one study found that African American children were more likely to continue to take daytime naps until a later age compared to Caucasian children, with no difference in total sleep time across the groups (i.e., the Caucasian children had earlier bedtimes and slept longer at night). The transition to kindergarten, particularly in those children who have not attended preschool, may be associated with a decrease in total weekday sleep time, largely due to loss of a nap opportunity.

Sleep-onset latency: Somewhat surprisingly, the average time to fall asleep after lights out seems fairly constant from preschool age to 12 years at around 17 to 19 minutes. However, it should be noted that this sleep parameter is highly subjective, particularly when estimated by caregivers, and studies suggest that parents tend to overestimate sleep-onset latency compared to objective (e.g., PSG) measures.

Developmental Issues and Sleep

- Expanded language and cognitive skills may lead to increased bedtime resistance, as children become more articulate about their needs and may engage in more limit-testing behavior.

![Diagram showing factors affecting sleep](image_url)

**Fig 2.1.** Factors that play a role in determining “optimal” sleep in children.

- Daytime naps may be instrumental in cognitive processes (e.g., certain types of memory consolidation).
- A developing capacity to delay gratification and anticipate consequences enables preschoolers to respond to positive reinforcement (e.g., sticker charts) for appropriate bedtime behavior.
- Further development of imagination and fantasy may heighten nighttime fears.
- Increasing interest in developing literacy skills reinforces the importance of reading aloud at bedtime as an integral part of the bedtime routine.
Temperament has an important influence on sleep behaviors; young children with “difficult” temperaments have an increased likelihood of sleep problems. However, this relationship is hardly straightforward. For example, consistent and positive sleep practices are associated with less bedtime resistance even in children with more challenging temperaments.

Common Sleep Issues

- **Co-sleeping and parent presence**: Persistent co-sleeping tends to be highly associated with sleep problems in this age group. Estimates suggest almost 50% of preschoolers have a parent present when falling asleep at bedtime, which may result in difficulty sleeping independently and prolonged nightwakings.

- **Sleep schedules**: Preschoolers need a set, consistent bedtime and waketime. A regular and consistent daytime routine (e.g., meals, playtimes) also helps to regularize the sleep-wake schedule. Children who are not in morning school or daycare settings may do fine with a relatively later bedtime and waketime but may need to readjust their schedule once school starts. Parents should be encouraged to start this gradual process several weeks in advance of the anticipated change in morning schedule.

- **Appropriate bedtime**: In setting a bedtime, some consideration should be given to the child’s circadian preference. Studies suggest that a “dissonance” between parent-selected bedtimes and children’s circadian physiology may contribute to the development of bedtime resistance in young children. On the other hand, a bedtime later than 9:00 p.m. has been shown to be associated with shorter sleep duration.

- **Second wind or “forbidden zone”**: The normal evening circadian-mediated surge in alertness and activity levels that occurs in everyone may have an exaggerated behavioral component in some children; it also may occur relatively later in children who are natural “night owls.” Thus, if (attempted) bedtime coincides with the timing of this circadian drive towards wakefulness, the likely result is bedtime resistance. Temporarily delaying the bedtime to coincide with the preferred sleep-onset time and then moving it gradually earlier often successfully addresses the problem of a child being consistently wide awake at bedtime.

Prevalence of Sleep Problems

Difficulties falling asleep and nightwakings are still common in this age group (15%-30%), in many cases coexisting in the same child (average time to fall asleep in this age group is around 15 minutes). A number of studies have suggested that sleep problems in this age group may become chronic; in one study, 84% of children aged 15 months to 48 months with bedtime struggles and/or nightwakings continued to have significant sleep disturbance at their 3-year follow-up.

Common Sleep Disorders

- Nighttime fears and nightmares (see Chapters 9 and 10)
- Bedtime problems (see Chapter 7)
- Nightwakings (see Chapter 8)
- Obstructive sleep apnea and sleep-disordered breathing (see Chapter 15)
- Disorders of arousal (sleepwalking, sleep terrors) (see Chapter 11)

Other Important Concepts

- The association between insufficient or disturbed sleep and behavioral problems tends to become more evident at this age.
Clinicians should be aware of and sensitive to the potential impact of “suboptimal sleep environments” on sleep-onset latency, nightwakings, and sleep duration, especially in low-income and minority children.

Increasingly, younger children are exposed to electronic media as part of the bedtime routine and to the presence of these devices in the bedroom (see below). Studies suggest that there is a bidirectional relationship between media use and sleep duration even in young children, with more media use predicting shorter sleep and vice versa.

Parents should be encouraged to make reading an integral part of the bedtime routine. In one study, children for whom reading was part of their bedtime routine obtained more total sleep and had fewer sleep disturbances.

Sleep health educational interventions in early childhood have been shown to have a positive impact on parents’ sleep knowledge, attitudes, and self-efficacy, as well as on children's sleep behavior.

**The Electronic Sandman**

Television viewing and electronic use at bedtime, as well as the presence of digital devices in a child's bedroom in particular, have been found to be associated with difficulties in falling and staying asleep in children, as well as reduced total sleep times. Digital device use may also play a role in increasing the likelihood of nightmares and nighttime anxiety. “Screens” (desk and laptop computers, handheld devices, electronic gaming systems, smartphones) not only provide cognitive, and sometimes psychological and physical stimulation, that interfere with the relaxed state required for sleep initiation, but may also provide enough light exposure to suppress the normal evening surge in melatonin and thus further delay sleep onset. Parents should be strongly encouraged to keep electronic devices out of (or remove them from) the child's sleeping environment.

**SCHOOL-AGED CHILDREN (6-12 YEARS)**

**Normal Sleeping Patterns**

**Hours of sleep:** Average is 9 to 10 hours over 24 hours

**Wake-up Call**

Because school-aged children are normally highly alert, behaviors such as napping, dozing off during short car rides or while watching TV, and persistent complaints or behavioral manifestation of daytime sleepiness reported by parents or teachers in school-aged children are a red flag and should be taken seriously. These symptoms suggest significant problems with sleep quality and quantity or, less frequently, a primary disorder of excessive daytime sleepiness, and require prompt evaluation by the primary care provider for the root cause(s).

**Developmental Issues and Sleep**

**Cognitive**

- Comprehension of existence of real dangers (e.g., burglars) may increase nighttime fears.

**Social/Emotional**
Increasing independence from parental supervision and a shift in responsibility for health habits as children approach adolescence may result in less enforcement of appropriate bedtimes and inadequate sleep duration; parents may also be less aware of sleep problems if they do exist.

Involvement in academic, social, athletic, and family activities as well as parent work schedules may conflict with time for sleep.

Increasing reliance on peer relationships can compete for sleep time.

Social anxiety and need for academic achievement may result in nighttime worrying, interfering with sleep onset.

Media and electronics, such as television, computer, video games, and the Internet, all compete increasingly for sleep time (see Chapter 5). Even the presence of “small screens” (e.g., “smartphones”) in the bedroom environment can adversely impact sleep duration and perception of sufficient sleep in school-aged children.

Healthy Sleep/Healthy Child
Middle childhood is a critical time for the development of health habits in general and healthy sleep habits in particular. Practitioners have a real opportunity in the context of well-child care to introduce concepts of sleep health promotion (good sleep habits) and disease prevention (impact of insufficient sleep) directly to children at this stage, and studies suggest that middle school-aged children may be particularly receptive to counseling and education about sleep from their primary care provider.

Common Sleep Issues
- **Irregularity of sleep-wake schedules** reflects increasing discrepancy between school-night and nonschool-night bedtimes and waketimes.
- **Later bedtimes** have been shown to be consistently linked to shorter, and frequently insufficient, sleep amounts in school-aged children. Several studies have suggested that lower-income and minority children, especially boys, are at increased risk for both later bedtimes and insufficient sleep.
- **Decreased nighttime sleep** has been shown to be associated with impairments in daytime functioning, including school performance. Several studies have found that restricting sleep in school-aged children by just 30 to 60 minutes for several nights leads to daytime behavior problems, including inattention and other cognitive effects. Sleep efficiency (that is, time asleep/time in bed) may also play a role in academic performance. Insufficient sleep has also been linked on both cross-sectional and longitudinal studies to the development of obesity in children (see Chapter 21).
- **Increased caffeine intake** may interfere with sleep onset and quality of sleep. Approximately 40% of school-aged children have a daily caffeinated beverage, which is associated with less total sleep time.
- **Parental presence at bedtime** is still quite common with school-aged children, occurring in about 30% of families. The requirement to have a caregiver present at sleep onset is associated with a six-fold increase in nighttime awakenings.
- **Daytime sleepiness** is rare in this age group; therefore, parents should be questioned about and encouraged to report any evidence of recurrent daytime sleepiness (e.g., napping, dozing off in school).

Prevalence of Sleep Problems
Although conventional wisdom has previously indicated that sleep problems are less common in middle childhood, more recent studies have reported an overall prevalence of significant parent-reported sleep problems
of 37% in this age group, with a 15% to 25% prevalence of bedtime resistance, a 10% prevalence of significant sleep-onset delay and anxiety at bedtime, and a 10% prevalence of teacher-reported and parent-reported daytime sleepiness. Another study found that almost one-third of school-aged children report difficulty waking in the morning and 10% feel tired during the day. It should also be noted that these figures might underestimate the magnitude of sleep problems because parents may be unaware of and, thus, underreport sleep concerns at this age.

### Common Sleep Disorders

- Sleepwalking and sleep terrors (see Chapter 11)
- Bruxism (see Chapter 13)
- Sleep enuresis (see Chapter 14)
- Obstructive sleep apnea and sleep-disordered breathing (see Chapter 15)
- Insufficient sleep (see Chapter 5)
- Unhealthy sleep habits (see Chapter 5)
- Restless legs syndrome/periodic limb-movement disorder (see Chapter 16)

### Other Important Concepts

- **Maturational issues:** Many of the developmental sleep changes associated with puberty described in the following section on adolescent sleep may be applicable to the early-maturing middle school-aged child.

  - **Circadian preference:** Children with an “eveningness” chronotype (e.g., prefer a later bedtime and rise time) may display sleep-onset problems and associated bedtime resistance when given a bedtime that is significantly earlier than their preferred sleep-onset time but fall asleep easily and quickly when given a later bedtime. Allowing a child to sleep on a *self-selected* sleep schedule for several days (e.g., during vacation) may help to clarify the issue of circadian preference. In addition to impacting sleep-wake schedules, mounting evidence suggests that chronotype (i.e., a strong circadian “eveningness” preference) may be independently associated with a number of adverse health, behavior, and performance outcomes, such as lower physical activity, depression, impulsivity, aggressive and antisocial behavior, substance and alcohol use, lower academic achievement, and increased perceived stress level.

- **Sociocultural factors:** A number of studies have indicated that sleep health disparities exist and that children and adolescents from families with low income or of racial or ethnic minorities may be at even greater risk of poor-quality and insufficient sleep. For example, a number of studies of school-aged children have reported an association between late bedtimes, unhealthy sleep practices, and short and/or disrupted sleep with socioeconomic and minority status.

  Encouragement of healthy sleep habits and an emphasis on the importance of sufficient sleep by parents, teachers, and healthcare practitioners is particularly important in the middle childhood years and may set the stage for development of continued positive sleep behaviors in adolescence and adulthood.

### Owls and Larks

Parents and providers should be sensitive to circadian preference, particularly for a delayed (later) sleep phase as a possible cause of bedtime struggles. Sleep schedules for these children may need to be adjusted accordingly to reduce sleep-onset delay while preserving adequate sleep amounts.
Clinical Practice Recommendations: Sleep Need

Screen

- Pediatric providers should incorporate screening questions regarding typical bedtimes, wake times, and sleep amounts on both school days and nonschool days in children and adolescents as part of the well-child health encounter. As parents are often unaware of the extent of sleep loss, adolescents should be directly queried about their sleep patterns.

- Pediatric providers should routinely inquire about potential signs of insufficient sleep in their adolescent patients, particularly in those adolescents getting less than the recommended 9 hours of sleep per night. Signs include dozing off under both low- (watching TV, passenger in a car) and high-stimulation (in school, driving a car) situations, poor concentration, irritability, and a decline in academic performance.

Educate

- Education regarding sleep needs should be included in anticipatory guidance for school-aged children and adolescents.

- Key health education messages for caregivers and patients include the following:
  - Studies have clearly demonstrated that adolescents require between 8.5 and 9.5 hours of sleep per night in order to function optimally. Middle school students typically require 9 to 10 hours of sleep.
  - Well-documented health risks associated with chronic insufficient sleep include weight gain and obesity, mood dysregulation and depression, metabolic dysfunction and impaired glucose metabolism, and increased risk of motor vehicle accidents. Cognitive function, attention, and memory, particularly on complex tasks and those involving an emotional component, are selectively impaired by sleep loss.
  - Significantly delayed weekend bedtimes, coupled with “sleeping in” on nonschool days in an attempt to compensate for insufficient school day sleep, does not adequately address the effects of sleep loss. This common adolescent practice may also further exacerbate the adolescent circadian-based sleep-phase delay, resulting in “jet lag” symptoms.
  - Basic principles of healthy sleep practices provide important guidelines for achieving sufficient sleep in adolescents.

ADOLESCENTS (12-18 YEARS)

Normal Sleeping Patterns

- **Hours of sleep:** Consensus findings across epidemiologic studies strongly suggest that both younger and older adolescents are not meeting recommended sleep amounts. Furthermore, US-based studies have revealed that as age increases, sleep duration declines. The NSF's Sleep in America Poll found that by the 12th grade, 75% of students self-reported sleep durations of less than 8 hours of sleep per night compared with 16% of sixth graders, and only 3% of 12th graders reported getting the recommended 9 or more hours of sleep. Average sleep times even on nonschool nights is only 9 hours; thus the attempt on weekends to compensate still falls short of even normal daily sleep requirements. Compounding the situation, the majority of parents polled (70%) think their teens are getting adequate sleep and thus may not be encouraging obtainment of sufficient sleep.

As noted above, the accumulated “sleep debt” on weekdays is typically accommodated by “weekend
oversleep” (the difference between weekday and weekend sleep durations) with up to 2 or more hours commonly reported. Moreover, early school start times require teens to wake and function at a time when circadian-mediated levels of alertness are at their 24-hour nadir. Thus, the sleep deficits typically experienced by adolescents represent a combination of circadian misalignment and chronic sleep loss, resulting in a host of short and potential longterm effects on physical and mental health, safety, and performance.

- **Bedtime:** Bedtimes become steadily later on school nights, from an average of 9:30 p.m. in sixth grade to 11:00 p.m. in 12th grade, with average waketimes consistent at approximately 6:30 a.m. Bedtimes on nonschool nights also steadily become later with age, with an average of 10:30 p.m. in sixth graders to 12:45 a.m. in 12th graders.

### Developmental Issues and Sleep

- **Circadian factors:** Typically around the time of puberty onset, adolescents develop an approximately 2-hour physiologically based phase delay (later sleep-onset and wake times), relative to sleep-wake cycles in middle childhood. This is a result of both pubertal and hormonal and environmental influences on circadian sleep-wake cycles, and appears to be linked to chronological age as well as Tanner stage. This shift to a later sleep-wake cycle is accompanied by a similar delay in timing of other circadian “clock-driven” events; for example, the “second wind” phenomenon of increased alertness just before sleep onset is shifted to a later time in the evening, and the nocturnal circadian wake-drive “trough” (3:00-5:00 a.m.) is also shifted, often coinciding with the rise time required by early school start schedules. In addition, some researchers have suggested that adolescents are relatively more sensitive to the melatonin-suppressing effects of evening-light exposure, further delaying sleep onset. Another sleep development in adolescence that relates to circadian timing is the increasingly large discrepancies between bedtimes and waketimes on school nights versus nonschool nights. Sleep onset and morning (or afternoon) waking times may differ by 3 to 4 hours or more, creating an eastward-direction “jet lag-like” situation by the end of the weekend (or vacation). In other words, on Sunday evening, the adolescent is attempting to shift sleep-onset time earlier by 3 to 4 hours, an almost impossible task for even the most seasoned traveler!

- **Sleep drive:** Studies suggest that adolescents may “accumulate” their sleep drive more slowly during periods of prolonged wakefulness compared to younger children. This results in an increased ability to voluntarily delay sleep onset and more difficulty in falling asleep at an earlier time. Maturational changes to these two bioregulatory processes beginning in adolescence present a major challenge to the ability of adolescents to fall asleep much before 11:00 p.m.

- **Sleep needs:** The rate of dissipation of the sleep drive during the night, and thus the amount of sleep required to restore normal alertness levels, does not change significantly in adolescence. Therefore, adolescent sleep needs do not differ dramatically from sleep needs of preadolescents. Optimal sleep amounts in teens remain at about 8.5 to 9.5 hours per night. Given that the majority of adolescents are only obtaining 7 hours on average, this situation has created a whole generation of profoundly sleepy individuals.

### Common Sleep Issues

- **Environmental and lifestyle factors:** Environmental factors and lifestyle and social demands, such as homework, afterschool jobs, academic pressure, participation in sports and other extracurricular activities, socializing, and lack of parental monitoring or even awareness of the adolescent’s sleep schedule impact significantly on sleep in adolescents, frequently resulting in delayed sleep onset.
The Burden of Chronic Sleep Loss in Teens

- **Cognitive and behavioral consequences** such as poor judgment, lack of motivation, and inattention and affective dysregulation resulting from sleep loss, as well as the effect of insufficient sleep on decision-making skills further compound the potential negative effects in adolescents.

- **Influence on mood** and the development of depressive symptoms in adolescents. Importantly, from a clinical standpoint, improvements in sleep may lead to improvements in mental health functioning (and vice versa). The association between sleep loss and increased suicidality in adolescents is particularly worrisome and is clearly important in the clinical setting.

- **Increased overweight/obesity risk** in children and adolescents; the body of evidence from studies assessing the relationship between short sleep and obesity is both compelling and potentially far-reaching in its public health implications. More research is urgently needed to identify specific metabolic, inflammatory, and hormonal mechanisms, as well as the interactions among sleepiness and activity levels, mood, cognition, and behavioral responses, in this complex equation.

- **Drowsy driving accidents**: At particular risk are young inexperienced drivers. A substantial percentage of young drivers admit to driving while sleep-impaired, and short sleep duration in this population is clearly linked to an increase in automobile crashes and “near-misses.”

- **Sleep-wake variability**: There is significant variability in sleep-wake patterns in adolescents from weekday to weekend, resulting in further circadian misalignment.

- **Early high school start times**: The early start times of many high schools, as well as some middle and junior high schools, often require that students wake up not only before they have had sufficient sleep, but also at the time of their lowest daily level of alertness. Not only do schools with earlier start times report significant negative consequences, such as higher rates of tardiness and decreased concentration in their students, but also research has documented positive outcomes in school districts that have delayed their school times. These benefits have included a positive impact on academic factors such as increased attendance, decreased tardiness, better grades, improvements in standardized test scores, improvements in measures of mental health (self-reported depression symptoms), and improved safety (e.g., associated decrease in teen driving accidents). Of note, students attending schools that have adopted delayed start times report increased sleep amounts; in other words, these students go to bed at the same time (i.e., do not stay up later) but, on average, sleep in an additional hour per day.

**Early Middle/High School Start Times: A Key Modifiable Factor in Teen Sleep Loss**

The AAP recognizes insufficient sleep in adolescents as an important public health issue that significantly affects the health and safety, as well as the academic success, of our nation’s middle and high school students. Although a number of factors, including biological changes in sleep associated with puberty, lifestyle choices, and academic demands, negatively affect middle and high school students’ ability to obtain sufficient sleep, the evidence strongly implicates earlier school start times (i.e., before 8:30 a.m.) as a key modifiable contributor to insufficient sleep, as well as circadian rhythm disruption, in this population. Furthermore, a substantial body of research has now demonstrated that delaying school start times is an effective countermeasure to chronic sleep loss and has a wide range of potential benefits to students with regard to physical and mental health, safety, and academic achievement. The AAP strongly supports the efforts of school districts to optimize sleep in students and urges high schools and middle schools to aim for start times that provide students the opportunity to achieve optimal levels of sleep (8.5-9.5 hours) and to
improve physical (e.g., reduced obesity risk) and mental (e.g., lower rates of depression) health, safety (e.g., drowsy driving crashes), academic performance, and quality of life.

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- **Significant daytime sleepiness:** All of the above factors often combine to produce significant sleepiness in many adolescents and consequent impairment in mood, attention, memory, behavioral control, and academic performance. Overall, in the NSF poll noted earlier, more than half (56%) of adolescents said they get less sleep than they think they need to feel their best, and 51% said they feel too tired or sleepy during the day. Over 25% of adolescents reported falling asleep in school at least once a week, and more than 1 in 5 fell asleep while doing homework. Perhaps most alarmingly, over one-half of adolescent drivers reported having driven while drowsy in the past year and 15% drove while sleepy at least once a week. Therefore, parents of teenagers should be strongly encouraged to monitor their adolescents’ sleep and possible negative consequences of sleep deprivation and to actively intervene if sleep is chronically insufficient.

- **Risk-taking behavior:** As with all adolescent risk-taking behavior, parents should be encouraged to maintain a level of open communication with their teenagers regarding the impact of insufficient sleep and poor sleep habits on social and emotional functioning, school performance, and health. Studies indicate that insufficient sleep is associated with other health-risk behaviors, such as substance use, binge drinking, poor nutrition, and decreased physical activity.

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**Sleep and School**

Many high-achieving college-bound high school students state that they “have to” stay up late in order to complete homework assignments, participate in extracurricular and athletic activities, and get involved in community service projects as well as the myriad of other activities that “look good” on college applications. While there is clearly a great deal of very real pressure on adolescents to perform and succeed, this reasoning has some fundamental flaws. Most studies, in fact, have shown that students who get better grades tend to sleep more, not less, and that standardized test scores are inversely correlated with sleep duration. Furthermore, sleep-deprived individuals are less efficient in completing cognitive tasks, thus establishing a cycle in which the student takes longer to complete the same amount of work, necessitating staying up later, perpetuating the cycle. Thus, helping high school students to understand the repercussions of insufficient sleep and to prioritize tasks to allow for adequate sleep opportunity are important components of anticipatory guidance in adolescence.

**Sleepy, Dopey, and Grumpy**

There is now substantial evidence to suggest that, as a group, adolescents in the United States are chronically sleep-deprived. Studies suggest that many teens function for a good part of the day in the “twilight zone,” which is a level of sleepiness equivalent to that in individuals with narcolepsy! Practitioners should be aware of this epidemic of insufficient sleep and carefully assess adolescent patients for chronic sleepiness and the accompanying effects on mood, behavior, and school performance.

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**Prevalence of Sleep Problems**

Data suggest that chronic partial sleep deprivation is a serious problem in adolescents and that particular group, including “high achievers” who are engaged in many extracurricular activities, may be at a relatively higher risk. Chronic sleep restriction in adolescents can lead to significant neurobehavioral consequences, including a negative impact on mood, vigilance and reaction time, attention, memory, behavioral control, and motivation.
Adolescents may also suffer from a number of sleep disorders. A number of studies have suggested that the prevalence of significant sleep problems in adolescents is high (at least 20%) and that particular group of adolescents, such as those with chronic medical or psychiatric problems (e.g., depression), may be at increased risk.

**Common Sleep Disorders**

- Insufficient sleep (see Chapter 5)
- Unhealthy sleep practices (see Chapter 5)
- Insomnia (see Chapter 19)
- Delayed sleep-wake phase disorder (see Chapter 18)
- Obstructive sleep apnea and sleep-disordered breathing (see Chapter 15)
- Restless legs syndrome/periodic limb-movement disorder (see Chapter 16)
- Narcolepsy (see Chapter 17)

**Asleep at the Wheel**

Older adolescent boys are among the highest risk group for drowsy driving-related accidents because of a combination of risk factors (decreased sleep amounts and increased sleepiness, driving inexperience, greater willingness to engage in risk-taking behavior, combined with drugs, marijuana, and/or alcohol). However, all adolescents with insufficient sleep are at risk. Many teens assume that ingesting caffeinated beverages (e.g., Red Bull) will counteract the effects of both sleepiness and alcohol, unfortunately sometimes with fatal results. In fact, studies have shown that individuals who consume alcohol plus “energy drinks” show equivalent levels of objective impairment (i.e., decreased vigilance on computerized tests) compared to alcohol alone, but rate themselves as more alert and less impaired. The most common drowsy driving accident scenario involves a single motor vehicle and driver who drifts off the highway late at night. Many sleepiness-related accidents are fatal, because the driver (who is asleep) makes no effort to avoid the crash.

**Clinical Practice Recommendations: Drowsy Driving**

**Screen**

- Pediatric providers should incorporate screening questions regarding previous drowsy driving incidents (fall-asleep accidents or near-accidents) in all adolescent drivers. Screening questions should also address situations in which a combination of insufficient sleep and alcohol and/or substance use may have been the contributors to driving crashes. Adolescents should be asked if they recognize the warning signs of drowsiness (i.e., head-nodding, eyes closing, dozing at stop lights, not remembering driving the last few miles/minutes) and how often they have driven when drowsy. Adolescent drivers and parents should also be queried about household rules regarding sleep duration and “fitness for driving.”

**Educate**

- Parents and teens should be educated about the risk factors and consequences of drowsy driving. Drawing an analogy between drunk driving, a situation which the vast majority of parents and
adolescents understand and avoid, and driving “under the influence” of insufficient sleep is often helpful in raising awareness in families regarding the level of impairment experienced by drowsy drivers (i.e., equivalent to moderate levels of intoxication or consumption of 3-4 alcoholic beverages).

- It may also be helpful to explain the concept of “microsleeps”; i.e., chronic sleep loss at the level experienced by most teens results in brief periods (several seconds) in which the sleep drive temporarily overrides voluntary control of wakefulness and the brain simply falls asleep. If this occurs while driving, the result can be fatal.

- Key health education messages for caregivers and patients include the following:
  - Adolescents should not be allowed to get behind the wheel if they are experiencing chronic sleep loss (<7 hours/night) and are impaired by sleepiness; i.e., “no ZZZs, no keys.”
  - Typically employed sleepiness countermeasures (e.g., rolling down the car window, turning up the radio, chewing gum) have been shown to be ineffective in reducing drowsy driving risk.
  - Caffeine consumption is not a substitute for sufficient sleep and should not be considered an effective countermeasure for drowsy driving in teens.
  - Individuals particularly high risk for drowsy driving crashes are young (18-25-year-olds), male, inexperienced drivers. Situations that pose a high risk in drowsy drivers include driving long distances (especially without breaks), driving alone, driving at night, and combining sleep loss and alcohol/substance use. However, accidents can occur in the absence of any of these conditions as a consequence of drowsiness alone.
  - Drowsy driving crashes are more likely to be fatal, as the driver (who is asleep) makes little or no effort to avoid the crash.
Evaluation of Pediatric Sleep Disorders

As is the case with other medical, neurodevelopmental, and psychiatric disorders, a key issue in the evaluation and management of pediatric sleep disorders is differential diagnosis. Because many sleep disorders present with similar symptomatology (e.g., excessive daytime sleepiness, delayed sleep onset) and sleep complaints can arise from multiple possible causes (e.g., prolonged sleep-onset latency may be associated with restless legs syndrome, delayed sleep-phase disorder, or behaviorally based insomnia in childhood), differential diagnosis is especially critical in the assessment of sleep complaints. Differentiation between a sleep disorder and other medical or mental health conditions that may present with similar symptoms is also important. For example, a child with apparent sleep terror symptoms may actually have a nocturnal seizure disorder. Similarly, what appears to be delayed sleep onset related to normal developmental nighttime fears may be a manifestation in some children of a more serious generalized anxiety disorder.

Furthermore, in many cases, two or more sleep disturbances may coexist, and medical and behavioral sleep disorders are frequently comorbid. For example, a child may have both obstructive sleep apnea and behavioral issues with bedtime refusal. Thus, treating the sleep apnea alone will alleviate the nighttime arousals associated with sleep-disordered breathing but will not eliminate bedtime resistance. In addition, the presence of one sleep problem may exacerbate another; for example, partial arousal parasomnias may be triggered by insufficient sleep in susceptible children and nocturnal enuresis can be a sequela of sleep-disordered breathing. Finally, because sleep disorders can be secondary to, or exacerbated by, physical or mental illness, as well as neurodevelopmental disorders, it is essential to evaluate for possible contributing factors to the sleep problem with a comprehensive and thorough history.

Dual Diagnoses in Sleep

There are a number of sleep disorders that are often related and commonly coexist:

- Obstructive sleep apnea and secondary enuresis
- Obstructive sleep apnea and partial arousal parasomnias (e.g., sleepwalking, sleep terrors)
- Chronic inadequate sleep and partial arousal parasomnias
- Insomnia and inadequate sleep hygiene
- Bedtime problems and nightwakings
- Restless legs syndrome and periodic limb movement disorder
- Delayed sleep-wake phase disorder and sleep-onset insomnia

CONCEPTUAL FRAMEWORK OF SLEEP DISTURBANCES IN CHILDREN

Most sleep problems in children may be broadly conceptualized as involving one or more basic mechanisms: duration of sleep that does not meet sleep needs (insufficient sleep quantity), disruption and fragmentation of sleep (poor sleep quality), inappropriate timing of the sleep period (as occurs in circadian sleep-wake disturbances), or primary disorders of excessive daytime sleepiness (central hypersomnias such as narcolepsy) (Figure 3.1). Insufficient sleep is usually the result of difficulty initiating (delayed sleep onset) or maintaining sleep (prolonged nightwakings), whereas sleep fragmentation most often results from frequent, repetitive, and brief
arousals during sleep. Insufficient sleep duration, especially in older children and adolescents, may also represent a conscious lifestyle decision to sacrifice sleep in favor of competing priorities, such as homework and social activities, that is, voluntary sleep curtailment resulting in chronic failure to meet sleep needs.

![Conceptual framework of sleep disturbances in children. EDS, excessive daytime sleepiness.]

**COMPONENTS OF THE PEDIATRIC SLEEP EVALUATION**

Sleep complaints in primary care, as well as mental health and subspecialty medical settings, may be identified as a result of routine screening by the practitioner or may be spontaneously offered by the caregiver (or less frequently by the patient) during the health encounter. In addition, although concerns about sleep are commonly raised to healthcare providers by parents of infants and toddlers, caregivers may be less aware of and less attuned to sleep issues beyond the early childhood period, especially in adolescents. Furthermore, it should be emphasized that caregivers may not recognize some symptoms as indicative of a sleep problem (e.g., daytime behavioral issues) or view them as abnormal or problematic (e.g., snoring). Thus, a systematic approach to screening for sleep disorders needs to be a key component of routine well-child care. The sleep questions presented below (often referred to as the “BEARS”) outline a screening strategy that is appropriate, with some age-related modifications, across most age groups from infancy through adolescence.

**BEARS Screening Questions**
- **B** = Bedtime problems
- **E** = Excessive daytime sleepiness
- **A** = Awakenings during the night
- **R** = Regularity and duration of sleep
- **S** = Sleep-disordered breathing

**Examples of Developmentally Appropriate Trigger Questions**
### Sleep Problems by Age Group

<table>
<thead>
<tr>
<th></th>
<th>Preschool (2-5 y)</th>
<th>School-Aged (6-12 y)</th>
<th>Adolescent (13-18 y)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bedtime problems</strong></td>
<td>Does your child have any problems going to bed? Falling asleep?</td>
<td>Does your child have any problems at bedtime? (P)</td>
<td>Do you have any problems falling asleep at bedtime? (C)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Do you have any problems going to bed? (C)</td>
<td></td>
</tr>
<tr>
<td>**Excessive daytime</td>
<td>Does your child seem overtired or sleepy a lot during the day? Does he or she still take naps?</td>
<td>Does your child have difficulty waking in the morning, seem sleepy during the day, or take naps? (P) Do you feel tired a lot? (C)</td>
<td>Do you feel sleepy a lot during the day in school? while driving? (C)</td>
</tr>
<tr>
<td>sleepiness</td>
<td></td>
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</tr>
<tr>
<td>**Awakenings during the</td>
<td>Does your child wake up a lot at night?</td>
<td>Does your child seem to wake up a lot at night? Any sleepwalking or nightmares? (P)</td>
<td>Do you wake up a lot at night? Do you have trouble getting back to sleep? (C)</td>
</tr>
<tr>
<td>night</td>
<td></td>
<td>Do you wake up a lot at night?</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Have trouble getting back to sleep? (C)</td>
<td></td>
</tr>
<tr>
<td><strong>Regularity and duration of sleep</strong></td>
<td>Does your child have a regular bedtime and wake time? What are they?</td>
<td>What time does your child go to bed and get up on school days? Weekends? Do you think he or she is getting enough sleep? (P)</td>
<td>What time do you usually go to bed on school nights? Weekends? How much sleep do you usually get? (C)</td>
</tr>
<tr>
<td><strong>Sleep-disordered breathing</strong></td>
<td>Does your child snore a lot or have difficulty breathing at night?</td>
<td>Does your child have loud or nightly snoring or any breathing difficulties at night? (P)</td>
<td>Does your teenager snore loudly or nightly? (P)</td>
</tr>
</tbody>
</table>

P = Parent; C = Child

Additional sleep screening questionnaires that can be utilized in clinical practice are provided in Appendix B1. These include the Brief Infant Sleep Questionnaire (for ages 0-3), the Children's Sleep Habits Questionnaire (for ages 3-12), and both a self-report and parent report version of the Sleep Habits Questionnaire for adolescents.

Once a sleep problem has been identified, a step-wise approach to gathering information in the clinical setting regarding the presenting sleep complaint; daytime sequelae; and related sleep, medical, developmental, and...
psychiatric problems is obviously needed both to generate an appropriate differential diagnosis and to begin the process of treatment planning.

An initial assessment of pediatric sleep disturbances typically includes the following:

- Sleep history
- Review of medical history
- Developmental history/assessment of school functioning
- Family history
- Psychosocial history
- Behavioral assessment
- Physical examination

Further evaluation may include the completion of sleep diaries and an overnight polysomnogram or other diagnostic tests as appropriate. Focused questions pertaining to specific sleep disorders, such as obstructive sleep apnea and sleepwalking, are presented in the appropriate corresponding chapters. Symptom-based algorithms are also provided in Chapter 6 to facilitate a basic approach to diagnosis and subsequent treatment. In addition, sleep evaluation intake questionnaires for different age groups (infants, toddlers, school-aged, and adolescents) are provided in Appendix B1.

**SLEEP HISTORY**

The first step in evaluating a child or adolescent for a sleep disorder is the completion of a thorough sleep history (including presenting complaint, sleep patterns and schedules, bedtime, nocturnal behaviors, and daytime behaviors).

**Presenting Complaint**

It is important to elicit a description of the presenting complaint from all primary caregivers if possible (e.g., both the custodial and noncustodial parent if the child spends time in two households, grandparent(s) or step-parent(s) (if largely responsible for bedtime) and from the child or adolescent whenever appropriate. Often differences in perception of the issues between parents emerge, as well as discrepancies between child and parent perspective. For example, parents may attribute sleep-onset delay following “lights out” to noncompliance, whereas the child may state that he or she is “not sleepy” at the regular bedtime (but can fall asleep easily an hour later, suggesting a delayed sleep phase or an inappropriately early bedtime for age). Included in the description of the presenting complaint should be a detailed review of the onset, duration, severity, and night-to-night variability of the current sleep problem, as well as any perceived precipitating factors. This is particularly important because, as is the case with many behavioral problems in childhood, the ultimate determinant of what constitutes a sleep “problem” is highly subjective and dependent on the context in which it occurs, including caregiver tolerance for the behavior and the psychosocial context. Thus, a sleep-onset delay of 30 minutes that involves multiple “curtain calls” may be defined as “problematic” for one family, but a sleep latency of 60 minutes during which the child lies quietly in bed may not be considered problematic for another. A history of events that seem to have immediately preceded the onset of the sleep problem should also be elicited. It is also often helpful to ask parents what prompted them to address the sleep complaint at this particular time (e.g., imminent birth of a sibling, school problems). In addition, parents should be questioned in regard to current and past attempts to manage the sleep problem; for example, it is not uncommon for parents to state that they have “already tried everything” to address nightwakings, but with further
questioning it becomes apparent that interventions were instituted inconsistently or for inadequate periods.

Finally, clarifying parental expectations in regard to treatment outcomes assists in the development of mutually acceptable treatment goals and active exploration of opportunities and obstacles as well as allowing ongoing communication of issues and concerns.

**Sleep Patterns, Sleep Schedules, and Sleep Habits**

No matter what the presenting sleep complaint, it is important to have a general sense of sleep patterns and behaviors. A review of sleep habits will often shed considerable light on the nature, duration, and cause of the presenting sleep complaint, and may also illuminate the presence of coexisting sleep problems and maladaptive sleep behaviors (unhealthy sleep practices; see Chapter 5). General sleep behaviors over the previous several weeks (or over a “typical” 2-week period, if the most recent weeks have been unusual) should be reviewed. Review of sleep schedules during the school year compared to the summer or holidays can also help evaluate factors such as circadian preference and sleep need in school-aged children and adolescents. Note that it is not unusual for parents to state that their child has “never” slept well, but this should prompt a more thorough probe of specific triggers or exacerbating factors, as well as parental expectations regarding “normal” sleep.

**Bedtime**

It is often easiest to have the family describe events from dinnertime until sleep onset in order to obtain a complete picture of evening and bedtime behaviors. Specific information that may be helpful in elucidating the causes of and contributing factors to sleep problems include the following.

**Bedtime Schedule**

- **Presence of a set bedtime**, as some parents do not establish a regular consistent bedtime and allow their child to fall asleep whenever (and wherever) they want.

- **Actual bedtime (e.g., 8:00 p.m.) appropriateness** for age and developmental level, and **consistency of bedtime**, both night-to-night and weekday-to-weekend discrepancies. The impact of environmental demands (e.g., homework, after-school employment) on sleep schedule should also be explored.

- **Caregiver supervision of bedtime** indicates whether parents (or responsible adult) are monitoring and enforcing bedtime. Many parents, especially of adolescents, do not enforce bed-time limits because they go to bed before their child does, and thus have little idea what time their child actually goes to sleep. Furthermore, some children will “only go to bed for one parent,” suggesting the possibility of limit-setting issues, separation issues, or family conflict. Other children may “wait up,” for example, for a parent to return from a third-shift job. In other cases, a grandparent, sibling, or babysitter may be the primary caregiver supervising bedtime, in which case psychoeducation and intervention strategies should include these individuals.

**Bedtime Routines**

- **Evening activities**, particularly the child’s involvement in stimulating evening activities, including engagement in electronic device use such as television viewing and playing video games, as well as sports practices or other extracurricular activities. Engaging in evening extracurricular activities can impinge on an appropriate bedtime, contributing to insufficient sleep and daytime consequences.

- **Presence of a bedtime routine**, that is a series of consistent nighttime activities leading to bedtime.

- **Time of initiating the bedtime routine**, that is the time the child or adolescent begins to get ready for bed and **duration of the bedtime routine**, because extended or elaborate routines lasting longer than 30 minutes
may indicate a limit-setting problem.

- **Specific nature of the bedtime routine activities**, appropriateness for age, and supervision of activities by parents. Also assess if these activities are likely to be stimulating for that child (e.g., baths are not relaxing for all children; in addition, the activity closest to bedtime should be the most soothing).

- **Location of the bedtime routine**, such as in the child's room versus the parents' room.

- **Electronics**, including the prominence of television viewing or computer or video games in the bedtime routine, especially after 9:00 p.m., and the presence and number of electronics in the child's bedroom. Many parents excuse the presence of a television in a child's bedroom by claiming “he can't fall asleep without it” or that the “sound is turned off so it's OK.” Resistance to removing or reducing dependence on electronics as part of the bedtime routine is one of the more challenging sleep practice issues healthcare providers face.

<table>
<thead>
<tr>
<th>Sleep Associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep associations are behaviors that occur at the time of sleep initiation (e.g., nursing, rocking). Inappropriate sleep associations can contribute to bedtime difficulties and nightwakings since behaviors that occur at bedtime (e.g., nursing, rocking) may be required to reinitiate sleep following normal nighttime arousals (see Chapter 8).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bedtime Stalling or Refusal Behaviors</th>
</tr>
</thead>
<tbody>
<tr>
<td>A review of noncompliant behaviors should be conducted, including <strong>types</strong> of behaviors (e.g., crying, climbing out of crib, leaving the bedroom); <strong>intensity</strong> of the behaviors (e.g., calling out versus tantrums); <strong>frequency</strong> (how often they occur in an average week); and <strong>duration</strong> (how long they typically last, as well as what typically terminates them (e.g., parents give in and allow the child to sleep in their bed), as well as the presence of bedtime fears. It is also important to assess any discrepancy between the child's behavior from the perspective of both parents (or multiple caregivers), as well as the parental response to the child's behavior at bedtime (see also Chapter 7).</td>
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</tbody>
</table>

<table>
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<tr>
<th>Falling Asleep</th>
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<tbody>
<tr>
<td><strong>Time of lights out in relation to sleep onset</strong>, that is, the time the child actually falls asleep. Specifying these time periods may narrow down the sleep problem and help distinguish whether the main issue is bedtime resistance (e.g., fights bedtime but falls asleep fairly easily once in bed) or difficulty falling asleep (e.g., does not protest bedtime but lies in bed awake for prolonged periods) or both. In children who are primarily having difficulty falling asleep, factors that may contribute to delayed sleep onset should be evaluated, including nighttime anxiety and potential symptoms of restless legs syndrome (e.g., urge to move, leg discomfort, restlessness). In these cases, direct questioning of the child may be particularly important. On the other hand, pinpointing the time that an older child/adolescent gets into bed (or turns out the light) with the intent of falling asleep (compared to, for example, getting into bed with the intention of reading or listening to music for an hour) may help to define the actual duration of sleep-onset latency.</td>
</tr>
</tbody>
</table>

| Level of sleepiness (“readiness” for bed), of the child at the typical bedtime. Children and adolescents who fall asleep easily at a later bedtime (e.g., on weekends) may have a delayed sleep phase. Alternatively, having a set bedtime that coincides with the surge of alertness typically preceding sleep onset (the “second wind” or “forbidden zone” phenomena) can create difficulties in falling asleep. |
Sleep location, particularly where sleep onset typically occurs (e.g., living room), the child's activity at the time (e.g., on the couch watching television), variability in location of sleep onset from night to night, and parental presence in that location.

Transfers after sleep onset, that is whether the child falls asleep elsewhere (e.g., in the parents' bed) and is subsequently transferred to his or her own bed. If a strong sleep-onset association is established with an "alternative" location, then upon waking during the night, the child will seek out that same location in order to fall back to sleep.

Sleeping Environment

Bedroom space and location, including bedroom-sharing with a parent, sibling, or other household member and location of child's bedroom in relation to parents' room (e.g., child's anxiety may be increased if the bedroom is far away or on a different floor). Even if it is a conscious choice, based on such variables as cultural values or space availability, sharing a room/bed with a caregiver may very well increase the likelihood that the caregiver will be aware of (and thus report) sleep disruption in children. For children who frequently sleep in different households besides their primary residence (e.g., noncustodial parent, grandparents), the sleeping arrangements and environment, as well as the sleep schedule in each, should be described.

Light, including use of nightlights and other lights in the room (child's need for bright lighting may be indicative of anxiety issues). Other light sources, especially those close to and directed into the eyes such as "screens" (e.g., television, computer, e-readers) in the evening may interfere with nighttime melatonin secretion and normal circadian rhythm patterns.

Noise, including amount and type of noise, from other family members and outdoor noise, as well as sound levels at bedtime, during the night, and in the early morning (e.g., parental early rising for work schedule).

Temperature, including availability of adequate temperature control (e.g., heat, air-conditioning).

Bed type, as a crib provides natural limits to out-of-bed behaviors, while a bed allows unlimited movement. Moving a child from a crib to a bed prematurely sometimes initiates or exacerbates sleep problems. Young children commonly do not have the cognitive development or behavioral control to stay within the essentially imaginary boundaries of a bed. If sleep is problematic with the switch to a bed, returning to a crib until the child is more developmentally ready may be appropriate and often resolves the sleep issue.

Bedding, including different sleeping surfaces (e.g., mattress, futon) and personal preference (e.g., hard versus soft mattress). Although there are no data on the relative advantage of different sleeping surfaces in regard to sleep quality in children, safety concerns (e.g., pillows for young infants) and medical issues (e.g., environmental allergies) may be considerations in some cases. Research, however, continues to indicate that infants who sleep on couches and other soft surfaces are at increased risk for suffocation and sudden infant death syndrome.

Co-sleeping, including assessment of type (room-sharing, bed-sharing, other), persons involved (siblings, parents, grandparents), intensity (all-night versus part-night co-sleeping), timing (starts out in own bed versus parents' bed), frequency (nightly versus intermittently), reasons for co-sleeping (lifestyle choice versus “reactive” co-sleeping in response to a sleep problem), and parents' reaction to co-sleeping (discrepancies in parental acceptance, displacement of one parent from the marital bed).

Daytime Sleep

Naps, including timing, duration, location, and consistency of napping patterns, as well as ease or difficulty of falling asleep at naptime. It should be emphasized that unplanned napping (e.g., dozing off in the car, while
engaged in sedentary activities) at all ages and the need for planned naps in a child after the age of 5 years is suggestive of insufficient sleep or an underlying sleep-disrupting factor.

**Nocturnal Behaviors**

Nighttime behaviors are important to review in order to obtain a complete picture of the sleep problem. These include a complete evaluation of *nightwakings* (see Chapter 8), including approximate date of initial presentation; frequency per week, number per night, timing, and duration of nightwaking episodes; identifiable triggers (e.g., getting up to void, respiratory disturbance, parent coming home from third-shift work); behaviors that occur upon waking (calling out, coming into parents’ bed); type and consistency of parental response to nightwaking (ignoring, intermittent reinforcement); and what assistance the child needs to return to sleep (parental soothing, breast-feeding, or bottle-feeding). In addition, the presence and nature of episodic nocturnal events such as *disorders of arousal* (see Chapter 11) and *nightmares* (see Chapter 10), as well as symptoms possibly suggestive of *sleep-disordered breathing* (see Chapter 15) and *periodic limb movement disorder* (PLMD) [see Chapter 16]) should be elicited.

**Daytime Behaviors**

It is critical that daytime behaviors be assessed, particularly those that may be contributing to or indicative of significant daytime sleepiness (morning waking, daytime sleepiness and low functioning, and fatigue), in order to quantify the impact of the sleep disturbance on the child and family. In addition, elucidation of the daily schedule, including timing of meals (e.g., too close to bedtime) and the regularity and structure of daytime activities (e.g., lack of exercise during the day or timing vigorous exercise periods, stimulating activities too close to bedtime, lack of a consistent daily schedule) is also important. Daytime naps may also impact nocturnal sleep.

**Morning Awakening**

- **Time of awakening**, including weekday versus weekend waketime. For example, children with a delayed circadian sleep-wake rhythm may select their preferred (later) waketime when there is an option on weekends and school vacations. In addition, it is particularly important in older children and adolescents to note the presence of *weekend oversleep*, i.e., sleeping in for 2 or more hours on weekend mornings to “catch up,” because this practice suggests that the weekday sleep duration is insufficient.

- **Difficulty waking in the morning**, and mood and cognitive functioning upon awakening, such as irritability and grogginess versus alertness and a “readiness to start the day.” The need for multiple attempts at waking by parents, siblings, or others versus spontaneous waking at the desired time is one of the best indicators of insufficient sleep. The time that the child spontaneously awakens (or would spontaneously awaken if allowed) is also important to note, especially during prolonged periods in which a self-selected sleep-wake schedule is an option (such as holiday or summer vacation). This helps identify both the child's “intrinsic” preferred timing of sleep as well as “average” sleep need (taking into account that a chronically sleep-deprived adolescent, for example, is likely to require a week of extended sleep during vacation

which involves “sleeping in” before reverting to sleeping the amount they actually need). Difficulty waking in the morning despite what appears to be a sufficient sleep quantity suggests that the quality of sleep may be compromised by an underlying sleep disorder (e.g., sleep-disordered breathing, PLMD). Extreme confusion, grogginess, and difficulty in awakening on school days (i.e., “sleep inertia”) may occur in adolescents with delayed sleep-wake phase disorder, both as a result of insufficient sleep duration and because they are attempting to wake up during their “circadian trough” of alertness (i.e., 6:00-8:00 a.m.).

- **Consequences of difficulty waking**, including habitual tardiness or school absences.
You Snooze, You Lose

In general, the habitual need for an alarm clock in order to awaken in the morning, especially multiple “snooze alarm” hits, strongly suggests insufficient sleep. This is particularly true in school-aged children, who should awaken spontaneously in the morning if adequately rested.

Daytime Sleepiness

Falling asleep in school, while riding in a car, and during passive, or in particular, stimulating (e.g., in conversation, at mealtime) activities is indicative of insufficient sleep, an underlying sleep disruption or, in rare case, a primary disorder of excessive daytime sleepiness. In younger children, daytime sleepiness may be manifested as “overtiredness,” which may be described by parents as irritability, aggressive, oppositional, or acting-out behavior, disinhibition and silliness, or hyperactivity and impulsivity.

Daytime Functioning

Insufficient or poor sleep may have negative consequences on a host of functional domains, including mood, behavior, attention, learning, school performance, social relationships, and health.

Fatigue/Tiredness

Sleepiness is the tendency to doze off or fall asleep, often in inappropriate settings, and generally implies insufficient and/or disrupted sleep. Complaints of fatigue or being tired, on the other hand, are often indicative of subjective feelings of low energy and low motivation, which may or may not be primarily a result of poor sleep. Fatigue may be defined as lethargy without sleep initiation and tends to be associated with medical (e.g., thyroid function) or psychiatric (e.g., depression) disorders. However, a distinction between sleepiness and fatigue is not always possible, as there may be considerable overlap in descriptions of these two states by parents and older children and adolescents.

MEDICAL HISTORY

A standard pediatric medical history and review of systems should be elicited, with particular emphasis on past and current medical conditions, prior hospitalizations and surgeries (especially history of adenotonsillectomy for symptoms of sleep-disordered breathing), and medications (see Chapter 22 for a detailed discussion on the interaction between sleep and medical issues and Chapter 20 for a complete review of the effects of specific medications on sleep).

DEVELOPMENTAL/SCHOOL HISTORY

A developmental history is important, including a history of delays in achievement of key developmental milestones, concerns about development, prematurity, and impaired neurologic functioning, as there is often an increased prevalence of sleep problems in children with neurodevelopmental disorders (see Chapter 21). Significant sleep disruption and inability to settle or self-soothe in a young child may be an early indication of a developmental delay. Furthermore, academic problems are one of the most important sequelae of insufficient and/or disrupted sleep, and provide an indication of the daytime impact of sleep disturbances. Developmental history questionnaires for three age groups (0-3 years, 4-12 years, and 13+ years) are provided in Appendix B1.

FAMILY HISTORY

A family history of sleep problems can be helpful in confirming the diagnosis of certain sleep disorders, especially for those disorders that may have a genetic component. Such diagnoses include partial arousal parasomnias
(sleep terrors and sleepwalking), RLS, obstructive sleep apnea, and narcolepsy. In many cases, these disorders have not been formally diagnosed in family members, but symptoms are clearly present if directly questioned (e.g., “His father snores so loudly, he has to sleep in another room!”). In addition, parental experience with sleep problems, such as chronic insomnia, may impact on parental attitudes toward and management of similar sleep problems in the child.

**Like Father, Like Son**

One of the most rewarding aspects of diagnosing and treating pediatric sleep disorders is the potential to impact the whole family. Not only does successful treatment of a child’s sleep problem result in everyone getting a better night's sleep, but it may encourage adult family members to obtain needed help for their own sleep problems. Parents may be much more willing to seek medical attention for loud snoring, daytime sleepiness, or symptoms of restless legs when their child is also being evaluated and treated for a similar symptom.

**PSYCHOSOCIAL HISTORY**

A complete psychosocial history is appropriate, including the following:

- **Family functioning**, including overall family functioning, effectiveness of parenting skills (including limit-setting), family structure (e.g., siblings, extended family members in household, one-parent versus two-parent household), parental psychological functioning (e.g., parental depression), and family discord. Some caregivers may have their own issues (e.g., mental illness, long work hours) that interfere with their ability to set clear limits both during the day and at bedtime. In other cases, there is a “mismatch” between parental expectations regarding sleep behaviors and the normal developmental trajectory.

- **Parental separation or divorce**, including time elapsed since the separation or divorce, visitation schedules and level of involvement of noncustodial parent, consistency of sleep patterns and habits across households, discrepancies in parenting styles, and stress on the child.

- **Significant life events**, such as death of a family member, change in school, or a recent move, that may result in adjustment sleep problems.

- **Family and cultural context**, including cultural influences on sleep behaviors, family values regarding health priorities, and family beliefs about sleep and the importance of sleep.

- **Impact on family of the sleep problem**, including the effect of the child's sleep on the parents' sleep, impact on parents' daytime functioning, and impact on marital and family satisfaction.

**BEHAVIORAL ASSESSMENT**

A behavioral and psychological screening assessment is important because sleep disturbances can result in psychiatric symptoms, such as mood changes and oppositional behavior; conversely, psychiatric disorders can result in sleep disturbances. Thus, particularly in older children and adolescents, it is important to elicit possible symptoms of depression, anxiety, and other psychiatric disorders. In younger children, bedtime problems are also often associated with child temperament. For example, “fussy” children may insist on a particular type of soothing or sleep-inducing technique, resisting any alternative that is less dependent on the caregiver.

**PHYSICAL EXAMINATION**
A physical examination should be conducted on all children and adolescents being evaluated for sleep complaints. The presence of specific symptoms (e.g., snoring, severe sleepiness) should prompt a more detailed examination according to the systems affected; more detailed aspects of the physical examination in various specific sleep disorders (e.g., obstructive sleep apnea) may be found in the respective chapters. Although the physical examination is normal in many children with sleep disorders, a complete physical examination is an essential part of the evaluation of sleep problems. In particular, careful attention should be paid to the following points:

- **Growth parameters**, including height, weight, and age- and gender-adjusted body mass index. For example, growth failure and obesity are both associated with sleep-disordered breathing. Assessment of pubertal status (Tanner stage) may be helpful in identifying a circadian component in patients presenting with recent onset of sleep initiation insomnia, because puberty is associated with a normal biological shift (delay) in sleep-wake cycles.

- **General appearance**, including activity level and evidence of fatigue and/or sleepiness. Children with severe obstructive sleep apnea or insufficient sleep, and especially those with narcolepsy, may actually doze off while waiting or even during the evaluation. Alternatively, sleepy children may appear hyperactive, irritable, or disinhibited in the office.

- **Head and neck examination**, including examination of the nose, mouth, and throat for possible risk factors (e.g., adenotonsillar hypertrophy, high Mallampati score) for suspected obstructive sleep apnea (see Chapter 15). The HEENT (head, eyes, ears, nose, throat) examination may also suggest evidence of atopic disease (e.g., allergic “shiners,” bogginess of the nasal mucosa, cobblestoning of the soft palate) contributing to sleep fragmentation and as a risk factor for sleep-disordered breathing. Evidence of midface hypoplasia or other craniofacial abnormalities should be noted. Examining the side profile may help identify micro/retrognathia as a contributor to sleep-disordered breathing.

- **Neurologic assessment**, particularly in cases where the concern is excessive sleepiness or where there is a possibility of nocturnal seizures.

**Observation of Family Interactions**

The process of obtaining a sleep history and examination of a child also affords the practitioner the opportunity to observe family interactions. For example, lack of limit-setting in the office can be an indication of similar issues in the home, which can contribute to bedtime problems and nightwakings.

**DIAGNOSTIC TOOLS**

**Sleep Diaries**

An important step in the evaluation of many sleep problems is the collection of sleep diaries or logs. A typical sleep diary collects information on bedtime (“lights out”), latency to sleep onset, number and duration of nighttime awakenings, time of morning waking, total sleep time, sleep efficiency (time asleep and time in bed), and duration and time of naps. Two weeks of baseline sleep diaries are usually adequate to delineate sleep patterns. (Samples of different styles of sleep diaries are provided in Appendix B2.) Parents typically complete these diaries; however, in older school-aged children and adolescents, more accurate information may be obtained by having the patient complete the sleep log.

**Overnight Polysomnography**

Once basic information regarding the sleep history as well as a review of medical, developmental, and behavioral
issues have been obtained, it is important to determine whether an overnight sleep

study (i.e., polysomnography [PSG]) is needed. A PSG is a diagnostic test typically performed in a sleep

laboratory that yields detailed information regarding sleep architecture (distribution and percentage of sleep

stages), cardiorespiratory parameters, body movements, and arousals during sleep. See Chapter 4 for a detailed

description of indications for and interpretation of PSG in children.

**Actigraphy**

Actigraphy is a diagnostic procedure that utilizes a compact, lightweight, computerized accelerometer-based

wristwatch-like device to record and store information regarding body movements over a period of time (minimum

of 72 hours; typically 7-14 consecutive days). Actigraphs collect and store data in “epochs,” which vary by device

and can range from 1 second to 5 minutes. They need to be connected to a computer to initialize and download

data. While actigraphy should not be viewed as a substitute for PSG when an overnight laboratory sleep study is

indicated (see Chapter 4), actigraphy provides evaluation of sleep patterns. It allows for continuous recording for
days or weeks in the home sleep environment and, therefore, can record information that is not captured during

a night in the sleep laboratory. It is also easier to use, more convenient and less expensive compared to PSG.

The data collected by the actigraph, in conjunction with a sleep diary, is then analyzed via computer software,

using preset activity thresholds to generate a printout of approximate sleep-wake patterns. Software programs

for the various available devices now allow for reliable automatic scoring based on established algorithms and

yield data reports and clinical summaries. Practice parameters for the clinical use of actigraphy in adults and in

infants and children have now been published. Actigraphy appears to be most useful in delineating sleep

patterns and to diagnose circadian rhythm disorders (advanced or delayed sleep-wake phase). Actigraphy may

also be used to more accurately document sleep duration, nightwakings, and, less reliably, sleep-onset latency

in patients for whom there appears to be a discrepancy between subjective sleep complaints and daytime

consequences (e.g., an adolescent who reports sleeping less than 4 hours per night but has no complaints of
daytime sleepiness). Actigraphy can also be helpful in determining sleep amounts in families in which the parents

are unable to provide adequate information regarding nighttime awakenings (e.g., child gets up to watch

television during the night and the parents are unaware of these behaviors). Actigraphy is becoming more

common in clinical assessment, although some challenges continue to exist regarding insurance compensation.

Finally, there are now a number of “direct-to-consumer,” relatively low-cost portable devices in the market (e.g.,

“Fitbit”), that purport to measure sleep-wake cycles. While these devices have a number of appealing features,

including integration with mobile devices and software, currently there are no available data to establish the

validity and reliability of sleep parameters measured by these devices. In fact, the few available studies suggest

they are not sufficiently valid for use in clinical settings for the measurement of sleep, and thus are not currently

recommended.
While a detailed description of the process of conducting, scoring, and interpreting nocturnal polysomnograms in children is beyond the scope of this chapter, it is important for all pediatric practitioners to have some basic understanding of and appreciation for this important diagnostic sleep test. In particular, working knowledge of the appropriate indications for ordering a sleep study in children and adolescents, guidelines for evaluating the qualifications of a sleep lab to adequately assess children, and the basics of understanding and utilizing a sleep study report are key components to ensuring that children and adolescents with sleep disorders receive optimal care.

Evidence-based practice parameters for the respiratory and nonrespiratory indications for polysomnography (PSG) in children were published by the American Academy of Sleep Medicine (AASM) in 2011 and 2013, respectively. The guidelines for respiratory PSG indications in children and adolescents are provided in Table 4.1. Table 4.2 presents the respiratory indications pertaining specifically to infants and special populations. Finally, the nonrespiratory indications for PSG in children can be seen in Table 4.3.

It is important to note the reasons sometimes cited by referring providers in ordering a PSG that are not included in these guidelines. For example, insomnia (difficulty initiating or maintaining sleep) is not an indication for PSG unless there is evidence to suggest the presence of a comorbid sleep disorder (see complete coverage of insomnia in Chapter 19). In general, the diagnosis of circadian rhythm disorders, such as delayed sleep-wake phase disorder (Chapter 18), also does not require PSG. In fact, PSG is often a waste of time, effort, and cost in patients with a circadian rhythm disorder; because of their inability to fall asleep at a “normal” bedtime, the amount of sleep captured on an overnight PSG is frequently inadequate to draw any conclusions. Thus, if a comorbid sleep disorder such as obstructive sleep apnea (OSA) that requires a PSG for diagnosis is suspected, it is prudent to address the sleep-wake schedule issues first and then obtain the PSG once the sleep schedule is regularized. Similarly, PSG is rarely indicated in the assessment of sleep disorders for which the diagnosis is based on clinical signs and symptoms, such as parasomnias, enuresis, restless legs syndrome, or bruxism. In these cases, an overnight sleep study is warranted only if an underlying sleep disorder, such as sleep apnea, is suspected. Finally, The multiple sleep latency test (MSLT) for evaluation of potential narcolepsy should not be routinely ordered without a thorough evaluation for other causes of hypersomnia (e.g., chronic insufficient sleep, delayed sleep-wake phase disorder, sleep-disordered breathing).

<table>
<thead>
<tr>
<th>TABLE 4.1. AASM Practice Parameters for the Respiratory Indications of Polysomnography in Children (2011)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Children with mild OSA preoperatively should have clinical evaluation following adenotonsillectomy to assess for residual symptoms. If there are residual symptoms of OSA, polysomnography (PSG) should be performed. (Standard)</td>
</tr>
<tr>
<td>2. PSG is indicated following adenotonsillectomy to assess for residual OSA in children with preoperative evidence for moderate to severe OSA, obesity, craniofacial anomalies that obstruct the upper airway, and neurologic disorders (e.g., Down syndrome, Prader-Willi syndrome, and myelomeningocele). (Standard)</td>
</tr>
<tr>
<td>3. PSG is indicated for positive airway pressure (PAP) titration in children with obstructive sleep apnea</td>
</tr>
</tbody>
</table>
4. Follow-up PSG in children on chronic PAP support is indicated to determine whether pressure
requirements have changed as a result of the child's growth and development, if symptoms recur while
on PAP, or if additional or alternate treatment is instituted. (Guideline)
5. PSG is indicated after treatment of children for OSA with rapid maxillary expansion to assess the level of
residual disease and to determine whether additional treatment is necessary. (Option)
6. Children with OSA treated with an oral appliance should have clinical follow-up and PSG to assess
response to treatment. (Option)

*Each recommendation is followed by an assessment of the strength of supporting evidence (in descending
order, Standard-Guideline-Option).

TABLE 4.2. AASM Practice Parameters for the Respiratory Indications for PSG in Infants and
Special Populations*

- Polysomnography (PSG) is indicated in selected cases of primary sleep apnea of infancy. (Guideline)
- PSG is indicated when there is clinical evidence of a sleep related breathing disorder in infants who
have experienced an apparent life-threatening event. (Guideline)
- PSG is indicated when the clinical assessment suggests the diagnosis of congenital central alveolar
hypoventilation syndrome or sleep related hypoventilation due to neuromuscular disorders or chest wall
deformities.
- PSG is indicated for noninvasive positive pressure ventilation titration in children with other sleep-related
breathing disorders. (Option)
- Children treated with mechanical ventilation may benefit from periodic evaluation with PSG to adjust
ventilator settings. (Option)
- Children treated with tracheostomy for sleep related breathing disorders benefit from PSG as part of the
evaluation prior to decannulation. These children should be followed clinically after decannulation to
assess for recurrence of symptoms of sleep related breathing disorders. (Option)
- PSG is indicated in the following respiratory disorders only if there is a clinical suspicion for an
accompanying sleep related breathing disorder: chronic asthma, cystic fibrosis, pulmonary hypertension,
bronchopulmonary dysplasia, or chest wall abnormality such as kyphoscoliosis. (Option)

Recommendations against PSG Use:
- Nap (abbreviated) PSG is not recommended for the evaluation of obstructive sleep apnea syndrome in
children. (Option)
- Children considered for treatment with supplemental oxygen do not routinely require PSG for
management of oxygen therapy. (Option)

*Each recommendation is followed by an assessment of the strength of supporting evidence (in descending
order, Standard-Guideline-Option).

TABLE 4.3. AASM Practice Parameters for the Nonrespiratory Indications for PSG and MSLT
in Children*
Polysomnography (PSG) is indicated for children suspected of having periodic limb movement disorder (PLMD) for diagnosing PLMD. (Standard)

The MSLT, preceded by nocturnal PSG, is indicated in children as part of the evaluation for suspected narcolepsy. (Standard)

Children with frequent NREM parasomnias, epilepsy, or nocturnal enuresis should be clinically screened for the presence of comorbid sleep disorders and PSG should be performed if there is a suspicion for sleep-disordered breathing or PLMD. (Guideline)

The MSLT, preceded by nocturnal PSG, is indicated in children suspected of having hypersomnia from causes other than narcolepsy to assess excessive sleepiness and to aid in differentiation from narcolepsy. (Option)

The polysomnogram using an expanded EEG montage is indicated in children to confirm the diagnosis of an atypical or potentially injurious parasomnia or to differentiate a parasomnia from sleep related epilepsy. (Option)

PSG is indicated in children suspected of having restless legs syndrome (RLS) who require supportive data for diagnosing RLS. (Option)

**Recommendations against PSG Use:**

- PSG is not routinely indicated for evaluation of children with sleep related bruxism. (Standard)

*Each recommendation is followed by an assessment of the strength of supporting evidence (in descending order, Standard-Guideline-Option).*

**NORMAL PSG PARAMETERS IN CHILDREN**

In order to fully appreciate the information yielded by a PSG, it is helpful to review the typical parameters measured and to understand “normal” values for different age groups (see Tables 4.4 and 4.5). With regard to the latter, although there are only a handful of studies that have examined ranges for key PSG parameters in typically developing healthy children, the studies that do exist provide a framework for comparison purposes for an individual patient. Moreover, it should also be kept in mind that “normal” in-lab PSG parameter values, particularly in regard to sleep architecture and especially in younger children, may not necessarily mirror typical sleeping conditions in the home setting. For example, the so-called “first night effect,” in which a child's sleep may be negatively influenced by sleeping in a strange environment with multiple monitors and sensors attached, frequently results in delayed sleep onset, more frequent arousals and prolonged awakenings from sleep, delayed onset of and reduced REM (rapid eye movement) sleep, atypical sleeping positions, and increased “light” sleep (N1 and N2). These alterations in sleep architecture potentially impact respiratory parameters. For example, sleep-disordered breathing is often worse in REM sleep and in the supine position, and thus, results in the lab may under-represent the severity of sleep-disordered breathing. On the other hand, the sleep lab environment may be considerably less “allergenic” compared to home and thus result in a temporary improvement in sleep-disordered breathing during the study. “Morning-after” questionnaires completed by the patient or the accompanying caregiver in the lab can help in clarifying whether the PSG night was “typical” in terms of breathing and sleep quality compared to home and may aid in interpretation of the results.

---

**TABLE 4.4. Normal PSG Values for Children Aged 3 to 9 Year**
<table>
<thead>
<tr>
<th>Parameter</th>
<th>3-6 y old: Mean (SD)</th>
<th>6-9 y old: Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Sleep Time (TST) (min)</td>
<td>475 (42)</td>
<td>472 (43)</td>
</tr>
<tr>
<td>Stage N1 (NREM 1) (%)</td>
<td>6.6 (4.8)</td>
<td>7.1 (5.5)</td>
</tr>
<tr>
<td>Stage N2 (NREM 2) (%)</td>
<td>41.6 (7.1)</td>
<td>46.1 (8.5)</td>
</tr>
<tr>
<td>Stage N3 (NREM 3) (%)</td>
<td>28.2 (8.8)</td>
<td>24 (9.5)</td>
</tr>
<tr>
<td>Stage R (REM) (%)</td>
<td>23.6 (4.8)</td>
<td>22.6 (5.2)</td>
</tr>
<tr>
<td>Sleep Efficiency (TST/Time in Bed [TIB])</td>
<td>90 (7)</td>
<td>89.3 (7.5)</td>
</tr>
<tr>
<td>Sleep-Onset Latency (SOL) (min)</td>
<td>24.1 (25.6)</td>
<td>23 (25.3)</td>
</tr>
<tr>
<td>Latency to REM (min)</td>
<td>87.8 (41.2)</td>
<td>132 (57.7)</td>
</tr>
<tr>
<td>Arousal Index (# of arousals + awakenings/h TST)</td>
<td>9.0 (3.4)</td>
<td>9.5 (5.3)</td>
</tr>
<tr>
<td>Periodic Limb Movement Index (PLMI) (# of periodic limb movements/h TST)</td>
<td>1.4 (1.4)</td>
<td>0.91 (1.2)</td>
</tr>
<tr>
<td>Periodic Limb Movement Arousal Index (PLMAI) (# of periodic limb movements associated with arousals/awakenings/h TST)</td>
<td>0.04 (0.12)</td>
<td>0.10 (0.24)</td>
</tr>
<tr>
<td>Obstructive Apnea Index (#/h TST)</td>
<td>0.03 (0.1)</td>
<td>0.05 (0.11)</td>
</tr>
<tr>
<td>Mixed Apnea Index (#/h TST)</td>
<td>0.01 (0.05)</td>
<td>0.01 (0.06)</td>
</tr>
<tr>
<td>Central Apnea Index (#/h TST)</td>
<td>0.03 (0.01)</td>
<td>0.45 (0.49)</td>
</tr>
<tr>
<td>Obstructive Apnea/Hypopnea Index (#/h TST)</td>
<td>0.08 (0.16)</td>
<td>0.14 (0.22)</td>
</tr>
<tr>
<td>Nadir SpO₂ (%)</td>
<td>92.7 (4.5)</td>
<td>92.6 (3.6)</td>
</tr>
<tr>
<td>Desaturation Index (&gt;4%)</td>
<td>0.29 (0.35)</td>
<td>0.47 (0.96)</td>
</tr>
<tr>
<td>Mean End-Tidal CO₂</td>
<td>40.6 (4.6)</td>
<td>40.7 (4.5)</td>
</tr>
<tr>
<td>Heart Rate (6-11 y of age)†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td>76 (8.2)</td>
</tr>
</tbody>
</table>
Female

79.6 (9.2)


†Values averaged across samples

### TABLE 4.5. Normal PSG Values for Children Aged 10 to 16 Year

<table>
<thead>
<tr>
<th>Parameter</th>
<th>10-13 y old: Mean (SD)</th>
<th>14-16 y old: Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage N1 (NREM 1) (%)</td>
<td>5.5 (3.0)</td>
<td>6.8 (4.8)</td>
</tr>
<tr>
<td>Stage N2 (NREM 2) (%)</td>
<td>50.1 (6.7)</td>
<td>58.2 (7.6)</td>
</tr>
<tr>
<td>Stage N3 (NREM 3) (%)*</td>
<td>26.4 (7.0)</td>
<td>17.3 (6.7)</td>
</tr>
<tr>
<td>Stage R (REM) (%)</td>
<td>17.8 (5.3)</td>
<td>17.8 (5.1)</td>
</tr>
<tr>
<td>Sleep Efficiency (TST/Time in Bed (TIB))</td>
<td>86.0</td>
<td>89.0</td>
</tr>
<tr>
<td>Sleep-Onset Latency (SOL) (min)</td>
<td>18.5</td>
<td>15.5</td>
</tr>
<tr>
<td>Latency to REM (min)</td>
<td>150.3</td>
<td>126.5</td>
</tr>
<tr>
<td>Arousal Index (# of arousals + awakenings/h TST)</td>
<td>10.4 (3.7)</td>
<td>10.1 (3.7)</td>
</tr>
<tr>
<td>Periodic Limb Movement Index (PLMI) (# of periodic limb movements/h TST)</td>
<td>0.2</td>
<td>0.1</td>
</tr>
<tr>
<td>Obstructive Apnea Index (#/h TST)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Central Apnea Index (#/h TST)</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Obstructive Apnea/Hypopnea Index (#/h TST)</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Nadir SpO₂ (%)</td>
<td>93.2</td>
<td>93.5</td>
</tr>
<tr>
<td>Mean End-Tidal CO₂</td>
<td>49.5</td>
<td>49.3</td>
</tr>
</tbody>
</table>
Stage N3 combines prior NREM stages 3 and 4, also known as slow wave, delta, or “deep” sleep Adapted from Tapia IE, Karamessinis L, Bandla P, et al. Polysomnographic values in children undergoing puberty: pediatric vs. adult respiratory rules in adolescents. Sleep 2008;31:1737-1744.

Finally, there are a number of critical differences in conducting, scoring, and interpretation of PSGs in infants, children, and adolescents compared to adults. Key distinctions of sleep staging rules for children are listed in Table 4.6 and respiratory rules in Table 4.7. Further discussion of what to look for in a sleep lab is provided below.

**WHAT TO LOOK FOR IN A SLEEP LAB**

Overnight PSG should be performed in an AASM-accredited sleep laboratory by technicians skilled in working with children and interpreted by a board-certified sleep medicine professional with pediatric experience. Required channels include those for sleep staging, arousal measurement, cardiovascular parameters, and body movements (EEG, EOG, chin and leg EMG, ECG, body position sensors, and video recording). There should also be a combination of respiratory monitors, including oronasal thermal sensor and nasal air pressure transducer for airflow, chest/abdominal monitors (e.g., inductance plethysmography) for respiratory effort, pulse oximetry for O₂ saturation, and end-tidal or transcutaneous CO₂ for hypercarbia, as well as a snore microphone.

At the current time, there is no separate accreditation process or designation for pediatric sleep labs or for primarily adult sleep labs that also study children. Despite this, a recent survey suggested that more than half of the 1,500+ AASM-accredited “adult” labs would accept children over 12 years, almost half would accept children over 5 years, and almost a third would study children under 3 years. There are relatively few sleep labs around the country that exclusively serve the pediatric population and most of these are located in academic centers and in large urban areas. Furthermore, out of the 163 children's hospitals in the United States, as of 2012, only 28 had pediatric sleep centers.

**TABLE 4.6. Not Just Short Adults: Sleep Staging Rules for PSG in Children**

Pediatric scoring rules should be used for children aged over 2 mo post-term (there is no precise upper age boundary)

- The following terminology is used to score sleep stages in children:
  -- Stage W (Wakefulness)
  -- Stage N1 (NREM)
  -- Stage N2 (NREM)
  -- Stage N3 (NREM)
  -- Stage R (REM)
- Posterior dominant rhythm is the term used to describe the EEG pattern seen in relaxed wakefulness with eyes closed in young children and is referred to as alpha rhythm in older children and adults.
- EEG sleep staging features unique to children include vertex sharp waves and hypnagogic hypersynchrony, which occur in drowsiness and lighter stages of sleep (N1 and N2); these are normal findings which can be misinterpreted as seizure activity.
- Delta waves (slow-wave activity) are much higher in amplitude in children.
- Arousals in children are scored using adult criteria (an abrupt shift in EEG frequency lasting at least 3 s with at least 10 s of preceding stable sleep); however, this definition is controversial, as it may fail to capture the more subtle manifestations of sleep fragmentation associated with respiratory events or movements due to the higher arousal threshold in children.
TABLE 4.7. Not Just Short Adults: Respiratory Scoring Rules for PSG in Children

- Because children have a higher normal respiratory rate, the definition of “pathologic” duration of respiratory events is at least 2 missed baseline breaths rather than 10 s, as in adults. For example, if the normal respiratory rate in a preschooler is 24, then 2 missed breaths is about 5 s in duration.
- In contrast to adults, central events in children are defined as those lasting >20 s or those with a duration >2 missed breaths and associated with >3% desaturation or awakening/arousal. Central apneas are more common in children and often occur after a movement or arousal. These latter events are a normal physiologic response to hypocapnia and are generally not considered pathologic.
- In contrast to adults, assessment for hypercapnia with either end-tidal or transcutaneous CO$_2$ monitoring is required as part of the standard polysomnogram.
- Sleep related hypoventilation in children is defined as a PCO$_2$ > 50mm/Hg for >25% of total sleep time. Obstructive hypoventilation without frank apneas or hypopneas can be used as part of the diagnostic criteria for OSA.
- Respiratory scoring rules for children can be used up to age 18, but some sleep labs use adult criteria for adolescents (aged 13 y and older).

Given the existing situation and the likelihood that many pediatric practitioners do not have ready access to a pediatric sleep lab, referring physicians need to be cognizant of basic sleep testing requirements that pediatric sleep experts would recommend as minimum standards for diagnosing children in either pediatric or “mixed” labs. Furthermore, not only does family- and child-centered care increase child and family satisfaction, but also better-quality PSG data increases the likelihood of more accurate medical decision making. Minimal standards include the following:

- **Required knowledge base** for both technical and medical staff regarding pediatric respiratory and neurophysiology, normal developmental changes in sleep architecture, and cognitive/motor/language/social developmental milestones.
- **Technical skills** in conducting and scoring pediatric sleep studies. These necessitate initial specialized training, ongoing education, and exposure to an adequate volume of pediatric patients.
- **Family-centered care** is a mandatory component of pediatric sleep diagnostic and treatment services and requires specific accommodations to the:
  - **Emotional and physical needs of children** across a range of ages and their caregivers.
  - **Implementation of pediatric-specific procedures** to insure safety, including child-proofing of sleeping environment, development of age-appropriate restraint policies, and appropriate emergency equipment and emergency plan.
  - **Environment and physical space**, including comfortable and child-friendly accommodations (e.g., age-appropriate books, toys, pictures, linens) and comfortable accommodations for parents. The lab should have policies regarding number of accompanying family members and co-sleeping.
  - **Increased staffing ratios**, for younger patients and those with developmental delays. This often requires 1:1 staffing.
  - **Extended staffing hours** to accommodate longer sleep periods in children.
- **Appropriate triaging** of pediatric patients is fundamental to successful and safe integration of pediatric sleep services:
  - Appropriate patient acceptance and referral procedures
  - Sleep lab personnel, environment, and policies prepared to accommodate children in a wide range of clinical situations
  - Policies and procedures specific to caring for children with special needs (e.g., medical, psychiatric, neurodevelopmental issues), very young children, and adolescents. While careful triaging reduces many potential issues, it cannot eliminate them, and labs serving children must be prepared to handle the exceptional child.

- **Comprehensive clinical care** includes follow-up care for pediatric sleep patients undergoing diagnostic procedures. Evaluation and management of children with the full range of sleep disorders is a necessary component of an integrated sleep medicine program.

### WHAT THE SLEEP LAB STAFF AND INTERPRETING PHYSICIAN NEED TO KNOW

When referring a pediatric patient for an overnight sleep study, there is key information that should be provided. A sample sleep study request form is included in Appendix B3 as an illustration of the type of information needed to conduct, score, and interpret a sleep study.

- Demographics, contact information (including multiple phone numbers and email), insurance
- Confirmation of test(s) requested (e.g., PSG, PAP titration, MSLT); if special diagnostic tests are required (e.g., tracheostomy plugged, split night (half baseline/half PAP titration, O₂ titration), there needs to be a detailed explanation of the rationale and explicit instructions (e.g., initiate PAP therapy if AHI > 10; initiate supplemental O₂ if O₂ < 89% for 5 minutes).
- Specific indication(s) for testing (especially for direct referrals) and presumed diagnosis
- Key presenting symptoms (e.g., snoring, breathing pauses, restless sleep)
- Additional sleep related symptoms (e.g., insomnia, delayed sleep phase, parasomnias, enuresis)
- Key risk factors for presumed diagnosis (e.g., adenotonsillar hypertrophy, allergies/asthma, obesity, family history of OSA)
- Previous interventions (e.g., adenotonsillectomy, PAP therapy)
- Results (and preferably copies) of previous PSGs
- Child's usual sleep-wake schedule
- Medical, psychiatric, developmental, and behavioral comorbidities
- Medications, including hypnotics and over-the-counter
- What the family has been told (anticipatory guidance)
- Physical examination; pertinent findings (e.g., BMI, blood pressure, tonsillar size, and Mallampati grading)

### What Is Involved in “Reading” a Sleep Study?

Many referring physicians have a limited understanding of what the process of reviewing a sleep study and generating a report entails, and assume this is analogous to “reading” an X-ray or interpreting a lab finding. In reality, the process is very time-consuming and involves the following steps:
**Scoring:** A trained sleep technologist reviews the entire sleep study in 30-second “epochs” (e.g., for the “average” pediatric study this means 10 hours = 600 minutes = 1,200 epochs) *three separate times*: (1) the first pass is to score sleep stages and arousals and identify abnormal EEG findings, (2) the second is to score respiratory events, and (3) the third to score limb movement and EKG abnormalities. This process typically takes about 3 hours, but may be considerably longer in more complex, severe (e.g., high AHI), or longer duration (e.g., infants) studies.

**Interpretation:** The interpreting sleep professional then reviews the entire scored study regarding all three parameters: (1) sleep staging and arousals, (2) respiratory events, and (3) movement/cardiac events, *in 30-second epochs*. This typically takes about an hour but may require considerably more time for the more complex, severe, or longer duration studies.

**Generate sleep study report:** This review of the scored study is combined with the available clinical information regarding presenting symptoms, risk factors, and other pertinent information to generate a sleep study report that summarizes and synthesizes the accumulated sleep data to provide the referring practitioner with clinically useful data that will guide management decisions.

**DECONSTRUCTING A SLEEP STUDY REPORT**

While sleep study reports/interpretations from different labs are often somewhat variable in regard to the specific information and level of detail included and the types of recommendations made by the interpreting physician as to treatment options, the referring practitioner needs to have a basic understanding of the typical features provided in the sleep study report to make the best clinical use of the information presented. In order to facilitate this process, a deconstruction of a “typical” sleep study report is presented below:
BASELINE POLYSOMNOGRAPHY REPORT (PEDIATRIC)

Signals recorded: Bilateral paraspiinaal EEG and eye movements (EOG), EMG of the chin, legs, and intercostal muscles, EKG, thoracoabdominal respiratory inductance plethysmography effort measurements, airflow using thermistor, pressure, ETCO, and stimulation, snore vibration sensor, oxygen-globin saturations, and heart rate with pulse waveform. Audio/video monitoring was utilized. This was a Type I attended study. The study was recorded on a SPECIFIC TYPE PSG data acquisition system.

Methodology: Sleep staging, EEG arousals, awakenings, respiratory events, and limb movements were scored according to The AASM Manual for the Scoring of Sleep and Associated Events, v. 2.0, 2012. Pediatric criteria were applied to patients less than 18 years of age.

Patient Information

Name: Study Date: Age:
Date of Birth: Sex:
Medical Records No: Weight:
Study No: Height:
Referred By: Body Mass Index: BMI%
Primary Care Physician: Interpreting Physician:
Scoring Tech Initials:
Start Time: 8:57:33 p.m. Lights Off Time: 8:59:33 p.m.
End Time: 5:27:31 a.m.

Reason for referral: The patient is a 4-year 5-month-old female who presents with a history of loud snoring, choking/gasping arousals, observed apneas in sleep, mouth breathing, and restless sleep. Risk factors for sleep-disordered breathing included mild environmental allergies, mild asthma, and 3+ tonsillar

An attended study is critical for the pediatric patient, as the sleep technologist is the "eyes and ears" during the study for the scoring technologist and the interpreting practitioner. Tech observations and documentation are absolutely essential to an accurate picture of what transpired during the study; for example, notation of observations ranging from technical difficulties in conducting the study to patient compliance challenges, caregiver co-sleeping or snoring, and occurrence of parasomnias (to name a few) is key to producing a clinically valid and useful interpretation. While currently the vast majority of pediatric sleep studies are in-lab, attended (i.e., by sleep technologists) full night PSGs, this is not the case in adult sleep labs, which have increasingly made the switch to in-home, unattended, overnight portable monitoring for adult sleep patients. It is possible that this trend will also impact pediatric sleep studies in the future.
Study Summary

Medications Reported: PKN albuterol inhaler

Details: Over the technica quality of the study was good. The patient slept in the supine position for 26.4% of the total sleep time (TST) with 3.9% REM sleep while supine.

Sleep Staging and Architecture: The Lights Out time was 8:59:33 p.m. to Lights On at 5:27:03 a.m., with a total recording time of 508:00 minutes, and a TST of 473:00 minutes, which is considered adequate for this diagnostic study.

Sleep onset occurred in 14.0 minutes indicating a normal sleep-onset latency. There were 16 (115 seconds of Wake) full awakenings from sleep. Wake time after sleep onset was 20.5 minutes. Sleep efficiency was appropriate at 93%. Sleep Staging revealed a somewhat reduced percentage of REM sleep of 15% for the patient's age and normal REM sleep latency of 107.5 minutes. The total percentage of NREM stage 3 sleep was 36% and was felt to be appropriate for this age group. The caregiver reported that the patient's sleep was similar to what is observed at home except that she was more restless.

Overall, sleep architecture was appropriate for age. The waking record showed a 94Hz well-formed background rhythm. All sleep stages were present and well-formed with good spindle and vertex activity. There were 40 total arousals for the night yielding an arousal index (AI) of 5.1/hour (1.3/hour following respiratory events; 0.8/hour following limb or body movements; and 3.3/hour which were spontaneous). The normal number of arousals for children in this age group is 9 per hour for 3 to 6 y.o.

EEG

There was no epileptiform activity noted in the sleep montage employed.

Respiratory Findings: During sleep, the respiratory rate ranged from 18 to 20 breaths/minute in NREM and 18 to 22 breaths/minute in REM. There were 30 total sleep related respiratory disturbances yielding an IRD of 4.4/hour (includes all apneas, hypopneas, and RERAs). The AH1 (includes all respiratory events except RERAs) was 3.8. No snoring was heard across the night and no paradoxical or out-of-phase breathing was noted. The caregiver noted that the patient's breathing was much quieter than usual and that she usually snored at home.

(continued)

It is critical to know what medications the patient is taking for several reasons; some drugs impact sleep (e.g., SOT1 suppresses slow-wave sleep) and/or wakefulness and/or ventilator control (e.g., may be respiratory depressants). In making recommendations for treatment, the interpreting practitioner needs to know what medications the patient is already on (e.g., fluticasone, montelukast).

Comments are included here that pertain to any technical difficulties encountered during the study which could impact its results; for example, the patient had difficulty tolerating nasal cannulae with resultant flow limitations or ETCO2 monitoring was inconsistent.

Since OSA is typically worse in the supine position and REM sleep, it is critical to know if these parameters were adequately represented during the study.

Typically, at least 6 hours of recorded sleep is considered adequate data collection.

While sleep-onset latency is often prolonged due to the patient's unfamiliarity with the sleep laboratory environment and thus may not reflect "usual" sleep, it may be a useful parameter to note. For example, patients with narcolepsy usually have a very short sleep latency on the PSG, as well as an abnormality onset latency to REM sleep.

Sleep efficiency is equal to TST divided by time in bed. It is a general measure of sleep consolidation.

An increased percentage of N1 sleep for age may be a reflection of chronic insomnia sleep.
There were 14 obstructive events for the night, yielding an obstructive index of 1.2/hour. Of these events, there were 0 (0.0/hour) obstructive apneas, 14 hypopneas (1.8/hour) and 7 RURAs (0.6/hour) which were events due to increased respiratory effort associated with disruption of sleep architecture despite a mild (>3%) SpO₂ desaturation. For the night the AI associated with all obstructive events was 0.6/hour.

There were five obstructive events in REM (3.3/hour) and nine in NREM (1.4/hour), yielding an S₉₅O₂ of 99% in the (99%-100%) percent range and an AI from a baseline 0% to 1% baseline of 0% to 1% range with a SpO₂ nadir of 99% associated with obstructive events. Of the obstructive events in REM sleep, there were 5.7/hour in the supine position and 5.6/hour in the prone position. Of the obstructive events in NREM, there were 5.6/hour in the supine position and 5.1/hour in the prone position. The obstructive events lasted 9.4 to 17.0 seconds in NREM and 10.0 to 18.9 seconds in REM sleep.

All apnea events were scored as either lasting ≥20 seconds or lasting two or more breaths, or both, and yielding ≥3% SpO₂ desaturation, or an arousal from sleep. There were 14 total events, 3.8/hour (nine events, 1.4/hour in NREM; five events, 3.8/hour in REM sleep), yielding a nadir SpO₂ associated with central apneas in NREM sleep of 92% and in REM sleep of 90%. The central events lasted 9.2 to 14.4 seconds in NREM and 9.0 to 21.1 seconds in REM sleep. Most of these events followed cortical arousals and/or an increase in tidal volume./text

Oxygenation and ETCO₂: Baseline arterial oxygen saturation (SpO₂) during sleep was normal, ranging from 95% to 100%. During sleep, SpO₂ ranged from 81 associated with obstructive to 100%, after movement artifact was excluded. Mean arterial oxygen saturation was 98% during sleep SpO₂, and was <90% for 0.2% of TST and 99% to 100% during 1.0 minutes, or 99.4% of TST. For the night, the index of desaturations >3% was 3.6/hour, 1.4/hour in REM sleep and 2.7/hour in NREM sleep.

End-tidal carbon dioxide (ETCO₂) levels were 45 to 50 mm Hg, and 50 to 55 mm Hg during 1% of TST, 50 to 55 mm Hg during 1% of TST, 5 and 55 mm Hg during 5% of TST. ETCO₂ averaged 42.4 in REM sleep and 38.1 in NREM sleep.

Limb Movement Findings: For the night, there were 39 total limb movements, yielding a limb movement index of 4.5/hour. Of these, there were 0 (0.0/hour) events lasting ≥20 seconds or lasting two or more breaths, or both, and yielding ≥3% SpO₂ desaturation, or an arousal from sleep.

Age on the night of the study and a slightly elevated AHI in REM, so it is possible that these results underestimated her typical degree of sleep-disordered breathing at home; her mother also reported that her breathing and snoring were "better than usual." She also had a slightly elevated central apnea index, with most events following arousals or body movements without desaturations; central events in this type of situation may also occur in conjunction with obstructive events.

Assessment for possible surgical intervention could be considered, especially given her tonsillar hypertrophy and history of recurrent tonsillitis; alternatively, a trial of a nasal steroid and a leukotriene esterase inhibitor could be considered. Allergy-proofing in the home environment may also be helpful in reducing exposure to allergens.

**Final Diagnosis: Mild OSA 327.23**

It has been my pleasure to interpret this polysomnogram. Please do not hesitate to contact us for any questions about this study.

**Disclaimer:** All treatment decisions should be based on the clinical presentation of the patient and the reason for the referral. These results should be viewed with respect to the full clinical presentation of the patient.

**Interpreting Physician:**

Signature: ___________________________ Date: ___________________________

Date results were sent to all providers: ____________ by (initials) __________

The graphic below is a hypnogram, which provides a capsule summary of the sleep architecture throughout the study night.

![Hypnogram](image-url)
Basic good sleep habits, or positive sleep practices (also known as “sleep hygiene”), are essential for healthy sleep. Unfortunately, poor sleep practices are very common and often contribute to inadequate and disrupted sleep. For example, while poor sleep habits can contribute significantly to the development of insomnia (see Chapter 19), patients with preexisting insomnia frequently engage in maladaptive sleep practices in an attempt to address their sleep problems.

BEST SLEEP PRACTICES
Healthy sleep practices include four categories of sleep related behaviors (handouts for families on positive sleep practices for children and adolescents are provided in Appendices C7 and C8):

Practices that Promote Sleep Regulation (Circadian Rhythms and Sleep Drive)

- **Maintain an organized and consistent sleep-wake pattern** to help regulate the internal clock and synchronize the sleep-wake cycle.
- **Set and enforce a consistent bedtime** (i.e., no more than an hour different) on weekdays and weekends.
- **Set and enforce a consistent wake time** on weekdays and weekends (regardless of the prior night's sleep). It is preferable for wake times to be no more than an hour different, but this is not realistic for adolescents who have to get up for very early high school times. In these cases, wake time should be no later than 9:00 to 10:00 a.m.
- **Keep a regular daily schedule** of activities, including meals.
- **Avoid evening direct light exposure** (particularly light shining directly into the eyes) in the bedroom at bedtime and during the night, as even relatively dim light may suppress melatonin (see below). However, dim nightlights that do not shine directly into the eyes are fine and some children find them comforting.
- **Increase light exposure in the morning** (to suppress melatonin and increase alertness). Getting daily exposure to the sun, especially in the morning, will help regulate the internal clock.
- **Establish an age-appropriate napping schedule** (see Chapter 2); for example, late daytime napping may not provide adequate time for sufficient buildup of the sleep homeostatic drive before bedtime and thus result in delayed sleep onset. See below for a discussion of “strategic napping.”

Practices that Promote Sleep Conditions

- **Establish a regular and consistent bedtime routine.** A bedtime routine should involve the same three to four activities every night in the same order, and the activities should be calming and relaxing. Appropriate bedtime activities include taking a bath or shower, reading or being read to, singing lullabies, listening to soft music, and discussing the child’s day. Younger and developmentally delayed children may benefit from having a pictorial representation of the bedtime routine activities (Figures 5.1, 5.2, 5.3, 5.4).

A recent study found that a consistent bedtime routine was associated with better sleep outcomes, including earlier bedtimes, shorter sleep-onset latency, reduced night wakings, and increased sleep duration. Decreased parent-perceived sleep problems and daytime behavior problems were also related to institution of a regular bedtime routine. Furthermore, there was a dose-dependent relationship, with better outcomes associated with increased “doses” of having a bedtime routine.
FIG 5.3. Pajamas
FIG 5.1. Bathtime
FIG 5.2. Brush teeth
Limit activities that promote wakefulness while in bed, including texting, gaming, and watching TV.

Don't use bed for punishment (time out). Also avoid using sleep as punishment ("if you don't listen to me, you'll have to go to bed right after supper") and staying up as a reward for good behavior. This gives the subtle message that “sleep is bad.”

Avoid sleeping in environments other than the bedroom, such as the couch or in the car.

Practices that Reduce Arousal and Promote Relaxation

Keep electronics, especially “screens,” out of the bedroom and limit use of electronics before bedtime (see below).

Reduce stimulating play at bedtime. Playing can increase arousal and make it difficult to fall asleep.

Avoid heavy meals within an hour or two of bedtime, as this may interfere with sleep onset. However, children should not go to bed hungry; thus, having a light healthy snack (e.g., peanut butter crackers and milk, apple slices) before bed can be beneficial.

Reduce cognitive and emotional stimulation before bedtime. Feeling stressed or excited, or engaging in any
demanding or stimulating mental activities (e.g., playing video games), can result in difficulty initiating sleep.

- Limit or eliminate caffeine consumption (see below).
- Include activities in the bedtime routine that are relaxing and calming, such as reading.

**Practices that Promote Adequate Sleep Quantity and Quality**

- **Set an age-appropriate bedtime and wake time to ensure adequate opportunity for** sleep. For example, studies have suggested that a bedtime later than 9:00 p.m. for younger children (less than age 10 years) was associated with shorter sleep duration. A sleep schedule that factors in general recommendations for sleep duration by age, individual sleep need, and lifestyle issues should be developed with the child or adolescent and the parents.

- **Maintain a safe and comfortable sleeping environment**, including low noise and light levels, cooler temperatures, and age-appropriate bedding and sleep surfaces.

**Individual Sleep Needs**

- Individual sleep needs are dependent on a number of factors, including age. Research also indicates that tolerance for insufficient sleep appears to vary across individuals and may in fact be genetically determined. That is, some individuals appear to function better or worse following sleep loss than do others. Although the general profile of performance deficits following sleep loss is fairly consistent in human beings (e.g., compromise of efficiency, deterioration of performance over time), there may be individual variation in relative susceptibility to the effects of inadequate sleep across domains (e.g., memory, attention, mood).

**FACTORS THAT IMPACT SLEEP**

While many of these sleep practices are based on common sense and “conventional wisdom” rather than empirical evidence per se, especially for the pediatric population, several have been the focus of considerable research (caffeine consumption, electronic media use, and napping) and are discussed in more detail here.

**The “Red Bull Generation”: Caffeine and Other Stimulants**

**Caffeine.** Wake-promoting agents such as caffeine and the related compounds, theophylline and theobromine (which are also found in varying amounts in beverages such as coffee, tea, and colas), stimulate alerting systems and slow sleep-promoting systems in the CNS. Neurotransmitters that are specifically impacted by caffeine include adenosine and GABA. Caffeine acts as a functional adenosine receptor antagonist, resulting in decreased sleepiness and increased subjective and objective alertness. Related CNS effects and the presence of these receptors in peripheral tissues (e.g., blood vessels, kidneys, heart, gastrointestinal system) help to explain some of the common direct side effects associated with caffeine, including “jitteriness,” anxiety, increased heart rate and resting/exercise blood pressure, headache, and abdominal discomfort.

However, the evidence for wake-promoting effects of caffeine in the pediatric population is quite limited. Although caffeine reduces some of the deficits associated with sleep loss, such as reaction time, it appears to have little in the way of cognitive-enhancing effects. Its impact is also temporary and limited. For example, caffeine does not counteract the effects of alcohol.

Development of complete or partial tolerance to the central effects, including temporarily increased alertness levels, is common, resulting in higher consumption levels to achieve similar results. Moreover, caffeine is not a
substitute for sufficient sleep, nor does it confer the same physiologic and mental benefits.

In addition, at least partially due to blockade of GABA receptors, caffeine is associated with negative effects on subsequent nocturnal sleep. Because it has an elimination half-life of approximately 3 to 7 hours in adults (the duration of action is extended at higher intake levels), evening and even afternoon consumption can have detrimental effects on sleep. Caffeine intake has been consistently associated with shorter sleep duration, increased sleep-onset latency, increased wake time after sleep onset, and increased morning fatigue and daytime sleepiness. The degree of impact on sleep is dose-dependent, and sensitivity to these effects likely varies across individuals. On the other hand, gradual cessation of caffeine consumption (as opposed to abrupt withdrawal, which especially in habitual consumers can lead to rebound insomnia) may result in improved sleep quality and duration.

Side effects of caffeine are also related to dependency and symptoms of withdrawal. Caffeine withdrawal effects in heavy users include sleepiness/fatigue, dysphoria/depression/irritability, poor concentration, and somatic complaints such as headache, nausea/vomiting, and muscle aches. These effects typically occur 12 to 24 hours after cessation, peak at 20 to 48 hours, and may last up to 1 week. These effects occur in youth too, especially adolescents who may be heavy users. Caffeine also may exacerbate symptoms of restless legs syndrome and periodic limb movements, as well as acts as a diuretic, further compromising sleep quality. Finally, there has been an alarming trend for caffeine to be increasingly involved in toxic ingestions in the pediatric population; many of these ingestions involved OTC medications containing caffeine (e.g., "Now-Doz") and energy drinks. Many easily accessible beverages and products contain caffeine, including a number of over-the-counter cold remedies, pain relievers, and weight loss preparations. There are a number of herbal stimulants that also contain caffeine as the active ingredient. However, because many beverages containing caffeine do not list the amount on the label, parents and providers may need to conduct some "detective work" to accurately assess daily caffeine consumption. Table 5.1 provides a list of common caffeinated products. In addition, a wide range of other caffeinated "vehicles" have been developed, including caffeinated water, syrups, tablets, candy, gum, and inhalable "shots," as well as novelty food and cosmetic products such as caffeinated beef jerky, cookies, and lip balm. These are easily available commercially and on the internet (e.g., at websites like "Thinkgeek.com").

Clinical Practice Recommendations: Caffeine

Screen

• Pediatric providers should routinely inquire about caffeine consumption (i.e., quantity, timing, type, and reason(s) for use) in children and adolescents. Screening should include not only beverage-related sources of caffeine such as sodas, coffee, and energy drinks, but also increasingly available alternative caffeinated products marketed as alertness-enhancers or “energy” supplements (e.g., over-the-counter medications, candy/gum).

Educate

• Education regarding caffeine use should be included in anticipatory guidance for school-aged children and adolescents.

• Key health education messages for caregivers and patients include the following:
  ■ Caffeine should be viewed as a psychoactive substance with stimulant properties and accompanying risks, rather than as a food substance or flavor additive.
  ■ Caffeine is not a substitute for sufficient sleep. While caffeine may temporarily partially mitigate the impact of sleep loss, negative effects on sleep associated with caffeine consumption (i.e., shortened sleep duration, increased sleep-onset latency, increased waketime during
the night, and earlier rise times) result in increased daytime sleepiness.

- Other consequences associated with caffeine use include increased anxiety, cardiovascular effects (e.g., increased blood pressure), and withdrawal symptoms (headache, fatigue, mood changes, poor concentration).

- The practice of mixing caffeine and alcohol in older adolescents and college students is increasingly common and highly dangerous, masking the perception of impairment without mitigating the consequences. This behavior is associated with higher likelihood of toxicity and overdose, increased rates of binge drinking, and increased injury risk.

### TABLE 5.1. Caffeine Intake

<table>
<thead>
<tr>
<th>Product</th>
<th>Serving Size</th>
<th>Caffeine Content (per serving)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sodas and Energy Drinks</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rockstar Energy</td>
<td>16 oz</td>
<td>160</td>
</tr>
<tr>
<td>Red Bull</td>
<td>8.4 oz</td>
<td>80</td>
</tr>
<tr>
<td>Vault Zero*</td>
<td>12 oz</td>
<td>74</td>
</tr>
<tr>
<td>Diet SunDrop*</td>
<td>12 oz</td>
<td>72</td>
</tr>
<tr>
<td>Diet Mountain Dew*</td>
<td>12 oz</td>
<td>55</td>
</tr>
<tr>
<td>Mountain Dew*</td>
<td>12 oz</td>
<td>55</td>
</tr>
<tr>
<td>Tab*</td>
<td>12 oz</td>
<td>48</td>
</tr>
<tr>
<td>Diet Coke*</td>
<td>12 oz</td>
<td>46</td>
</tr>
<tr>
<td>Diet Dr. Pepper*</td>
<td>12 oz</td>
<td>44</td>
</tr>
<tr>
<td>Dr. Pepper*</td>
<td>12 oz</td>
<td>43</td>
</tr>
<tr>
<td>Diet Sunkist*</td>
<td>12 oz</td>
<td>42</td>
</tr>
<tr>
<td>Sunkist*</td>
<td>12 oz</td>
<td>41</td>
</tr>
<tr>
<td>Pepsi*</td>
<td>12 oz</td>
<td>39</td>
</tr>
<tr>
<td>Beverage</td>
<td>Size</td>
<td>Calories</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------</td>
<td>----------</td>
</tr>
<tr>
<td>Diet Pepsi*</td>
<td>12 oz</td>
<td>37</td>
</tr>
<tr>
<td>Coca-Cola*</td>
<td>12 oz</td>
<td>34</td>
</tr>
<tr>
<td>Big Red*</td>
<td>12 oz</td>
<td>34</td>
</tr>
<tr>
<td>Barq's Root Beer*</td>
<td>12 oz</td>
<td>22</td>
</tr>
<tr>
<td>Yoo-hoo Chocolate</td>
<td>8 oz</td>
<td>0</td>
</tr>
<tr>
<td><strong>Coffee and Tea</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Starbucks coffee, tall</td>
<td>12 oz</td>
<td>260</td>
</tr>
<tr>
<td>Starbucks coffee, grande</td>
<td>16 oz</td>
<td>330</td>
</tr>
<tr>
<td>Dunkin Donuts coffee</td>
<td>14 oz</td>
<td>178</td>
</tr>
<tr>
<td>Arizona Green Tea Energy</td>
<td>16 oz</td>
<td>200</td>
</tr>
<tr>
<td>McDonald's coffee</td>
<td>12 oz</td>
<td>109</td>
</tr>
<tr>
<td>Tazo chai</td>
<td>16 oz</td>
<td>95</td>
</tr>
<tr>
<td>Snapple iced tea (all kinds)</td>
<td>16 oz</td>
<td>62</td>
</tr>
<tr>
<td>Coffee, decaf</td>
<td>8 oz</td>
<td>5</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hershey's Special Dark Chocolate bar</td>
<td>1.5 oz</td>
<td>20</td>
</tr>
<tr>
<td>Hershey's Milk Chocolate bar</td>
<td>1.5 oz</td>
<td>9</td>
</tr>
<tr>
<td>Starbucks Hot Chocolate, grande</td>
<td>16 oz</td>
<td>25</td>
</tr>
<tr>
<td>Hershey's Lowfat Chocolate Milk</td>
<td>12 oz</td>
<td>2</td>
</tr>
<tr>
<td><strong>Frozen Desserts</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ben &amp; Jerry's Coffee Heath Bar</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crunch</td>
<td>4 oz</td>
<td>42</td>
</tr>
</tbody>
</table>
The availability of highly caffeinated products, such as energy drinks, has increased exposure to these beverages in children and adolescents. For example, recent data suggest that energy drink consumption is on the rise, more than doubling in children aged 2 to 11 years and tripling in adolescents (from 4%-12%). Furthermore, energy drinks have replaced soda and coffee as major sources of caffeine for many children and adolescents. Moreover, there appears to be an increasing trend for these products to be consumed by older children and adolescents not only for their alleged positive effect on mood and performance but also for their perceived role as a countermeasure to sleepiness and fatigue. However, the scientific evidence supporting the effectiveness of these beverages, including energy drinks, in enhancing alertness, mood, and performance, specifically in the pediatric population, is still quite limited.

Finally, and not surprisingly, increased caffeine use frequently coexists with other behaviors that negatively affect sleep, such as late-night use of electronics, later bedtimes, and greater discrepancy in weekday-weekend sleep schedules. Caffeine consumption is also linked to nicotine use in adolescents, which in turn may further disrupt sleep and perpetuate the cycle of sleep fragmentation daytime sleepiness and stimulant use.

**Nicotine.** Cigarette smoking is associated with increased sleep-onset latency and disrupted nonrestorative sleep. It also increases restless legs symptoms and increases the risk of sleep-disordered breathing. Furthermore, withdrawal from nicotine may result in disrupted sleep and daytime somnolence.

**Plugged In and Wired Up: Electronics and Sleep**
Watching television and using other electronics is one of the most common unhealthy sleep practices found in children and adolescents. The National Sleep Foundation (NSF) 2006 poll, for example, reported that 97% of adolescents have at least one electronic media device in their bedroom (music players: 90%, televisions: 57%, video game consoles: 43%, mobile: 42% or fixed-line telephones: 34%, computers: 28%, and internet access: 21%). Primary school children have also been reported
to commonly have bedroom devices such as televisions (18%-43%), computers/video games (18%-20%), and mobile phones (14%).

Clinical Practice Recommendations: Sleep and Electronic Media Screen

- Pediatric providers should question adolescents with insufficient sleep about the use of electronics including computers, mobile phones, gaming systems, MP3 players, and televisions in the hours before bedtime.

Educate

- Parents and adolescents should be educated about the relationship between the use of electronics in the evening hours and insufficient sleep.
- In an attempt to optimize sleep, education should be directed at convincing adolescents and their parents of the advantages of avoiding the use of and exposure to these devices in the hour before bedtime.
- Education should be further directed of keeping electronic devices out of the bedroom.

In the NSF poll, adolescents with less than four media devices in the bedroom got significantly less sleep on both school nights and nonschool nights, were more likely to have fallen asleep at school or while doing homework, and drank more caffeinated beverages during the day. On the other hand, adolescents who reported getting more than 8 hours of sleep per night engaged in less technology-related activities after 9:00 p.m. compared with adolescents who report getting less sleep. Another recent study found that children who sleep with a small screen (e.g., smartphone) in their bed or next to their bed get 20 minutes less sleep per night. Furthermore, children who sleep in a bedroom with a television get 18 minutes less sleep.

In terms of specific devices in the bedroom, TV is associated with delayed bedtimes, shorter total sleep time, increased bedtime resistance, and higher overall level of sleep disturbance; computers or electronic gaming with later bedtimes, delayed sleep onset, shorter total sleep time, and increased prevalence of disorders of bedtime resistance; and a phone with delayed bedtime on weekend days and increased variability in weekday-weekend bedtimes; in addition, adolescents who reported being woken up at least occasionally by text messages reported being significantly more tired.

The type, amount, and timing of electronic device use are also important to consider in regard to sleep problems in children. For example, increased amounts of television exposure during the day, including both viewing and just having the television switched on, is associated with sleep problems such as bedtime resistance and delayed sleep onset and decreased sleep duration. As noted earlier, watching television and computer use after 9:00 p.m. in particular has been associated with shorter sleep duration, increased daytime sleepiness, and a generally higher frequency of sleep problems. Furthermore, self-described use of TV as “sleep aid” is also associated with increased sleep-onset latency and reduced total sleep time. Increased mobile phone use during the day has also been associated in some (although not all) studies with a negative impact on sleep and increased daytime sleepiness. Note that smartphone use may not only delay bedtimes, but sleep is also disrupted by middle-of-the-night texts and other notifications. More than a third of adolescents report being awakened by text messaging at least once a month. Finally, there is some evidence that the content of the viewed material (e.g., violent versus nonviolent video games) may impact sleep differentially.

There are a number of proposed potential mechanisms regarding the link between electronic media use and sleep disruption:

- **Media use directly displaces sleep**, as more time watching TV (playing on the computer, texting) translates
Media use replaces good sleep and health practices, as electronic use at bedtime supersedes a regular relaxing bedtime routine or supplants healthier behaviors during the day (e.g., physical activity).

Media use results in increased mental/emotional/physiological arousal, making it more difficult to initiate and maintain sleep.

Direct screen light (especially blue light) exposure suppresses melatonin and results in delayed sleep onset, and thus there are physiological effects of electronics on sleep mechanisms.

Electromagnetic radiation cell phones delay melatonin onset and alter sleep architecture.

**Recommendations Regarding Electronic Media Use and Sleep**

- No media devices (including cell phones/smartphones) in the bedroom, especially after lights out.
- Limit viewing/use to 2 hours/day.
- Avoid evening use of screens (after 9:00 p.m.).
- Reduce exposure to violent content.
- Do not use electronics as a sleep aid.

**Napping 101: Pros and Cons**

While Chapter 2 details developmentally appropriate napping patterns in young children, this section addresses the utility of “strategic” or planned naps in older children and adolescents. It should be acknowledged that while there may be important racial/ethnic and culturally based differences in children's daytime sleep patterns (e.g., school-aged African American children are more likely to nap compared to Caucasian children, but total 24-hour sleep duration is similar), the concept of napping as a countermeasure to chronic insufficient sleep may be a useful one as long as the limitations are acknowledged.

In adults, numerous studies have demonstrated the benefits of napping either prior to anticipated sleep loss or “on the job” as a means of improving alertness and enhancing cognitive function (including memory retention) and performance. However, the timing and duration of naps are key variables in determining utility. A recent review concluded that a nap (for as short as 10 minutes) during the afternoon restores wakefulness and promotes performance and learning. In particular, naps of less than 30 minutes duration have been shown to confer a number of benefits, including enhanced wakefulness, whereas longer naps may be associated with sleep inertia (see below) and a potential loss of productivity. Furthermore, recent epidemiological studies indicate that more frequent and longer naps may lead to adverse long-term health effects.

The rationale for optimal timing and duration of naps is as follows:

**Timing.** While naps in the early afternoon appear to confer the most benefit in regard to cognitive function, napping in the mid to late afternoon is often more practical (especially for students) and takes advantage of the “circadian trough” of alertness that typically occurs between 3:00 and 5:00 p.m. and results in increased sleep propensity.

**Duration.** During a short (i.e., <30 minutes) nap, entry into slow-wave sleep (SWS) is less likely; a forced awakening out of SWS is more likely to result in “sleep inertia,” the groggy, irritable confused state characterized by prolonged difficulty in becoming alert (also known in its severe form as “sleep drunkenness”), which may last up to 15 to 30 minutes. The latter phenomenon may account for the common perception that “I
don't nap because I just feel worse after I do." The effects of sleep inertia may be relatively more pronounced for more complex tasks (i.e., those involving executive functions). The benefits of even a very brief nap of 5 to 15 minutes are almost immediate after the nap but last for a limited period (1-3 hours). In contrast, the cognitive and memory benefits of planned longer naps (>30 minutes), once sleep inertia is dissipated, produce improved cognitive performance for a more sustained period (up to many hours). Interestingly, individuals who are “habitual” nappers may benefit more than “occasional” nappers, implying there may be a "practice effect."

**Naps**

- The bottom line is that short planned naps can immediately restore alertness for a relatively short period of time, whereas longer naps, when the opportunity arises, may have a more lasting effect on cognitive function. Therefore, scheduled naps can provide a temporary respite from daytime sleepiness and compromised alertness when sufficient nocturnal sleep is not achievable on a regular basis, and may be a preferable strategy to attempting to “catch up” on sleep when given the opportunity (i.e., on weekends).
The following algorithms present a clinical framework for evaluating three of the most commonly presenting sleep complaints in pediatric patients (bedtime resistance or prolonged sleep onset, nightwakings, and daytime sleepiness in adolescents) (Figures 6.1, 6.2, and 6.3). The major diagnostic categories to consider for each presenting complaint are printed in bold; important differentiating features are outlined in italics; and suggested diagnostic tests are underlined. It is important to keep in mind that there may be more than one etiology for a sleep complaint in any given child or adolescent; thus, several diagnostic categories may need to be considered. In addition, children may present with more than one sleep complaint (e.g., delayed sleep onset and nightwakings). Finally, these algorithms represent only a guideline for clinical evaluation. Specific assessment, diagnostic, and treatment recommendations for each disorder are found within the individual chapters.
FIG 6.1. Bedtime resistance/prolonged sleep onset. ADHD, attention-deficit/hyperactivity disorder; FH, family history.

FIG 6.2. Nightwakings. Note: The etiology of sleep disturbances in children is frequently multifactorial. Therefore, any evaluation of nightwakings should take into consideration all of the factors listed above. PSG, polysomnography; MSLT, multiple sleep latency test; EEG, electroencephalogram; ADHD, attention-deficit/hyperactivity disorder.
FIG 6.3. Daytime sleepiness (adolescents). PSG, polysomnography; MSLT, multiple sleep latency test.
Bedtime problems, including stalling and refusing to go to bed, are often the result of parental difficulties in setting limits and managing behavior (note: for a discussion of sleep onset problems in older children and adolescents, see Chapter 19 on Insomnia). Sleep disturbances of this type were previously referred to as behavioral insomnia of childhood, limit-setting type, but now fall under the diagnostic category of insomnia. These sleep problems typically begin after the age of 2 years, when children are either able to climb out of the crib or have been moved to a bed. Bedtime stalling behaviors include attempts to delay bedtime (e.g., by watching additional television) and the bedtime routine (e.g., by reading another story) or occur following lights out (“curtain calls,” such as requests for a drink of water or an additional hug). Typically, the requests are based on what the child has learned will elicit a parental response, for example, a trip to the bathroom or being “afraid” of monsters under the bed. Bedtime refusal includes behaviors such as the child refusing to get ready for bed or to remain in the bed or the bedroom after lights out, “curtain calls,” and following the parent(s) back to the living room or other rooms of the house.

**Bedtime Problems: Limit-setting Issues**

Bedtime problems are often characterized by the inadequate enforcement of bedtime limits by a parent or caregiver, resulting in the child stalling or refusing to go to bed at an appropriate time. When limits are not set and enforced, or are enforced only sporadically, sleep will be delayed, and total sleep may not be enough to meet the child's sleep needs.

Another type of limit-setting problem occurs when there are few or no limits instituted by parents around sleep behaviors; examples include allowing the child to fall asleep while watching television in the living room and allowing an older child to play computer games in his or her bedroom until lights out. Parents may also institute limits in an unpredictable or inconsistent way (e.g., intermittently allowing the child to fall asleep in the parents’ bed). This type of inconsistent parental response provides intermittent reinforcement, which maintains the child's inappropriate behavior. Limit-setting sleep problems may also affect parental response to nightwakings, as in the case of the child who is allowed to join the parents in their bed during the night. Parental limit-setting is also likely to be the problem if the child goes to bed and falls asleep quickly for other caregivers (e.g., babysitter, other parent) or has no difficulty falling asleep at the desired bedtime in other situations (e.g., allowed to stay in living room and falls asleep on couch watching television).

The environment may also contribute to limit-setting problems. For example, limits may be difficult to set in homes in which the child shares a bedroom with his or her parents or siblings or in which a grandparent resides. Parents of children with a current or past history of medical problems may also have difficulty setting limits because of guilt, a sense that the child is “vulnerable,” or concerns about doing psychological harm (see Chapter 22 for more information on children with medical problems).

**Impact on the Family**

Successful behavioral intervention for bedtime resistance will not only result in improvement in the child's sleep problems but has also been shown to alleviate parental stress and improve parental sleep. Implementation of behavioral management strategies for sleep may also lead to improvements in parenting skills and management of daytime behaviors.
EPIDEMIOLOGY

Bedtime resistance, found in 10% to 30% of toddlers and preschoolers, is one of the most common concerns of parents and may coexist with nightwakings. Not only are bedtime problems common, but this behaviorally based sleep problem often becomes chronic; in one study, 84% of children aged 15 to 48 months with bedtime struggles and/or nightwaking continued to have significant sleep disturbance at the 3-year follow-up. School-aged children may also have significant limit-setting sleep issues; in one study, 15% of children aged 4 to 10 years had problematic bedtime resistance reported by parents.

ETIOLOGY AND RISK FACTORS

There are both precipitating and perpetuating factors that contribute to bedtime difficulties and include both extrinsic factors (e.g., environment, parental issues) and intrinsic factors (e.g., temperament, circadian rhythm).

- **Permissive parenting style**, in which there is minimal limit setting regarding most discipline issues. This ineffective parenting may be related to parenting style or may reflect caregiver problems, such as depression or substance use.

- **Conflicting parental discipline styles**, especially when parents frequently disagree about how to handle behavioral issues. In this situation, one parent is often the authoritarian, whereas the other is more lenient (“good cop versus bad cop”). Children often sense (and sometimes exploit) this ambivalence, leading to increased noncompliant behavior.

- **Parent expectations**, which may contribute to bedtime issues. Parents may not have realistic expectations regarding sleep behaviors and normal developmental.

- **Age**, as toddlers and preschool-aged children are more likely to assert their independence and refuse to comply with parental requests. As children get older, they typically require less parental interaction at bedtime.

- **Temperament**, especially difficult and unpredictable temperament styles.

- **Oppositional behavior during the day**, which makes noncompliance at bedtime more likely.

- **Environments**, which inhibit limit setting, such as parents and child sharing a bedroom or living with grandparents.

- **Circadian timing**, which can also play a role in bedtime struggles. There is a normal circadian-mediated surge (i.e., peak) in alertness, often referred to as the “second wind” or “forbidden zone.” This phenomenon typically occurs during the hour or so before natural sleep onset. This increase in alertness counteracts the sleep drive that has built up over the course of the day and allows us to stay awake in the evening. If parents set a bedtime that happens to coincide with the second wind period (usually relatively early in the evening), a child may have significantly more difficulty settling, which can result in bedtime resistance. Children with an “owl” circadian preference for later sleep-onset and wake times also tend to have a later circadian alertness peak and are, thus, particularly likely to have a settling problem if bedtime is set too early. These children may also resist going to bed because they are not ready to fall asleep at the parent-determined bedtime. On the other hand, a late bedtime can lead to a child being overtired and taking longer to fall asleep. One study found that a bedtime after 9:00 p.m. resulted in children actually having a longer sleep-onset latency. These situations are not primarily caused by parental failure to set limits, but rather by a “mismatch” between parental expectations and biological rhythms; however, the resulting sleep-onset problems may be compounded by parental management.
PRESENTATION AND SYMPTOMS

- **Noncompliant behavior**, in response to parental requests to get ready for bed (e.g., change into pajamas, brush teeth).

- **Bedtime resistance**, including refusal to go to bed or requiring a parent to be present at bedtime.

- **“Curtain calls,”** characterized by frequent requests for parental attention (e.g., drinks, kisses) after lights out.

- **Delayed sleep onset**, of 30 minutes or more following lights out.

**Diagnostic Criteria**

See Table 7.1.

**Associated Features**

- **Nightwakings**: Many children with bedtime struggles also present with frequent nightwakings, resulting from similar limit-setting issues or as a result of negative sleep associations (e.g., parental presence required to fall asleep) that have developed at bedtime (see Chapter 8).

- **"Fearful behaviors":** Some children present with nighttime fears characterized by fearful behaviors (e.g., crying, clinging, leaving bedroom to seek parental reassurance) that are a manifestation of bedtime stalling rather than being anxiety based.

- **Evening stress**: For some families, the stress around bedtime battles may begin several hours before bed, even as early as dinnertime, in anticipation of the nightly bedtime battle.

- **Daytime behavior problems**: Daytime behavior problems appear to be more common in “poor sleepers.” This relationship may reflect an increase in behavior problems because of inadequate sleep or may be related to the fact that children with daytime behavior problems are more likely to have similar problems at night, or a combination of both.

### TABLE 7.1. Diagnostic Criteria: Chronic Insomnia Disorder

**A.** The patient reports, or the patient's parents or caregiver observes, one or more of the following:

1. Difficulty initiating sleep.
2. Difficulty maintaining sleep.
3. Waking up earlier than desired.
4. Resistance going to be on appropriate schedule.
5. Difficulty sleeping without parent or caregiver intervention.

**B.** The patient reports, or the patient's parents or caregiver observes, one or more of the following related to the nighttime sleep difficulty:

1. Fatigue/malaise.
2. Attention, concentration, or memory impairment.
3. Impaired social, family, occupational, or academic performance.
5. Daytime sleepiness.
6. Behavioral problems (e.g., hyperactivity, impulsivity, aggression).
7. Reduced motivation/energy/initiative.
8. Proneness for errors/accidents.
9. Concerns about or dissatisfaction with sleep.
C. The reported sleep-wake complaints cannot be explained purely by inadequate opportunity (i.e., enough time allotted for sleep) or inadequate circumstances (i.e., the environment is safe, dark, quiet, and comfortable) for sleep.
D. The sleep disturbance and associated daytime symptoms occur at least 3 times per week.
E. The sleep disturbance and associated daytime symptoms have been present for at least 3 months.
F. The sleep-wake difficulty is not better explained by another sleep disorder.


- **Family tension:** Bedtime resistance often results in significant family tension, including arguments between the child and the parents and marital discord. This tension may contribute to heightened arousal and anxiety in the child, making sleep onset even more difficult. Parents may also focus more attention on the child's behavior at bedtime to avoid dealing with underlying marital conflicts.

**EVALUATION**

- **Medical history:** Children with medical conditions may be more likely to have bedtime struggles, related to a number of physical (pain, medication) and behavioral issues (see Chapter 22); however, the history is typically benign.

- **Developmental history:** Developmentally delayed children, especially those with sensory integration issues, may have more difficulty with self-soothing at bedtime.

- **Family history:** The history should include an evaluation of parenting skills and limit-setting abilities.

- **Behavioral assessment:** A history of more global behavior problems, especially noncompliance and oppositional defiant disorder (ODD), may be present. Children with attention-deficit/hyperactivity disorder (ADHD) often have more difficulty settling at bedtime (see Chapter 21).

- **Physical examination:** Physical symptoms are usually noncontributory.

- **Diagnostic tests:** Such tests are not indicated.

**DIFFERENTIAL DIAGNOSIS**

Bedtime struggles may be the result of a more global problem with noncompliance, including ODD. In addition, alternative reasons for delayed sleep onset should be considered:

- **Inappropriate sleep schedules,** which may contribute to delayed sleep onset. Naps, including napping past 4:00 p.m. or continued napping in an older preschooler, may result in a later sleep onset. In addition, an irregular sleep schedule (inconsistent bedtime and waketime) may conflict with the circadian sleep-wake rhythm and contribute to difficulties in falling asleep. Similarly, significant discrepancies between weekday and weekend sleep schedules can result in problems falling asleep on Sunday evenings, especially if the child or adolescent is sleeping in late on weekend mornings. Finally, parents may not be attentive to the child's natural sleep-onset time, which may be later than the desired bedtime.
Delayed sleep phase, related to a “night-owl” tendency or, in some cases, delayed sleep-wake phase syndrome (see Chapter 18). In both situations, sleep onset occurs at a consistent time each night, and no problems are noted if bedtime is set closer to the usual fall-asleep time.

Nighttime fears and separation anxiety, in which anxiety is a significant component of the child's bedtime behavior. Typically, if the parent remains with an anxious child at bedtime or lets the child sleep in a room with another person present, resistance disappears and sleep-onset is not delayed.

Transient or “adjustment” insomnia, which is likely to present in children with previously normal sleep. Transient insomnia can be the result of sleeping in an unfamiliar or nonconducive (e.g., too noisy, too hot) sleep environment, stressful life event, disruption of sleep schedule (e.g., trip, jet lag), or illness.

Restless legs syndrome, which often presents with difficulty falling asleep at bedtime. Restless legs syndrome is characterized by an urge to move the legs accompanied by uncomfortable sensations that interfere with sleep onset (see Chapter 16).

Medication effects, including stimulant medications for ADHD, may result in delayed sleep onset (see Chapter 20 for more information on medication effects).

Mental health issues, including anxiety disorders (especially separation anxiety) and ritualistic behaviors associated with obsessive compulsive disorder, may delay bedtime and sleep onset.

MANAGEMENT

When to Refer
Children or adolescents with persistent or severe bedtime issues that are not responsive to simple behavioral measures or that are extremely disruptive should be referred to a mental health professional for evaluation and treatment. Collaboration with a behavioral therapist in designing and implementing treatment plans may be prudent (1) if there is anxiety; (2) if there are complex, chronic, or multiple sleep problems; or (3) if initial behavioral strategies have failed.

Treatment
Treatment for limit-setting sleep problems should include the institution of appropriate sleep habits, development of an appropriate sleep schedule that coincides with the child's natural circadian rhythm, and appropriate and consistent parental limit setting. A summary of standards of practice for bedtime problems and nightwakings can be found in Chapter 8.

Sleep Habits

Establish a set bedtime, one that coincides with the child's natural sleep-onset time. A consistent nightly bedtime will help to set the circadian clock and enable the child to fall asleep more easily. If, however, the attempted bedtime coincides with the late-day circadian peak in alertness, the likely result is bedtime resistance. In this case, delaying the bedtime or moving the bedtime earlier, when the child is more physiologically ready for sleep, may be warranted. With children who have an evening circadian preference, temporarily delaying the bedtime to coincide with the natural sleep-onset time and then gradually advancing the bedtime over a period of several weeks may be a reasonable approach (see discussion of bedtime fading, below).

Institute bedtime fading, which involves temporarily setting the bedtime at the current sleep-onset time and then gradually advancing it by short intervals (e.g., 15 minutes) until the desired bedtime is reached. This has the advantage of temporarily eliminating the struggles that occur between bedtime and sleep.
onset, thus reducing tension in the household. In addition, children usually view this procedure positively (being “allowed” to stay up later), which increases the likelihood of compliance with the rest of the behavioral program.

- **Evaluate daytime sleep habits**, particularly in older children, that may be interfering with sleep onset such as napping. Preschoolers often continue to require a daytime nap, but daytime sleep past 3:00 or 4:00 in the afternoon may interfere with an early bedtime. In these cases, bedtimes need to be moved later and parental expectations need to be adjusted. Caffeine consumption, particularly later in the day, may also interfere with sleep onset and increase bedtime resistance.

### Nap Problems

Parents of young children often struggle with naptime issues. Similar to bedtime problems and nightwakings (see Chapter 8), nap problems are often related to scheduling problems and/or poor sleep associations. To deal with naptime problems, the following strategies can be beneficial:

- Set nap times that are either based on the same time each day (e.g., 9:00 a.m. and 2:00 p.m.) or occur within 2 to 3 hours after the last awakening. Up to the age of 6 to 9 months, most babies are ready to take a nap exactly 2 hours after awakening.
- Set a consistent naptime routine that is similar to, but shorter than, the bedtime routine. For children who are taking only one nap per day, have the nap occur immediately following lunch so that there is a clear routine to the day.
- Develop positive sleep habits, including self-soothing skills. As with nightwakings, an infant or toddler who develops negative sleep associations (e.g., being rocked to sleep) will require this to occur at the onset of the nap and will be unable to return to sleep following a brief arousal. Negative sleep associations often account for short nap periods, for example, 20 to 45 minutes.
- Establish clear naptime rules, especially with toddlers.

- **Establish a consistent bedtime routine**, one that is approximately 20 to 45 minutes and includes three or four soothing activities (e.g., bath, pajamas, stories) rather than high-energy or stimulating activities (e.g., playing outside, watching television). The final activity (e.g., stories) should be a preferred one to motivate compliance with prior activities (e.g., brushing teeth). A chart of the bedtime routine, or the creation of a picture book of the bedtime routine, can be highly beneficial.

### Importance of Bedtime Routines

Institution of a bedtime routine results in decreased behavior problems at bedtime and improved nighttime sleep. Furthermore, a recent study found a dose-dependent relationship between the frequency per week of instituting a bedtime routine and sleep outcomes. That is, the more nights a family engages in a bedtime routine each week, the better a child sleeps.

- **Provide a transitional object**, such as a blanket, doll, or stuffed animal.
- **Exposure to morning bright light**, as well as avoidance of light in the evening, can help set the circadian clock for the day and increase sleepiness at bedtime.
- **Maintain healthy sleep practices**, including avoiding caffeine and electronics in the bedroom, as well as getting regular exercise.
Behavior Management Strategies

Bedtime problems are similar to any other behavioral issue and, thus, respond to general behavior management strategies. Suggestions for parents include the following:

- **Use positive reinforcement** to increase appropriate behaviors.
- **Avoid punishment** to decrease inappropriate behaviors, as punishment is not an effective way to change a child's behavior. In particular, avoid using the child's bed/bedroom for punishment or making going to bed early a consequence of negative behavior during the day (“Because you were naughty today, you have to go to bed at 7:00 instead of 8:00”).
- **Focus on increasing positive behaviors** rather than decreasing negative behaviors.
- **Be consistent** in responding, as this is the key to behavior change.
- **Do not ask questions** (e.g., “Ready for bed?”) when the intention is to state a fact (e.g., “It's time for bed”).
- **Set clear limits and follow through.**
- **Provide acceptable choices** (e.g., “Do you want to go to bed now or in 5 minutes?”) to give the child some control but within reasonable limits.

**Limit Setting: Guidelines for Parents**

- **Establish clear bedtime rules**, including activities involved in getting ready for bed (e.g., change into pajamas, brush teeth). Parents should also identify specific appropriate behaviors (e.g., staying in bed) and inappropriate bedtime behaviors (e.g., calling for parents).

- **Ignore any complaints or protests about bedtime**, such as “I'm not tired,” or “I don't want to go to bed.” Parents should also avoid discussing or arguing about bedtime as this will lead to a struggle, often reinforcing bedtime problems with increased attention. Parents should firmly and calmly let their child know that it is time for bed and continue with the established routine.

- **Put the child to bed drowsy but awake**, as it is beneficial for children to learn to fall asleep independently.

- **Check on the child**, especially if he or she is upset or crying. The visits should be brief (1 minute) and nonstimulating and occur at a frequency that is comfortable for both parents and child. These “check-ins” provide reassurance, but at the same time should be structured to reinforce limits.

- **Return child to bed or room**, being firm and calm if he or she comes out of the bedroom. For some children, simply returning them to bed multiple times works. For others, establishing a system in which the bedroom door is closed for a brief period (1 minute) if the child gets out of bed can be helpful. The time can be increased by 1 minute for each successive time out of bed. In addition, parents should praise their child for positive behaviors (e.g., staying in bed, not calling).

- **Use positive reinforcement**, including sticker charts and reward systems. Small rewards should be given contingent on positive behaviors, and, especially in younger children, rewards should be presented immediately (first thing in the morning). Larger rewards can be offered for continued positive behaviors, such as three nights of going to bed without protest. The specific behaviors and number of days and nights required to earn a reward should be based on the child's developmental level (younger children need more frequent rewards) and should be based on the number of successes rather than on number of consecutive nights of success (e.g., three stickers obtain a trip to the playground, not three consecutive stickers). The reward schedule should also be set up initially to ensure a reasonable expectation of
immediate success, which is likely to increase the child's investment in the process.

- **Stick to firm bedtime limits**, once clear rules have been established. Parents must follow through on their expectations and not inadvertently reinforce inappropriate behaviors.

- **Be persistent and consistent**, in responding to the child. Consistency is the key to any behavior change. It is very important to explain to parents that intermittent reinforcement of an undesired behavior (occasionally allowing the child to fall asleep in the parent's bed) actually makes it more difficult to extinguish or eliminate the behavior. The analogy of “playing a slot machine” may help parents understand this important behavioral concept; that is, the more unpredictable the reward is (“winning the jackpot” or “getting to stay in Mom's bed”), the more likely the individual is to persist in the behavior (“pulling the slot machine lever” or “coming into the bedroom”).

- **Expect an “extinction burst,”** after initiation of a behavioral program. It is very important to warn parents that the behavior will often become worse (intensify in severity and frequency) for several days before significant improvement occurs. Anticipating this often prevents parents from immediately abandoning the behavioral program. An extinction burst can also occur days or weeks later, so parents should be prepared for future testing of limits.

**Bedtime Pass**
A bedtime pass can help parents set limits and can be extremely helpful with children who make multiple requests after lights out. Parents can provide their child with one or two “bedtime passes,” which can be as simple as an index card that has been decorated. The child must turn in a card for each request made. No more passes means no more requests. This simple system allows children a way to make a reasonable request while setting clear limits. Another aspect that parents can add is providing a reward (e.g., trip to the library) for any pass that the child still has in the morning to discourage requests. Research shows that the bedtime pass can be highly effective in reducing problematic bedtime behaviors. It may be especially helpful for anxious children, for whom just having the option to seek out parents if they “really need it” is often sufficiently reassuring.

**PROGNOSIS**
Bedtime struggles can usually be well controlled with behavioral interventions.

**Tips for Talking to Parents**
- **Explain the role of limit setting** in bedtime stalling and bedtime refusal.
- **Discuss developmentally appropriate parental responses** to and strategies for handling noncompliant bedtime behaviors.
- **Develop an appropriate sleep schedule** that ensures adequate sleep and avoids sleep deprivation. Be sure the set bedtime is appropriate given the child's circadian rhythm and preference.
- **Develop a behavior management plan** that incorporates all potential pitfalls.
- **Establish a reward system** to reinforce positive behaviors.
- **Refer for behavioral management** if the bedtime problems are indicative of more global parenting limit-setting issues or the bedtime problems are unresponsive to simple behavioral interventions.

*See Appendix D1 for a parent handout on bedtime problems.*
Nightwakings are one of the most common sleep problems experienced by infants and toddlers (note: for a discussion of nightwaking problems in older children, see Chapter 19 on Insomnia). By 3 months of age, most healthy full-term babies are physiologically capable of “sleeping through the night” (i.e., sleep consolidation) and by 6 months of age no longer require nighttime feedings. However, 25% to 50% continue to awaken during the night at 9 to 12 months. Studies also indicate that problematic nightwakings are likely to persist, as young children often do not “outgrow” the problem.

Difficulties with sleep maintenance occur for many different reasons, but persistent and prolonged nightwakings are most commonly related to inappropriate sleep-onset associations. Sleep-onset associations are conditions that the child learns to need in order to fall asleep at bedtime. These same sleep-onset associations are also typically needed in order to fall back to sleep after naturally occurring arousals or awakenings during the night. Thus, when a child develops bedtime sleep-onset associations that are not readily available during the night (being rocked or held, having a parent present), prolonged nightwakings may occur. As infants and children typically arouse briefly on average 2 to 6 times throughout the night as a result of the normal ultradian rhythm of sleep cycles, these nightwakings (or, more accurately, these failures to fall back to sleep) may occur as often as every 90 to 120 minutes. Studies have found that parental presence at bedtime is the strongest predictor of disrupted sleep patterns and is associated with increased nightwakings and reduced total sleep time. For example, in one study of infants, parental presence was associated with a decreased sleep duration of 1.7 hours. Furthermore, approximately 30% of school-aged children are reported to fall asleep with a parent present, and this practice is associated with a six-fold increase in the likelihood of nightwakings.

Children who are able to soothe themselves back to sleep without parental intervention (“self-soothers”) generally experience brief arousals rather than prolonged nightwakings. Thus, parents are generally unaware of these brief arousals. In contrast, “signalers” are those children who alert their parents by crying or going into the parents’ bedroom upon awakening. Whether an infant or child is a self-soother or a signaler is highly influenced by the “appropriateness” of the bedtime sleep-onset associations provided (i.e., those that will also be readily available to the child during the night and do not require parental intervention). Thus, the capacity to self-soothe is clearly associated with the practice of being put to bed while drowsy but still awake (avoiding associating sleep onset with being held or rocked). In addition, parents, especially with infants and toddlers, then choose whether or not to respond to the signaled arousal or waking with such reinforcing behaviors as verbal soothing, rocking, or feeding. The practice of parents both responding immediately to and providing social reinforcement for nightwakings further increases the likelihood that they will occur and may eventually result in a “trained night crier.” Finally, although there is often considerable night-to-night and week-to-week variability in all of these behaviors, and neither infants nor parents are always consistent in the way they behave and interact, it is clear that a persistent pattern of difficulty in self-soothing and parental reinforcement of nightwakings is associated with sleep disruption in infants and children.

Sleep Associations

Sleep associations are those conditions that are habitually present at the time of sleep onset and in the presence of which the infant or child has learned to fall asleep. These same conditions are then required in order for the infant or child to fall back to sleep following periodic normal nighttime arousals. These sleep associations can be appropriate (e.g., thumbsucking) or inappropriate (e.g., rocking,
nursing, swinging). Inappropriate, or problematic, sleep associations are those that require parental intervention and thus cannot be reestablished independently by the child upon awakening during the night. Inappropriate sleep associations are the primary cause of frequent nightwakings.

**EPIDEMIOLOGY**

Overall, studies indicate that 25% to 50% of 6- to 12-month-olds and 30% of 1-year-olds have problematic nightwakings. In toddlers (aged 1-3 years), 15% to 20% continue to have nightwakings. However, some studies indicate prevalence rates of nightwakings as high as 70% in infants and 50% in toddlers. In preschoolers and school-aged children, prevalence of nightwakings is approximately 15% to 20%; thus, this is not an issue exclusive to very young children.

**ETIOLOGY AND RISK FACTORS**

As presented in Chapter 7, nightwakings are often the result of both extrinsic factors (e.g., environmental issues, parent behavior) and intrinsic factors (e.g., temperament). These intrinsic factors include a complex combination of biological, circadian, and neurodevelopmental factors that are then influenced by and interact with environmental and behavioral variables. The inability to self-soothe may represent a delay in the emergence of or a regression in behavior associated with the neurodevelopmental processes of sleep consolidation and sleep regulation that occur over the first few years of life. Developmental maturation typically governs the emergence of these “sleep milestones,” but they are also influenced by the context and environment in which they occur; thus, these are learned behaviors and amenable to modification by behavioral strategies.

In addition, the genesis of nightwakings typically involves predisposing, precipitating, and perpetuating factors. Multiple studies have found that frequent nightwakings are associated with a number of factors, including parental presence while falling asleep, room-sharing and bed-sharing, feeding to sleep and to go back to sleep during the night, and not having a consistent bedtime routine. Thus, the overriding factor, as discussed above, is negative sleep associations. An additional consideration intertwined with negative sleep associations is that children who experience more frequent arousals or disruptions during sleep, such as those related to sleep apnea, also have more opportunities to have prolonged nightwakings that require parental intervention. Conditions that may increase the likelihood of nocturnal arousals and sleep fragmentation include the following:

- **Co-sleeping**, as studies have found that babies who either share a room or share a bed with their parent(s) are highly likely to awaken during the night and seek interaction with a parent, typically as a result of an increased likelihood of parental presence at sleep onset.

- **Breast-feeding**, given that breast milk is more easily digested and breast-fed babies need to be fed more frequently. Breast-feeding is recommended until the age of 12 months by the American Academy of Pediatrics and provides a myriad of benefits. However, breast-fed babies are also more likely to be nursed to sleep at bedtime and thus to become dependent upon nursing to fall back to sleep during the night. Furthermore, one study found that nursing back to sleep following a nightwaking is associated with decreased sleep consolidation and increased nightwakings in breast-fed babies.

- ** Acquisition of normal developmental milestones**, both motor (e.g., pulling to stand, crawling) and cognitive milestones (e.g., separation anxiety), often coincide with an exacerbation or resurgence of nightwakings, especially between the ages of 9 and 12 months.

- **Sleep-disrupting events**, such as illness, vacations, and scheduling changes, can result in fragmented sleep and increased nightwakings.
Colic or other medical conditions, such as otitis media, may result in more frequent nighttime arousals. In addition, in those conditions that typically require nighttime parental intervention (e.g., colic), it may be difficult for parents to differentiate between nightwakings due to ongoing physical symptoms and those related to learned behaviors (parental attention to crying).

Other risk factors for problematic nightwakings include the following:

- **Difficult temperament**, which has been noted to be related to sleep difficulties, especially nightwakings, likely due to the child's inability to self-soothe. In addition, children who are fussy or difficult may insist on specific behaviors at bedtime and resist any attempts by parents to initiate more independent settling.

- **Insecure maternal-child attachment**, as infants who are insecurely attached are reported to have more sleep problems, although the presence of sleep problems does not necessarily imply that there should be concerns about attachment.

- **Parental anxiety**, especially common in first-time parents, as these parents are more likely to respond immediately to their baby throughout the night, often interfering with the child's learning to return to sleep independently. In addition, negative emotional states and heightened arousal in parents are likely to increase arousal levels in the infant as well, making settling more difficult.

- **Maternal depression**, as longitudinal studies indicate an increased risk of sleep disturbances in children of mothers who have previously been identified as depressed. However, infant and toddler sleep disturbances clearly contribute to maternal depression.

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**PRESENTATION AND SYMPTOMS**

Nightwakings, which require parental intervention for the child to return to sleep, are the presenting symptom. It should be noted that research, clinical, and individual parental definitions of “problematic” nightwakings in terms of frequency, length, and chronicity of the wakings may differ substantially. In clinical practice, however, it is the parents' definition of a “problem” that warrants further assessment and possibly intervention.

**Diagnostic Criteria**
See Table 7.1 in Chapter 7 (Bedtime Problems).

**Associated Features**

- **Difficulties at naptime**, also related to the development of negative sleep associations.

- **Delayed sleep onset**, as the transition to sleep may be prolonged if the association requires continued effort, such as a parent continuously rubbing the child's back or a child sucking on a pacifier without letting it fall out of the mouth. Furthermore, many parents who rock or hold their child to sleep find it difficult to make the transition to putting their child down asleep in the crib without waking their child and, thus, needing to start the pattern again.

  It should also be noted that delayed sleep onset in children with nightwakings may be primarily due to caregivers’ difficulties with setting limits at bedtime (see Chapter 7). Bedtime problems and nightwakings often coexist, and many children present with both.

- **Daytime behavior problems**, such as irritability and an increase in temper tantrums, associated with the sleep fragmentation resulting from nighttime awakenings.

- **Family stress**, including parental sleep disruption, psychiatric symptomatology (including postpartum depression), marital discord, and overall family tension, often results from nighttime sleep disturbances in infants and children. Studies indicate that successful behavioral intervention not only results in fewer
sleep problems for the child, but may also alleviate parental stress, improve parental sleep, and lead to improvements in parenting skills and management of daytime behaviors.

Early Risers
There are two groups of young children who awaken “too early” in the morning (usually determined by parental definition). The first group includes those children who have a relative advance in their circadian sleep-wake pattern (i.e., “morning larks”). In these children, both sleep-onset and morning waking time are shifted relatively earlier, and they typically have adequate sleep duration if allowed to sleep on their “preferred” schedule. The second group involves those children who often get up for the day before they have had enough sleep. These “early morning awakenings” in actuality represent the final arousal of the night, after which the child fails to return to sleep for the remainder of the nocturnal sleep period. This may be due to inappropriate sleep associations (e.g., nursing, rocking) and an inability to self-soothe, poor limit setting (e.g., parent allows child to join him or her in the parent's bed), or inadvertent reinforcement of the waking (e.g., the child is allowed to watch early morning television). These children typically return to sleep within an hour or so of getting up for what seems to be an early morning nap or may appear tired and irritable during the day. Once behavioral interventions are instituted, these children begin to sleep until a more appropriate waketime.

EVALUATION

- **Medical history**: Although generally benign, the presence of medical issues in a history may predispose a child to nightwakings and may also increase the likelihood that parents will intervene during the night. Comorbid medical problems may also make behavioral strategies that involve “extinction” procedures (see below) and distress on the part of the child more challenging for parents to successfully implement.

- **Developmental history**: Developmentally delayed children often have sleep problems that reflect the child's developmental (younger) age rather than chronological age.

- **Family history**: The history may be positive for psychopathology, especially maternal depression.

- **Behavioral assessment**: Children with a difficult temperament are at increased risk for sleep disturbances related to dysregulation.

- **Nightwaking history**: Parents often identify sleep associations as the “trick” that gets the child to fall asleep or return to sleep (e.g., nursing, bottle-feeding, rocking). Many of these children have not learned to self-soothe and have never slept through the night.

- **Physical examination**: Physical symptoms are usually noncontributory.

- **Diagnostic tests**: Tests are not indicated, although medical evaluations such as a pH probe to assess for gastroesophageal reflux or an overnight sleep study to diagnose obstructive sleep apnea may be considered if there are symptoms suggestive of an underlying medical or sleep disorder.

DIFFERENTIAL DIAGNOSIS
The diagnosis of behaviorally based nightwakings is usually straightforward. However, other causes of nighttime awakenings need to be considered:

- **Medical concerns**, including reflux and pain (especially related to otitis media), which often result in

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prolonged nighttime awakenings, delayed return to sleep, and parental inability to console the child. However, continued parental reinforcement of these nightwakings after the medical condition has resolved may contribute to maintaining them.

- **Underlying sleep disrupter**, such as periodic limb movement disorder (see Chapter 16) or obstructive sleep apnea (see Chapter 15), that contributes to increased nighttime arousals and awakenings.

- **Poor limit setting**, which is usually characterized by the parent's failure to set limits during the night (e.g., allowing the child to watch television in the middle of the night) or reinforcement of inappropriate nighttime behaviors (e.g., bringing the child to the parents' bed on a nightly basis).

- **Insufficient sleep**, which may increase arousals during sleep. Insufficient sleep can be the result of poor napping or premature discontinuation of naps by parents, an inappropriately late bedtime, inadequate time in bed, or the result of another sleep disorder. Conversely, developmentally excessive time in bed may lead to poor sleep consolidation.

- **Transient sleep disturbances**, which usually occur in a child with previous normal sleep. Transient nightwakings (i.e., “adjustment insomnia”) can be the result of a stressful life event, disruption of sleep schedule (e.g., trip, jet lag), or an illness. Short-term sleep disturbances, however, can become chronic if parents respond in a way (e.g., reinforcement of the nightwakings) that fosters inappropriate sleep habits.

- **Unhealthy sleep practices**, including maintaining an erratic sleep schedule, use of caffeine or other substances, and inadequate sleep, may contribute to disturbed sleep and nightwakings.

- **Environmental contributors**, such as a bedroom that is not conducive to sleep (e.g., noisy, hot) or disruption of sleep by others (e.g., parent waking in early morning for work), can cause an increase in the number and duration of awakenings.

### MANAGEMENT

#### When to Refer

Children or adolescents with persistent nightwakings that are not responsive to simple behavioral measures or that are extremely disruptive to the family should be referred to a sleep specialist or a professional who specializes in behavior management. If there is a concern about the existence of an underlying sleep disorder or a medical problem, appropriate referral is warranted.

#### Standards of Practice

In 2006, the American Academy of Sleep Medicine released a standards of practice for the treatment of bedtime problems and nightwakings in young children. An overall review of the literature found that 94% of studies report that treatment was efficacious and 80% of treated children demonstrate clinically significant improvement. These results were maintained at 3- to 6-month follow-up. Empirical evidence from controlled group studies utilizing Sackett criteria for evidence-based treatments provides strong support for unmodified extinction and preventive parent education. Furthermore, there is support for graduated extinction and bedtime fading and other positive routines. Note that given these guidelines and considering parental acceptance of treatment recommendations, as well as overall child response, we recommend the use of graduated extinction in the treatment of bedtime problems and nightwakings.

### Treatment
Management of nightwakings should include establishment of a set sleep schedule and bedtime routine. In addition, an intervention strategy should be chosen based on the temperament of the child and parental tolerance, as well as the parenting style of the family. Thus, behavioral interventions that are tailored to the needs and special circumstances of the individual child and family have a higher likelihood of success. For example, some families are likely to accept and tolerate prolonged crying at bedtime. Other families will require a more moderate approach in which they gradually make changes (e.g., first 3 nights establish a bedtime routine only, next 3 nights rock baby to sleep while gradually weaning off bottle).

- **Sleep Habits**
  - **Sleep schedule**, one that ensures a developmentally appropriate amount of sleep, as both inadequate sleep and excessive time in bed may result in increased nighttime arousals.

A consistent nightly bedtime will also help to reinforce the circadian sleep-wake rhythm and enable the child to fall asleep more easily.

- **Consistent bedtime routine**, one that is approximately 20 to 45 minutes and includes three to four soothing activities (e.g., bath, pajamas, stories). Research shows that simply instituting a nightly bedtime routine significantly improves both bedtime difficulties and nighttime awakenings. The mechanism is unknown, but it may be due to an overall decrease in arousal.

- **Maintenance of daytime sleep (naps)**, at least through the age of 3 to 3.5 years, as insufficient sleep in a young child will increase nighttime arousals and, thus, increase sleep problems.

- **Transitional objects**, such as a blanket, doll, or stuffed animal.

- **“Wait and see approach,”** in which parents avoid responding immediately to a baby's movements or sounds during the night. This allows the baby a chance to return to sleep independently, without parental interference, and avoids reinforcement of nightwakings.

**“Drowsy but Awake”**

The key to a successful transition from relying on parental intervention to self-soothing to fall asleep is to put the child to bed “drowsy but awake” at bedtime. This will encourage the development of self-soothing skills, which will lead to self-soothing back to sleep following normal nighttime arousals. Anticipatory guidance during well-child visits should begin to introduce this concept at the 2-week and 2-month visits, encouraging parents to implement this strategy by about 12 weeks.

- **Extinction (“crying it out”)** involves putting the child to bed at a designated bedtime and then systematically ignoring the child until a set time the next morning. Extinction has been documented to be a successful treatment (see Standards of Practice); however, it is often not an acceptable choice for families. Parents are often concerned about the effects of the treatment on their child's emotional development and are, thus, less likely to be compliant.

**Will Sleep Training Harm My Child?**

Parents are often concerned that any sleep training that involves crying will result in psychological harm to their child. There is no research that supports this belief. Rather, research indicates that immediately following sleep interventions, infants are found to be more secure, more predictable, less irritable, and to cry less following treatment. No negative impact on breast-feeding has been found. In addition, there are often significant improvements in daytime behaviors, likely as a result of increased sleep time and improved sleep quality.
A recent landmark study found that 5 years following behavioral treatment for bedtime problems and nightwakings there were no negative effects on (1) child mental health, psychosocial functioning, or stress regulation, (2) child-parent relationship, including closeness and attachment, and (3) parental mental health or parenting styles. Thus, there was a complete lack of long-term negative outcomes after sleep training.

Finally, it is also important for parents to understand that an infant learning to self-soothe is a developmental skill that enhances self-regulation as the child matures.

**Graduated extinction** involves putting the child to bed drowsy but awake and waiting progressively longer periods of time, usually in 5-minute increments, before checking on the child. On each subsequent night, the initial waiting period before checking is increased by 5 minutes. Clinical experience indicates that there is no recommended “optimal” period of time between checks, and that the exact amount of time should be determined by the parents' tolerance for crying and the child's temperament (e.g., some children become more agitated with brief parental checks). Since the key to this treatment is to allow the child to fall asleep independently and, thus, develop self-soothing skills, parents can choose how frequently or infrequently they check. When parents check on their child, they should reassure the child but keep contact brief (1-2 minutes) and neutral (e.g., pat on shoulder rather than pick up and cuddle). Research has also indicated that graduated extinction is effective even if instituted initially only at bedtime rather than throughout the night, as generalization of self-soothing skills to nighttime arousals will typically occur within 1 to 2 weeks of the child's falling asleep easily and quickly at bedtime. Thus, parents can institute checking at bedtime only, responding to their child in their usual manner throughout the night (e.g., nursing, bringing child to parents' bed). A similar checking routine during the night may need to be instituted several weeks later if nightwakings persist.

The success of graduated extinction is usually based on the parents' ability to be consistent and follow through. Thus, “proactive problem-solving” with the family, including discussing potential pitfalls (e.g., child becoming so upset that he or she vomits) and anticipation of problems (e.g., waking an older sibling), greatly increases the likelihood of success. Parents should also be warned that the first few nights of intervention are often more difficult because of a so-called “extinction burst” (increase in intensity and duration of crying); so it is critical to encourage them to anticipate this and to persevere with the intervention. In general, parents may expect significant improvement in nightwakings within 3 to 7 days.

**“Sleep Training” Steps**

- **Step 1.** Establish a set bedtime and regular sleep schedule.
- **Step 2.** Develop a consistent bedtime routine in which the activities occurring closest to “lights out” take place in the room where your child sleeps. Avoid making bedtime feedings part of the routine after 6 months.
- **Step 3.** Make the bedroom environment the same at bedtime as it is throughout the night (e.g., lighting, music).
- **Step 4.** Put your child to bed drowsy but awake.
- **Step 5.** Check on your child on a preestablished schedule that takes into account the child's temperament and your tolerance for crying. The goal is to allow your child to fall asleep
independently.

- **Step 6.** Respond to your child as usual (rocking, soothing) following nighttime awakenings. The self-soothing skills (falling asleep easily and quickly) that the child develops at bedtime are highly likely to lead to self-soothing during the night within 2 weeks.

**Good Morning Light**

A “good morning light” is a simple strategy to combat early morning awakenings or continued nighttime awakenings that are perpetuated by parents bringing their child to their bed in the early morning hours or in other ways reinforcing awakenings (e.g., parent brings child to bed once it is 5:00 a.m.). A “good morning light” involves attaching a nightlight to a timer that is set for a reasonable morning time (e.g., 6:00 a.m.). The child is told that he or she may get out of bed or go to the parents’ bed once the good morning light turns on. Clinically, it has been found to be highly effective.

- **More gradual fading of adult intervention** is appropriate for families who are unable to tolerate the above extinction approaches or consider them to be unacceptable. A plan should be developed that gradually fades (eliminates) adult intervention. In order to develop such a strategy, the end goal should be identified (e.g., falling asleep independently at bedtime) and successive steps to achieving that goal specifically outlined (e.g., 3 nights of establishing a bedtime routine and setting bedtime; 3 nights of parents sitting with baby while baby falls asleep in crib; 3 nights of parents sitting 3 feet from crib while baby falls asleep; 3 nights of sitting in doorway; 3 nights of sitting outside doorway; and so forth). Similar strategies can be implemented during the night. Again, initially parents can start with instituting treatment at bedtime only and responding to their child in their usual manner throughout the night, as generalization to a decrease in nightwakings often occurs (although nighttime intervention may be required 2-3 weeks later). However, some parents may decide to respond to their child's nightwakings in the exact same manner as at bedtime (e.g., if sitting 3 feet from crib at bedtime, will sit 3 feet from crib when child awakens) to provide a consistent response at all sleep times.

- **Discontinuing nighttime feedings** is appropriate for a baby older than 6 months to avoid inappropriate sleep associations and reinforcement of nightwakings. There is no evidence that night feedings increase the quality or quantity of sleep and, in most cases, are not physiologically necessary after 6 months of age. A signal that an infant no longer requires night feedings is a very rapid return to sleep after feeding is initiated (e.g., taking 1 oz or less, or nursing for only 1-3 minutes before falling back to sleep). The need for feedings during the night may also become a learned behavior (“learned hunger”), which leads to more frequent and prolonged nightwakings. Nighttime feedings can be weaned abruptly (“cold turkey approach”) or can be decreased gradually by volume (e.g., 1 oz every few days) in bottle-fed babies or duration (e.g., decrease by 1 minute per night) in breast-fed infants.

- **Reinforcement strategies** (e.g., sticker charts) can be beneficial with preschoolers and older children. In devising such a system, note that it is most effective if rewards are given immediately (e.g., applying sticker to chart first thing in the morning with an immediate reward) and obtainable goals are established (e.g., sticker may be earned initially just for sleeping in own bed all night, even in the face of frequent calls to parents) to reinforce success. With time, more challenging goals can be implemented (e.g., sticker obtained for sleeping in bed all night without calling to parents), with less frequent rewards (e.g., five stickers per week instead of three required to obtain a reward).
Collaboration with a behavioral therapist in designing and implementing treatment plans may be prudent if there are complex, chronic, or multiple sleep problems or if initial behavioral strategies have failed.

“Family Bed” versus “Ferber Method”

There is a great deal of public debate by family bed advocates (e.g., Sears) against graduated-extinction approaches (dubbed the “Ferber method” or “cry-it-out” approaches). Family bed advocates claim that co-sleeping (typically bed-sharing) is more natural and promotes attachment. In contrast, family bed proponents feel that allowing a child to "cry it out" at bedtime or during the night will result in psychological damage and will interfere with parent-child attachment and prolonged breast-feeding. At this time, there are no research studies that have provided support for better parent-child attachment related to the family bed or for negative repercussions of behavioral interventions (nor have they confirmed any long-term psychological sequelae resulting from prolonged co-sleeping). In actuality, research has indicated improved attachment and mood in children, as well as improved family well-being, following behavioral treatment.

Overall, there is no correct response to this age-old debate; rather, it is an individually based family decision. The choice will depend on parenting style, parental tolerance, and child temperament. However, there are two caveats that should be noted. The first is the American Academy of Pediatricians recommendation against bed-sharing in the first year of life due to its role as a potential risk factor for sudden infant death syndrome. The second is the distinction between voluntary “lifestyle” (i.e., family bed) co-sleeping and “reactive” co-sleeping, in which the parent institutes bed-sharing as a behavioral strategy for nightwakings; the latter is associated with significant sleep disruption and is often difficult to reverse without specific behavioral intervention.

PROGNOSIS

Studies indicate that nightwakings are likely to persist without intervention, although negative sleep associations that are established during infancy often taper off following the age of 3 or 4 years, when these behaviors markedly decrease (e.g., bottles/pacifiers/nursing; rocking, holding). However, if the child requires parental presence to fall asleep, the sleep disturbance may continue through middle childhood.

Tips for Talking to Parents

- **Explain that the nighttime arousals are part of the normal sleep pattern** and that the nightwakings per se are not problematic. The concern is rather that the child fails to return to sleep independently (has not learned to self-soothe) and, thus, requires parental intervention to go back to sleep.

- **Discuss sleep associations at bedtime** and their role in perpetuating nightwakings.

- **Develop an appropriate sleep schedule** that ensures adequate sleep and avoids sleep deprivation.

- **Discuss developmentally appropriate parental responses** to and strategies for handling nightwakings.

- **Institute sleep training at bedtime only** to start, with the goal of developing self-soothing skills and more appropriate sleep associations that will likely generalize to nighttime arousals.

- **Refer for behavioral management** if the nightwakings are severe and unresponsive to simple behavioral interventions.
See Appendix D2 for a parent handout on nightwakings.
Nighttime fears in children are common and typically both normal and benign. Most children experience bedtime or middle-of-the-night fears at some point during childhood, and these are usually considered a normal aspect of development. These fears characteristically begin to occur during the preschool years as children develop the cognitive capacity to understand that they can get hurt or be harmed. The types of fears that children typically have vary at different developmental stages; while young children are often afraid of monsters and other imaginary creatures, older children are more likely to fear being harmed or hurt by more realistic dangers, such as burglars, natural disasters, family/friend's security, and worries about the day's events. The most common fear is of environmental threats (e.g., inside or outside noises) and personal security (e.g., intruders).

Nighttime fears, however, can become persistent and intense, resulting in interference in daily functioning. Severe nighttime fears can result in refusal to go to bed, sleep with the lights off, or enter dark places. Some children and adolescents even refuse to be left alone or sleep in a separate room.

Nighttime Fears

Nighttime fears are highly prevalent in children and adolescents, paralleling cognitive development. Fears are usually short lived and benign, but must be differentiated from pathologic fears, nightmares, and anxiety disorders.

Epidemiology

Studies indicate that the majority of children experience nighttime fears. A study of nighttime fears in children aged 4 to 12 years found that 59% of 4- to 6-year-olds, 85% of 7- to 9-year-olds, and 80% of 10- to 12-year-olds reported nighttime fears. A more recent study of children aged 8 to 16 years found that 64% reported nighttime fears (79% of 8-12-year olds, 49% of 13-16-year-olds), with fear of intruders and home invasion the most commonly reported concern. Report of fears decreased with age, with 79% of children reporting nighttime fears compared to 49% of adolescents. In contrast to 73% of children (aged 4-12 years) reporting nighttime fears, only 34% of their parents were aware of and thus reported these same fears. Approximately one-third of children and adolescents do not disclose their fears to another person.

Severe nighttime fears are less common, occurring in 20% to 30% of children and accounting for 15% of referrals for the treatment of childhood phobias.

Etiology and Risk Factors

Anxiety, stress, and traumatic events have been linked to nighttime fears. Parental anxiety and family conflict may also play a role in exacerbating nighttime fears in children by increasing the level of emotional arousal in the child. The most common anxiety disorders associated with nighttime fears are separation anxiety, generalized anxiety disorder, and specific phobias (including animal and environmental phobias). Overall, girls (73%) report more fears than do boys (55%).

As stated above, it is important to note that the onset of nighttime fears is developmentally normal, coinciding with cognitive development and representational capabilities of children. The
types of fears also progress with age, with fears of scary dreams and imaginary creatures decreasing with age and fears of personal harm increasing with age. However, fears of darkness and worrying about the security of family and friends and about daily events are universal across all ages.

### Common Fears at Different Ages
- **Infants:** stimuli in the immediate environment (loud noises, sudden moves)
- **Toddlers:** strangers, separation, strange places, heights
- **Preschoolers:** being alone, the dark, imaginary creatures, animals, bad dreams, thunder, bodily injury, blood, needles
- **School-aged children:** personal harm (home intruders), threats to self-esteem, social or testing situations, bodily injury, illness, supernatural phenomenon, natural disasters
- **Adolescents:** future events, the unknown, performance failure

### PRESENTATION AND SYMPTOMS
Children express their fears in many ways. Note, however, that reports indicate that parents typically underreport nighttime fears. Thus, it is important to ask children directly about their nighttime fears.

- **Fearful behaviors,** such as crying, clinging, and leaving the bedroom to seek parental reassurance at bedtime or in the middle of the night.
- **Bedtime resistance,** including refusal to go to bed or requiring a parent to be present at bedtime.
- **“Curtain calls,”** characterized by frequent requests of parents (e.g., drinks, kisses) after lights out.
- **Partial or complete alleviation of fears,** in the context of sharing a room with a sibling or other household member.
- **Disrupted and inadequate sleep** characterized by delayed sleep onset, increased night wakings, decreased continuous sleep, and decreased total sleep time.

### Associated Features
- **Daytime fears,** which may involve similar or different fears.
- **Somatic complaints,** including headaches and stomachaches, especially at bedtime.
- **Family distress,** as nighttime fears can be disturbing to the entire family.
- **Anxiety disorders,** as approximately 10% of children with nighttime fears have a diagnosable anxiety disorder.
- **Daytime behavior problems,** including both externalizing and internalizing problems.
- **Decreased neurobehavioral functioning,** including working memory, attention control, and slower motor responses.

### EVALUATION
- **Medical history:** The history is generally benign.
- **Developmental history:** Types of fears experienced by developmentally delayed children generally reflect the child's developmental age rather than chronological age.
- **Family history:** Anxiety disorders in the family suggest the potential for more global anxiety problems in the
child.

- **Behavioral assessment:** The presence of daytime anxiety symptoms or oppositional behavior may suggest more global behavioral problems. It is also important to elicit a detailed description of the typical parental response to the fearful behavior.

- **Physical examination:** Physical symptoms are usually noncontributory.

- **Diagnostic tests:** Tests are not indicated.

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**DIFFERENTIAL DIAGNOSIS**

Diagnosis of nighttime fears is usually straightforward; however, other disorders should be considered:

- **Bedtime resistance:** Some children learn that expressing fears is an effective stalling tactic or a way to avoid bedtime, especially because parents may respond more positively to fearful behaviors than they do to oppositional behavior. These parental responses may then serve to reinforce fearful behaviors. The bedtime resistance, in turn, may be related to any number of different sleep problems, including limit-setting issues (see Chapter 7) and sleep-wake phase delay (see Chapter 18).

- **Noncompliance or oppositional behavior:** The presentation of bedtime difficulties and presentation of “fears” may be indicative of more global noncompliant behavior that is also present throughout the day. Overall daytime behavior and parental management skills should be assessed.

- **Nightmares:** Frightening dreams that awaken a child during the night may contribute to the development of a conditioned fear as well as avoidance of bedtime and falling asleep (see Chapter 10).

- **Phobias:** Phobias are conditioned, persistent, and often quite dramatic fear responses to specific stimuli (dogs) or circumstances (riding on elevators) that typically occur during the day, as well as significantly impact daytime functioning.

- **Anxiety disorders:** Children and adolescents with anxiety disorders, including separation anxiety disorder, generalized anxiety disorder, and posttraumatic stress disorder, frequently present with nighttime fears. The fearful behavior is usually present concomitantly during the day, and functioning is impacted. In addition, the degree, duration, and pervasiveness of the symptoms help differentiate situational and transient nighttime anxiety from a more chronic and global anxiety disorder.

- **Child abuse:** Children who have experienced physical, sexual, or emotional abuse may have nighttime fears, but these are generally severe and persistent and are accompanied by significant physiologic arousal. Many of these children also have significant daytime symptoms, including anxiety, withdrawal, and depressive symptomatology.

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**MANAGEMENT**

Most children are able to cope effectively with nighttime fears. The most commonly reported coping mechanisms include distraction (thinking of other things) and social support (talking to parents about their fears). Less frequently reported management activities include turning on a light or flashlight, hugging an inanimate object (e.g., stuffed animal), or interacting with others (e.g., asking for a drink). Many parents end up co-sleeping with their child to attenuate the fears, but this often rewards the fear behaviors and perpetuates the problem. It may also create a new problem for parents who are not interested in co-sleeping as a solution. Adolescents are much more likely to cope independently with their fears, whereas children are more likely to engage their parents.
When to Refer
Children or adolescents with persistent or severe bedtime fears that are not responsive to simple behavioral measures should be referred to a mental health professional.

Treatment
An important aspect in counseling parents on how to respond to nighttime fears is to have the parents maintain a balance between reassuring the child and avoiding reinforcement of the fears. If a child or adolescent is reassured too much, the parents may be subtly providing positive attention for the fearful behavior, thus increasing the likelihood that it will recur. In addition, some children may interpret their parents' concern about the fears as tacit proof that the fears are well founded. In general, strategies aimed at younger children more often involve parental reassurance, while older children typically benefit from an approach that includes teaching and positive reinforcement for independent coping skills.

Following are suggestions for ways parents can respond to their child's nighttime fears.

- **Reassure and communicate the idea of safety**: For example, have parents repeatedly tell the child that he or she is safe and that they are always nearby and will make sure that nothing bad happens (e.g., "Mommy and Daddy are right downstairs, and we'll always make sure that you are safe").

- **Teach the child developmentally appropriate coping skills and use of positive self-statements**: Discuss alternative ways to respond to nighttime fears, such as "being brave" and making positive self-statements (e.g., "Monsters are just pretend," “I can take care of myself," and “The dark is fun"). Practicing these self-statements well before bedtime can be helpful.

- **Implement a gradual exposure to fears**: The establishment of a step-wise approach to exposure to specific fears can be developed. For example, if a child is afraid of the dark, spending increasing amounts of time in dark places (starting with 30 seconds and increasing in 1-minute increments) or gradually diminishing nighttime lighting can be beneficial.

- **Develop creative solutions**: The use of "monster spray" (parent fills a spray bottle with water and sprays the child's room and closet at bedtime), for example, can be effective, although it should be noted that some young children might view this as evidence that a monster actually exists. Having a pet as a nighttime companion or having siblings share a bedroom are alternative strategies that work for some families. Taking on the character of a superhero before bed can be empowering for some children. Whenever possible, the child should be actively involved in generating solutions to foster a sense of mastery and control.

- **Encourage the use of security objects**: Blankets and stuffed animals, for example, can be comforting to the child.

- **Introduce a stuffed animal to “protect” or be “protected by”**: Provide the child with a stuffed animal that the child can protect or that will protect the child. The stuffed animal can be introduced as scared and needy, requiring the child’s protection. Conversely, the stuffed animal can be introduced as a friend who will help the child overcome his or her fears.

- **Have fun in the dark**: Expose the child to the dark through fun activities, such as playing flashlight tag or reading books in bed by flashlight. Hide favorite objects in the room that the child has to find with a flashlight.

- **Use a nightlight**: Having a light on can decrease a child’s fear of the dark or monsters.

- **Leave the bedroom door open**: Children can feel isolated behind a closed door.
Avoid stimulating television shows and movies: Scary movies can exacerbate fears or may be overstimulating, particularly just before bedtime. It should also be kept in mind that young children, in particular, may interpret media content that would be considered “innocuous” by adults (e.g., news programs, reality television shows) as disturbing or frightening.

Teach the child relaxation strategies: Implementing strategies such as deep breathing or visual imagery (e.g., imagining a beach or other favorite scene) can help a child relax at bedtime and fall asleep more easily.

Discuss the child's fears: Explore alternative ways to respond to the fears during the day rather than in the evening, as this is less likely to provoke anxiety.

Set appropriate, firm, and consistent limits: Set limits on bedtime behavior to avoid reinforcing the child's “being scared.” For example, a parent might say, “Remember, no crying and no calling at bedtime.”

Institute a “checking system” at bedtime: Provide the child with a predictable schedule (e.g., every 10 minutes) of parental reassurance. This has the benefit of making parental contact noncontingent on the child's behavior (e.g., calling out).

Encourage the child to remain in bed or in the bedroom: If parental presence is temporarily required to alleviate the child's fears, it is generally better for parents to stay in the child's room rather than to have the child join the parents in their room so that he or she does not become conditioned to avoid the bedroom.

Develop a reward system for appropriate bedtime behavior: Use rewards for appropriate bedtime behavior (stickers for being “brave”) rather than reinforcing (with attention) the learned fearful behavior (e.g., saying, “I'm scared”). Appropriate behaviors that are required for positive reinforcement should be as specific as possible (staying in bed all night, not calling out after lights out), and the reward schedule should be set up to offer a high likelihood of the child's being successful (e.g., nightly reward). Parents can reinforce for progressively appropriate behaviors, such as lower illumination levels or staying in room for longer periods.

Children's books regarding being scared at night (e.g., What to Do When You Dread Your Bed) or stories about commonly feared things, such as monsters (e.g., Goodnight, Little Monster), can be helpful. Books can also provide examples of coping role models of children who are afraid and conquer their fears. On the other hand, certain books (e.g., the “Goosebumps” series, Harry Potter), especially if read just prior to bedtime, may increase nighttime fears for some children.

PROGNOSIS

Nighttime fears that are part of normal developmental and are usually short lived and disappear by age 5 or 6 years. Fears may become more chronic in a child or adolescent with an anxiety disorder.

Tips for Talking to Parents

- Distinguish between normal and pathologic fears.
- Explain that normal nighttime fears are part of normal cognitive development.
- Discuss treatment options and parents' response to fearful behavior.
- Develop a plan of gradual exposure to fears and use of coping skills.
- Reinforce establishment of appropriate sleep habits and parental limit setting.

http://obgynebooks.com
See Appendix D3 for a parent handout on nighttime fears.
**Nightmares** are frightening dreams that usually awaken a child or adolescent, leaving the child upset and in need of comfort. Both the ICSD-3 and DSM-5 define them as “repeated occurrences of extended, extremely dysphoric, and well-remembered dreams,” which usually involve threats to survival, security, and physical integrity. Other terms synonymous with “nightmares” include bad dreams, scary dreams, and anxiety dreams. Although some individuals distinguish between nightmares, which awaken the sleeper, and bad dreams, which do not, this distinction is arbitrary. When the nightmare involves awakening, most children are afraid to return to sleep and seek comfort. Given that very young children often cannot distinguish between a dream and reality, they may insist that something fearful continues to exist.

**Nightmares**

Nightmares are frightening dreams, occurring during REM sleep, that usually result in an awakening from sleep.

Nightmares usually involve fear or anxiety but can also include other negative emotions, such as anger, sadness, embarrassment, or disgust. They also typically involve imminent physical harm to the child. The content of nightmares usually differs across the ages and reflects common developmental issues. Most young toddlers have concerns about being separated from their parents. By age 2 years, nightmares typically begin to incorporate monsters and other frightening imaginary creatures. For young children, nightmares may also involve a recent traumatic event (e.g., getting lost, being immunized, being barked at by a large dog). Older children often have nightmares related to frightening or upsetting movies, television programs, stories, or a disturbing daytime experience. Nightmares may also coincide with a stressor or traumatic event (e.g., being away overnight, starting a new school).

**Epidemiology**

Studies indicate that approximately 75% of children report experiencing at least one nightmare in their lifetime, and 10% to 50% of young children have nightmares that result in parental interaction during the night starting as young as 2.5 years. Although experiencing infrequent nightmares is quite common, the prevalence of frequent nightmares is less common. One study reported prevalence rates for chronic nightmares, defined as a nightmare problem lasting longer than 3 months, as 24% for ages 2 to 5 years and 41% for ages 6 to 10 years. Peak nightmare prevalence is between 6 and 10 years of age, with surprisingly few parents of preschool children reporting frequent nightmares (1.9%-3.9%). Another study of children aged 5 to 15 years found that 5% of children experienced nightmares at least once a week.

Nightmares are equally experienced by boys and girls before the age of 12 years. After age 12 years, some studies have found that girls are more likely to report nightmares. Twin studies have found support for a genetic basis to frequent nightmares.

**Etiology and Risk Factors**

- Prior nightmares, as having bad dreams appears to be a somewhat stable characteristic.
- Stress and/or traumatic events, including child abuse. Nightmares typically begin within 3 months of the
traumatic event.

- **Anxiety and anxiety disorders**, which can lead to both increased frequency and severity of nightmares. Separation anxiety is commonly associated with nightmares or bad dreams.

- **Insufficient sleep**, which can result in intense and vivid dreams due to increased rebound REM sleep.

- **Insomnia**, which is often comorbid with nightmares, and reported by approximately 20% of children with nightmares.

- **Nightmares experienced by parents** has been found to be associated with nightmares in children. This may be due to heredity or environmental factors.

- **Medications**, particularly those that have a direct effect on REM sleep. These may be medications that increase the amount of density of REM or drugs that suppress REM and thus result in REM “rebound” when discontinued (see Chapter 20).

**PRESENTATION AND SYMPTOMS**

Children or adolescents with nightmares will usually recall at least fragmented and often detailed scary dream content and will awaken frightened but coherent. In addition, if awakening occurs, the child or adolescent is afraid to return to sleep and will often seek parental reassurance.

**Diagnostic Criteria**

See Table 10.1.

**Associated Features**

- **Daytime fears**: Many children with nightmares also report nighttime fears (see Chapter 9). Some may also have symptoms of a more global anxiety disorder that includes daytime fears.

- **Bedtime resistance**: Some children develop a conditioned aversion to their bed or bedtime because they associate sleep with nightmares.

- **Daytime behavior problems**: Children who experience nightmares are more likely to have symptoms of hyperactivity, frequent temper tantrums and mood disturbance, and poor academic performance.

**TABLE 10.1. Diagnostic Criteria: Nightmare Disorder**

A. Repeated occurrences of extended, extremely dysphoric, and well-remembered dreams that usually involve threats to survival, security, or physical integrity.

B. On awakening from the dysphoric dreams, the person rapidly becomes oriented and alert.

C. The dream experience, or the sleep disturbance produced by awakening from it, causes clinically significant distress or impairment in social, occupational, or other important areas of functioning as indicated by the report of at least one of the following:

1. Mood disturbance (e.g., persistence of nightmare affect, anxiety, dysphoria)
2. Sleep resistance (e.g., bedtime anxiety, fear of sleep/subsequent nightmares)
3. Cognitive impairments (e.g., intrusive nightmare imagery, impaired concentration, or memory)
4. Negative impact on caregiver or family functioning (e.g., nighttime disruption)
5. Behavioral problems (e.g., bedtime avoidance, fear of the dark)
6. Daytime sleepiness
7. Fatigue or low energy
EVALUATION

- **Nightmare history**: In evaluating nightmares, both chronicity and severity should be carefully assessed, as nightmare severity is more likely to be related to psychopathology.

- **Medical history**: The history is generally benign.

- **Developmental history**: Developmentally delayed children may be less able to verbalize concerns about nightmares.

- **Family history**: The ubiquitous nature of nightmares in the general population makes a positive family history nonspecific. However, parents who have themselves experienced frequent or troublesome frightening dreams may respond differently to their child's nightmares (e.g., more attention, anxiety).

- **Behavioral assessment**: A history of more global anxiety, regression in behavior, or extreme arousal in association with nightmares should raise concerns about the possibility of abuse or trauma.

- **Physical examination**: Physical symptoms are usually noncontributory.

- **Diagnostic tests**: A sleep diary that documents the frequency of nightmares over a period of several weeks and the duration of associated nightwakings may be helpful.

DIFFERENTIAL DIAGNOSIS

- **Sleep terrors and sleepwalking**: Parents often have a difficult time distinguishing between nightmares and disorders of arousal, such as sleep terrors and sleepwalking (see Chapter 11); it should be noted that many children with sleep terrors or sleepwalking will also have nightmares. Nightmares, in comparison to disorders of arousal, usually have the following features:
  - Occurrence in the latter half of the night, when REM sleep predominates
  - Complete or partial recollection of dream content
  - Total recall for the event
  - No confusion or disorientation
  - Delayed return to sleep

- **Other episodic nocturnal phenomena**: Nocturnal seizures, for example, are occasionally confused with nightmares, but typically have associated motor and sensory features and often include stereotypic characteristics (see chart in Chapter 11 for a comparison of clinical features).

- **Psychiatric disorders**: Frequent nightmares may be associated with a psychiatric disorder, including anxiety disorders, bipolar disorder, schizophrenia, and, most notably, posttraumatic stress disorder.

- **REM behavior disorder (RBD)**: RBD is an unusual sleep disorder characterized by loss of muscle atonia (e.g., paralysis) that normally occurs during REM sleep. The consequent “acting out” of vivid, disturbing, and frequently violent dreams can result in severe injury to the sleeper and also to any bed partners.
partner. This disorder is associated with neurodegenerative processes, such as Parkinson's disease, in older adults, and although reported in children in association with neurological disorders, is considered extremely rare in childhood.

**MANAGEMENT**

**When to Refer**

Children or adolescents with persistent or severe nightmares that are not responsive to simple behavioral measures or whose nightmares are extremely disruptive should be referred to a mental health professional for evaluation and treatment.

**Treatment**

In general, as with other anxiety-associated behavioral issues, strategies for dealing with nightmares aimed at younger children more often involve active parental reassurance, whereas older children may benefit more from an approach that includes teaching and positive reinforcement for independent coping skills.

Suggestions for ways parents can manage their child's nightmares follow.

*Reduce the Likelihood of Nightmares*

- **Avoid exposure to frightening or overstimulating images,** especially just before bedtime, including frightening stories, movies, and television shows. What children interpret as disturbing or frightening may not be interpreted as such by adults and thus heightened sensitivity to developmental and idiosyncratic differences is important.

- **Reduce stressors,** as persistent nightmares may indicate stress or an ongoing concern.

- **Ensure adequate sleep,** as insufficient sleep contributes to increased nightmare frequency.

*Parental Response to Nightmares*

- **Reassurance by parents** that “It was only a dream.” It is important that parents remain calm and matter of fact, striking a balance between reassuring the child and not providing excessive attention. If the child gets out of bed, the parent can calmly escort the child back to bed and briefly provide reassurance there. Further discussion of the nightmare should be postponed until the following day, when alternative coping strategies for the future may be discussed.

- **Security objects** can be comforting and facilitate a faster return to sleep. Some children find the presence of a family pet reassuring.

- **Dim, low-level nightlight** can be helpful.

- **Encouragement of verbal children to use their imagination** may help alleviate nightmares. Effective strategies may include drawing a picture that represents the bad dreams and then crumpling it up and throwing it away, devising a positive ending to the dream (see imagery rehearsal below), hanging a dream catcher over the bed, or flipping their pillow over to “change the channel.”

*Management of Frequent Nightmares*

- **Imagery rehearsal** has been found to be a highly effective treatment strategy in managing nightmares. Imagery rehearsal involves developing an alternative ending to a frequently experienced dream and practicing that imagery during the day. For example, changing the ending of a dream from a monster attacking someone to a monster offering ice cream or playing ball with the monster. It can also involve the
child taking on a powerful person in the dream, so vanquishing the monster or turning the monster into a stuffed animal. To initiate imagery rehearsal, a child should be encouraged to create an alternative ending or a different dream and practice throughout the day. Upon awakening from a nightmare, the child should again practice the new dream content replacing the former imagery.

- **Relaxation strategies**, including progressive muscle relaxation and guided imagery. Relaxation tapes can be obtained through the Child Anxiety Network (www.childanxiety.net). Other resources are available at the Anxiety Disorder Association of America; www.adaa.org.

- **Systematic desensitization**, combined with relaxation strategies, can be used to countercondition the anxiety response. Systematic desensitization involves developing a hierarchy of fear-invoking activities or thoughts from least to most frightening with the child (e.g., looking at a picture of a dog, watching a friend play with a puppy, petting a large dog). These activities or thoughts are paired with a relaxing activity (deep breathing, progressive muscle relaxation) that counters the fear response. This technique may be particularly helpful with nightmares that have a recurrent specific theme.

- **Children’s books** regarding frequent nightmares or ways to manage being scared at night (e.g., *What to Do When You Dread Your Bed*) can be helpful.

- **See Chapter 9** (Nighttime Fears) for additional treatment strategies.

**PROGNOSIS**

Nightmares are usually short-lived but may persist in some children or adolescents, especially if the nightmares are related to a traumatic event.

**Tips for Talking to Parents**

- **Explain that nightmares** are virtually universal and are part of normal cognitive development, usually peaking between 6 and 10 years of age.

- **Discuss developmentally appropriate parental responses** to and strategies for handling nightmares.

- **Develop an appropriate sleep schedule** that ensures adequate sleep and avoids sleep deprivation.

- **Discuss any recent stressors or traumatic events that may be contributory**, while reassuring parents that the vast majority of the time nightmares are isolated events.

- **Refer for psychological evaluation** if the nightmares are highly persistent or severe and unresponsive to simple behavioral interventions.

- **Recommend strategies** for managing nightmares such as imagery rehearsal and relaxation strategies.

*See Appendix D4 for a parent handout on nightmares.*
Parasomnias are defined as undesirable physical events or experiences that occur during entry into sleep, within sleep, or during arousal from sleep. Parasomnias may be further characterized as occurring primarily during non-REM (NREM-related parasomnias) sleep (referred to as disorders of arousal) or in association with REM sleep, including nightmares (see Chapter 10) and sleep paralysis (see Chapter 17). Other parasomnias include sleep enuresis (see Chapter 14) and sleeptalking.

This chapter will discuss the clinical presentation and management of the disorders of arousal, which include confusional arousals, sleepwalking (somnambulism), and sleep terrors (pavor nocturnus). These are related sleep disorders that share a common underlying pathophysiology, have similar clinical features, and commonly co-occur in children. The disorders of arousal share characteristics of both the waking and deep sleep states, and involve autonomic or skeletal muscle disturbances, autonomic behaviors, and disorientation. Sleepwalking, sleep terrors, and confusional arousals occur almost exclusively during slow-wave sleep (SWS; stage N3 of NREM sleep), although episodes can also occur during a nap if SWS is present.

These disorders of arousal usually occur within a few hours after sleep onset, last from a few minutes to as long as 30 to 40 minutes, and are characterized by amnesia for the event. During an episode, children or adolescents may have the appearance of being awake, and most children actively avoid comfort or soothing, which may be disturbing to parents. During an episode, children may be very difficult to awaken and will usually appear confused if awakened. Although they may be triggered by stress, these disorders do not indicate the presence of a primary underlying psychological problem or trauma. Finally, because the relative percentage of SWS is highest in childhood and drops fairly dramatically in the second decade of life, disorders of arousal are most common in young children and their prevalence declines with increasing age.

**Common Conditions of Disorders of Arousal (from NREM Sleep)**

- Similar genetic and familial patterns
- Similar pathophysiology of partial arousals from deep sleep
- Similar priming by sleep deprivation and biopsychosocial stressors
- Not secondary to psychiatric disorders
- Not generally secondary to neuropathology or head injury
- Associated with absent or minimal cognitive functioning during the event
- Associated with amnesia for the prior episode
- May be triggered by sound, touch, or other stimuli

**CONFUSIONAL AROUSALS**

Confusional arousals originate during SWS and involve disorientation and unresponsiveness to the environment, as well as amnesia for the event. They are often triggered by forced awakening, particularly early in the night or upon attempting to awaken in the morning. They often start with the child sitting up in bed, may involve thrashing around, and are often accompanied by agitation and combative behavior. If the child gets out of bed, then it is considered a sleepwalking event. Although exacerbated by stress and anxiety, comorbid
Confusional Arousals
Confusional arousals are nocturnal episodes characterized by confusion, disorientation, grogginess, and, at times, significant agitation upon awakening from SWS or following forced awakenings.

SLEEPWALKING
Sleepwalking is a common and generally benign sleep behavior, although potential safety concerns (see below) should be taken into consideration. Sleepwalking episodes usually begin as confusional arousals, although they can also begin with a child immediately getting out of bed. During sleepwalking episodes, the child appears confused or dazed, the eyes are usually open, and he or she may mumble or give inappropriate answers to questions. Occasionally, a sleepwalking child may appear agitated. A sleepwalker is typically clumsy and may perform bizarre or strange actions such as urinating in a closet. The sleepwalking may range from walking calmly to the parents' bedroom, to walking downstairs, to leaving the house or stepping out onto a balcony or rooftop. Sleepwalking can occur infrequently or on a nightly basis. Injuries can be common during sleepwalking, ranging from bruises to more serious injuries as a result of falling down stairs, heading outside, or engaging in other risky behaviors.

Sleepwalking
Sleepwalking is a common parasomnia in children, occurring during the first few hours of the night when SWS is predominant. Although usually benign and self-limited (typically disappearing by adolescence), there are often associated safety concerns (e.g., falling out of windows, wandering outside) that should be addressed.

SLEEP TERRORS
Sleep terrors, or night terrors, are dramatic events and, as such, can be highly distressing to parents or caregivers (these episodes are referred to as “sleep terrors” rather than “night terrors” because they can occur during any sleep period, including naps). As disturbing and frightening as these events appear to the observer, the child is totally unaware of his or her behavior. Paradoxically, sleep terrors are much worse to watch than to experience, and much less traumatic to the child than a nightmare or bad dream.

Sleep Terrors
Sleep terrors are characterized by a sudden arousal from SWS, accompanied by autonomic and behavioral manifestations of intense fear.

EPIDEMIOLOGY
There appears to be no difference between boys and girls in the occurrence of these behaviors.

- Confusional arousals: Because confusional arousals may not be recognized or brought to medical attention,
the prevalence is difficult to determine. But, reported prevalence rates are approximately 17% in children aged 3 to 13 years and 2.9% to 4.2% in adults older than 15 years. Lifetime prevalence in one study was reported to be 18.5%. Confusional arousals commonly co-occur with sleepwalking and sleep terrors. Onset is typically before age 5 years, and the duration is described as ranging from 6 months to 13 years.

- **Sleepwalking:** Many children (15%-40%) sleepwalk on at least one occasion, with studies indicating a prevalence of approximately 17% of children regularly sleepwalking, and 3% to 4% with frequent episodes. Sleepwalking may persist into adulthood, with the prevalence in adults of about 4%. The prevalence is approximately 10 times greater in children with a family history of sleepwalking. It should be noted that the prevalence of reported sleepwalking may be an underestimate because episodes may be unobserved or misinterpreted as nightwakings. Onset of episodes is usually between ages 4 and 6 years, and peak occurrence is between ages 8 and 12 years. About one-third of sleepwalkers have episodes over a 5-year span; about 10% will continue to sleepwalk for 10 years. Most children who sleepwalk had confusional arousals at an earlier age.

- **Sleep terrors:** Approximately 1% to 6.5% of children experience sleep terrors, primarily during the preschool and elementary school years. The age of onset is usually between ages 4 and 12 years. The frequency of episodes is often highest at the onset and tends to be higher with younger age of onset. Because of the common genetic predisposition, the prevalence of sleep terrors in children who sleepwalk is about 10%. Although sleep terrors can occur at any age from infancy through adulthood, most individuals outgrow sleep terrors by adolescence.

### ETIOLOGY AND RISK FACTORS

Disorders of arousal are considered to represent a dissociation of the regions of the brain with activation of locomotor centers, combined with sleep inertia and sleep state instability. There are a number of factors that contribute to their presence.

- **Positive family history:** There is often a genetic component in disorders of arousal. Twin studies indicate genetics are involved in 50% to 65% of cases of sleepwalking. The likelihood of sleepwalking is 22% if neither parent has the disorder, 45% if one parent has the disorder, and 60% if both parents are affected.

- **Other associated conditions:** Disorders of arousal appear to be more common in individuals with migraine headaches and Tourette syndrome, possibly related to alterations in serotonin metabolism.

- **Factors that exacerbate or trigger disorders of arousal:** Disorders of arousal occur almost exclusively in SWS. Inadequate sleep and sleep disruption often result in a rebound compensatory increase in SWS. Therefore, in general, any condition that disrupts sleep or reduces sleep duration tends to increase the likelihood that a partial arousal parasomnia episode will occur in a susceptible individual. Environmental conditions (e.g., noise) that increase arousals, especially during SWS, may also trigger events. Some conditions may also increase the likelihood of partial arousal episodes by decreasing the arousal threshold.

Exacerbating and trigger factors include:

- Inadequate sleep (acute or chronic)
- Irregular sleep schedule
- Change in sleep schedule, such as discontinuing daytime naps, beginning attend day care, or start of school
- Sleep disturbances, especially sleep-disordered breathing (see Chapter 15), as well as periodic limb
movements (PLMs) (see Chapter 16)

- Fever and illness
- Medications that increase SWS (e.g., lithium) or result in rebound SWS upon withdrawal (e.g., benzodiazepines, tricyclic antidepressants)
- Caffeine
- Sleeping with a full bladder

- Sleeping in a different environment, especially when traveling
- Noise and light
- Situational stress and anxiety

PRESENTATION AND SYMPTOMS

Disorders of arousal usually occur in the first few hours after sleep onset and last a few minutes to half an hour. Frequency ranges from a one-time event to a nightly occurrence; some children may have multiple episodes in a single night. In addition, occurrences may be episodic, happening in nightly cycles for several nights to a few weeks followed by periods of no episodes.

Injury Risk

Sleepwalking in particular can lead to physical harm, resulting from falling down stairs, walking into traffic, or going outside in cold weather without adequate clothing. Although sleep terrors and confusional arousals do not typically involve displacement from bed, children may injure themselves by flailing around, falling out of bed, or actively trying to resist attempts at restraint or comforting by the parent. Ensuring safety should be one of the primary concerns of healthcare practitioners when dealing with disorders of arousal.

Confusional Arousals

Clinical characteristics of confusional arousals include agitation, crying or moaning (“no, no!”), disorientation, and, particularly, slow mentation on arousal from sleep (sleep inertia). The duration of episodes is typically 5 to 15 minutes but may last up to several hours.

Sleepwalking

Parents are generally able to identify an event as sleepwalking; occasionally, the presenting complaint is nocturnal episodes that involve unusual or bizarre behaviors (going to a sibling's room in the middle of the night, wandering around downstairs, urinating in a waste basket), confusion, agitation, and incoherent responses to questions. Parents, however, are not always able to tell whether their child is awake or asleep at the time.

Sleep Terrors

Sleep terrors usually have a sudden onset, and the child's appearance during one of these episodes is that of extreme agitation, fright, and confusion, often involving crying and/or screaming. Extreme physiologic arousal (e.g., hyperventilation, tachycardia, diaphoresis, dilated pupils) is common. However, sleep terrors may be much milder (sometimes described as a confusional arousal), with the child simply appearing agitated. A child having a sleep terror is often clumsy and may flail, push a parent away, or behave in other
strange ways. Because of the dramatic nature of these episodes, parents may be concerned that their child has experienced some emotional or physical trauma; alternatively, parents may also assume that the events themselves result in psychological damage to the child. The presentation may be atypical in very young children, with prolonged episodes (30-45 minutes) that are less dramatic (whimpering, rocking).

Other Related Disorders

- **Sleep related eating disorder** is considered to be a unique variant of disorders of arousal, although rare in childhood. This disorder is more common in females than in males (2:1). Symptoms include dysfunctional eating associated with an arousal from sleep, as well as one of the following: (1) consumption of peculiar foods or combinations of foods, or inedible or toxic substances, (2) sleep related injurious behaviors (e.g., knife lacerations, burns), or (3) adverse health consequences, such as weight gain. There is usually partial or complete loss of awareness during the event and impaired recall. The eating is perceived as involuntary or “out-of-control.” This disorder has been associated with hypnotic use. Individuals with sleep related eating disorder may also have daytime eating disorders, such as bulimia or anorexia, but the compensatory purging behaviors do not occur during the nighttime episodes.

- **Sleeptalking** can be idiopathic or associated with disorders of arousal. Sleeptalking can occur during REM or NREM sleep. It is highly prevalent, with lifetime prevalence rates of 66%. The content of sleeptalking has not been found to be associated with prior waking behavior or memories.

Diagnostic Criteria


<table>
<thead>
<tr>
<th>TABLE 11.1. General Diagnostic Criteria for Disorders of Arousal</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Recurrent episodes of incomplete awakening from sleep</td>
</tr>
<tr>
<td>B. Inappropriate or absent responsiveness to efforts of others to intervene or redirect the person during the episode</td>
</tr>
<tr>
<td>C. Limited (e.g., a single visual scene) or no associated cognitive or dream imagery</td>
</tr>
<tr>
<td>D. Partial or complete amnesia for the episode</td>
</tr>
<tr>
<td>E. The disturbance is not better explained by another sleep disorder, mental disorder, medical condition, medication, or substance use.</td>
</tr>
</tbody>
</table>

Note:

1. **The events usually occur during the first third of the major sleep episode.**

2. **The individual may continue to appear confused and disoriented for several minutes or longer following the episode.**

### TABLE 11.2. Diagnostic Criteria: Confusional Arousals

A. The disorder meets general criteria for NREM disorders of arousal.  
B. The episodes are characterized by mental confusion or confused behavior that occurs while the patient is in bed.  
C. There is an absence of terror or ambulation outside of the bed.

*ICD-9-CM: 327.41; ICD-10-CM: G47.51

Note:  
1. *There is typically a lack of autonomic arousal such as mydriasis, tachycardia, tachypnea, and diaphoresis during an episode.*


### TABLE 11.3. Diagnostic Criteria: Sleepwalking

A. The disorder meets general criteria for NREM disorders of arousal.  
B. The arousals are associated with ambulation and other complex behaviors out of bed.

*ICD-9-CM: 307.46; ICD-10-CM: F51.3


### TABLE 11.4. Diagnostic Criteria: Sleep Terrors

A. The disorder meets general criteria for NREM disorders of arousal.  
B. The arousals are characterized by episodes of abrupt terror, typically beginning with an alarming vocalization such as a frightening scream.  
C. There is intense fear and signs of autonomic arousal, including mydriasis, tachycardia, tachypnea, and diaphoresis during an episode.

*ICD-9-CM: 307.46; ICD-10-CM: F51.4

Associated Features

- **Impact on social functioning:** Because of the potential embarrassment and the high likelihood of harm, many children and adolescents with disorders of arousal avoid social situations such as overnight visits to friends and summer camp.

- **Parental anxiety:** Because of the unusual nature of these events, parents are often highly anxious about whether there is an underlying meaning to the episodes as well as how to respond to the events, and are often concerned about not being present during an event (e.g., avoid use of babysitters, discourage sleepovers).

**EVALUATION**

- **Medical history:** The history is generally benign. However, it may reveal evidence suggestive of a sleep disturbance that results in disrupted and/or insufficient sleep, including obstructive sleep apnea, restless legs syndrome, or periodic limb movement disorder (PLMD). Frequent nighttime awakenings that are behaviorally-based can also result in insufficient sleep and contribute to episodes. The medical history may also suggest the need to rule out a seizure disorder. Possible risk factors for seizures include a history of seizures as well as unusual characteristics of the episodes themselves, such as stereotypic features, multiple nightly occurrences, and late onset (adolescence).

- **Developmental and school history:** The history is usually normal. The presence of developmental delay may increase suspicion for seizures.

- **Family history:** The family history is often positive for sleeptalking, sleepwalking, or sleep terrors.

- **Behavioral assessment:** Most children do not have significant behavioral concerns. Because individuals with disorders of arousal are actually asleep during the episodes, sleep disruption and associated evidence of daytime sleepiness are unusual.

- **Psychiatric issues:** A recent study found that disorders of arousal were associated with an increased risk of psychotic experiences.

- **Physical examination:** The physical examination is generally unremarkable.

- **Diagnostic tests:**
  - **Overnight polysomnography (PSG)** is not a routine part of the evaluation for disorders of arousal. Because these are episodic events, they may not be captured on a single-night study. However, if there is a concern about another underlying sleep disrupter (e.g., sleep-disordered breathing, PLMs), an overnight sleep study is appropriate. PSG may also be warranted to differentiate between a disorder of arousal and a seizure disorder (note that only some sleep centers have the capability of a full seizure montage).

  - **Home videotaping** of nocturnal episodes by parents can be a more effective way of capturing and recording events that occur infrequently. Reviewing the tapes may be helpful in distinguishing disorders of arousal from other nocturnal behaviors, especially seizures.

  - **Sleep diaries** can help the assessment for contributing factors, such as insufficient and irregular sleep schedules.
Overnight Sleep Study (Polysomnography)

According to practice parameters published by the American Academy of Sleep Medicine, children with frequent NREM parasomnias should be clinically screened for the presence of comorbid sleep disorders and PSG should be performed if there is a suspicion for sleep-disordered breathing or PLMD.

DIFFERENTIAL DIAGNOSIS

- **Disorders of arousal (confusional arousals, sleepwalking, sleep terrors) versus seizure disorder**: It may be very difficult to differentiate nocturnal seizures from disorders of arousal, particularly if atypical features are present. Major characteristics of the episodes that are more likely to indicate the presence of a seizure disorder are summarized in Table 11.5; these include stereotypic behaviors and tonic-clonic movements, multiple events per night, occurrence at sleep-wake transitions or after the first third of the night, and associated daytime sleepiness. Enuresis may occur during or after disorders of arousal, particularly confusional arousals, but more commonly indicates seizure activity. The presence of developmental delays or neurological disorders, daytime seizures, or a family history of seizures increases the risk of nocturnal seizures (see also Chapter 22).

**TABLE 11.5. Differentiation of Episodic Nocturnal Phenomenon**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Sleepwalking/Sleep Terrors/Confusional Arousals</th>
<th>Nightmares</th>
<th>Nocturnal Seizures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Timing during night</td>
<td>First third</td>
<td>Last third</td>
<td>Variable; often at sleep-wake transition</td>
</tr>
<tr>
<td>Sleep stage</td>
<td>Slow-wave sleep</td>
<td>REM</td>
<td>Non-REM &gt; Wake &gt; REM</td>
</tr>
<tr>
<td>Age of onset</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autonomic arousal</td>
<td>Low/high/medium</td>
<td>Mild to high</td>
<td>Variable</td>
</tr>
<tr>
<td>Arousal threshold</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Daytime sleepiness</td>
<td>None</td>
<td>+/-</td>
<td>Often</td>
</tr>
<tr>
<td>Increased by insufficient sleep</td>
<td>Yes</td>
<td>Sometimes</td>
<td>+/-</td>
</tr>
<tr>
<td>Incontinence, tongue-biting, drooling, stereotypic, repetitive</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>
Disorders of arousal versus nightmares: Parents often confuse disorders of arousal with nightmares, often assuming that their child is having a nightmare during one of these episodes. As summarized in Table 11.5, disorders of arousal typically occur in the first half of the night and there is no or limited recall of the event the next day. On the other hand, following a nightmare a child can relate the dream sequence and will recall the event the next day. A child is often reluctant to return to sleep following a nightmare and will seek reassurance.

Nocturnal panic attacks: The patient typically has similar episodes that occur in the daytime, and there is recall of the episode the next morning.

MANAGEMENT

When to Refer

Children or adolescents with disorders of arousal who also present with symptoms of another underlying sleep disrupter (e.g., sleep-disordered breathing) should be referred to a sleep specialist. If pharmacologic treatment is being considered, it is also best to consult with a pediatric sleep specialist. When there are concerns regarding stress or other psychological issues, a referral to a mental health specialist is warranted.

Treatment

Management of disorders of arousal should first include reassurance and education of the child and family regarding the benign and self-limited nature of the disorder. Parents may be told that most children stop having episodes by adolescence, when the presence of SWS greatly diminishes. Interim management should include institution of appropriate safety measures and discussion of trigger and exacerbating factors. Sleep hygiene and behavioral management of the episodes should be reviewed. However, the decision about whether to actively treat the events is based on the frequency and severity of the episodes and can involve medication management or scheduled awakenings.

Safety

- Safety measures, including use of gates (doorways, top of staircases), locking of outside doors and windows, lighting of hallways, and ensuring the safety of the sleeping environment (e.g., removing clutter on floors, locking away kitchen knives).
- Parent notification measures, such as alarm systems or a bell attached to the bedroom door.
- Managing sleepovers away from home, including informing other caregivers of potential

<table>
<thead>
<tr>
<th>Recall of event</th>
<th>None or fragmentary</th>
<th>Vivid</th>
<th>Not usual</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple episodes per night</td>
<td>Rare</td>
<td>Occasional</td>
<td>More common</td>
</tr>
<tr>
<td>Family history</td>
<td>Common</td>
<td>Rare</td>
<td>Variable</td>
</tr>
<tr>
<td>Polysomnography</td>
<td>Indicated if atypical features</td>
<td>Not indicated</td>
<td>Indicated if atypical features; requires extended EEG montage</td>
</tr>
</tbody>
</table>
sleepwalking behavior.

- **Healthy sleep habits**
  - **Ensure adequate sleep and maintain regular sleep-wake schedule**, as insufficient sleep is the primary contributor to these events.
  - **Avoid caffeine**, as caffeine can increase sleep disruption, decrease sleep efficiency, and contribute to sleep deprivation.
  - **Address behaviorally-based nightwakings or bedtime refusal**, which may be contributing to insufficient sleep.

- **Parental response during an event**
  - **Avoid awakening**, as attempts to awaken a child during an episode will typically increase agitation and prolong the event.
  - **Guide the child back to bed**, to encourage return to normal sleep.
  - **Avoid interfering**, as this can prolong the event. The normal response of parents is to try and comfort their child during one of these episodes, which may increase the child's agitation. It is best for a parent to quietly stand nearby to ensure safety, but not to interact.
  - **Avoid next-day discussions**, as this is likely to worry the child and may lead to bedtime resistance and insufficient sleep.

- **Additional treatment**
  - **Pharmacologic treatment** may be indicated in cases of frequent or severe episodes, high risk of injury, violent behavior, or serious disruption to the family. The primary pharmacologic agents used are potent SWS suppressants, primarily benzodiazepines and tricyclic antidepressants.
    - **Short-acting benzodiazepines**: Benzodiazepines (e.g., diazepam, 1-2 mg) can be given as a single small dose at bedtime for 3 to 6 months (until episodes are totally suppressed). Small doses of longer-acting benzodiazepines (e.g., lorazepam, clonazepam) may also be effective, although more likely to result in morning grogginess. Intermittent drug therapy has also been used in patients who have a pattern of events that occur in clusters of days or weeks separated by episode-free periods. Abrupt discontinuation often results in significantly increased SWS (rebound), so it is important to taper medication slowly over several weeks.
    - **Antidepressants**: Tricyclic antidepressants (clomipramine, desipramine, imipramine) at bedtime have also been used in patients who are nonresponsive to benzodiazepines. Although selective serotonin reuptake inhibitors are also potent SWS suppressants, they are rarely used to treat disorders of arousal.
  - **Scheduled awakening** is a behavioral technique that is most likely to be successful in situations in which episodes occur on a nightly basis. Studies have indicated that in children with frequent disorders of arousal that occur at a highly predictive time, scheduled awakening can be highly effective. Scheduled awakening involves having the parent wake the child approximately 15 to 30 minutes prior to the time of night that the first episode typically occurs. Thus, parents first need to keep a sleep diary that includes the exact time of each episode. The parent then begins awakening the child on a nightly basis, approximately 30 minutes before a usual event, just to the point of arousal (e.g., child changes position or mumbles). For example, if the child usually falls asleep at 8:30 p.m. and sleepwalks at 10:00 p.m., then the parent should institute scheduled awakenings at 9:30 p.m. These nightly awakenings should be continued for 2 to 4 weeks. If the events recur following discontinuation of the
scheduled awakenings, they may be reinstituted for several weeks.

PROGNOSIS
Most children naturally stop sleepwalking or experiencing sleep terrors in childhood. By age 8 years, 50% of children with sleepwalking or sleep terrors no longer experience episodes, and most cases resolve spontaneously following puberty as a result of the dramatic decrease in SWS. However, about 10% of individuals continue to experience sleepwalking episodes for 10 or more years, and thus persistence into young adulthood does occur. This is particularly relevant for college students, who are more likely to have trigger factors for disorders of arousal, including inadequate and/or disrupted sleep and excessive caffeine consumption. Patients should be encouraged to share this information with college health services and roommates, and to request a first-floor living space in dormitories.

Tips for Talking to Parents
- Explain that confusional arousals, sleepwalking, and sleep terrors are a neurodevelopmental phenomenon, and they are neither indicative of an underlying psychological issue nor result in psychological harm. Help parents understand that sleep terrors in particular are far more upsetting for the observing caregiver than for the child experiencing them.
- Ensure safety measures, including locking of all outside doors and windows and installing an alarm system to notify parents that the child has left the bedroom.
- Encourage parents to share information about sleepwalking and sleep terrors with other caregivers, especially if the child is sleeping away from home.
- Suggest an appropriate sleep schedule, which will ensure adequate sleep.
- Discourage parental intervention during an episode, as this is likely to increase agitation and prolong the event.
- Discuss potential trigger and exacerbating factors, including insufficient sleep, sleeping in an unfamiliar environment, and caffeine.
- Discuss the risks and benefits of treatment options, including medications and scheduled awakening in more problematic cases.
- Discuss prognosis, including the likelihood of disappearance by adolescence. If episodes do persist, discuss safety measures in regard to living away from home (e.g., avoidance of bedrooms on upper floors).

See Appendix D5 for a parent handout on sleepwalking and D6 on sleep terrors.

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Sleep Related Rhythmic Movements: Head Banging, Body Rocking, and Head Rolling

**Head banging** (*jactatio capitis nocturna*), **body rocking**, and **head rolling** are specific sleep related phenomena that fall under the diagnostic category of sleep related movement disorders. This diagnostic category includes restless legs syndrome (RLS), periodic limb movement disorder (PLMD), and bruxism. Sleep related rhythmic movements are characterized by repetitive, stereotyped, and rhythmic movements or behaviors that involve large muscle groups. These behaviors typically occur with the transition to sleep at bedtime, but also at naptimes and following nighttime arousals. They can also occur during sleep, usually in stages 1 and 2 sleep, occasionally in slow-wave sleep, and rarely in REM sleep. Children typically engage in these behaviors as a means of soothing themselves to (or back to) sleep, and the duration of these behaviors can be from minutes to several hours.

In most instances, rhythmic movement behaviors are benign. Sleep is not significantly disrupted as a result of these movements, and, although sometimes distressing to caregivers, associated significant injury is rare. These behaviors typically occur in normally developing children, and in the vast majority of cases their presence does not indicate that there is some underlying neurologic or psychological problem. However, in situations that involve children with psychiatric or neurologic disorders, such as developmental delays, autism, or blindness, there may be a risk of potential injury due to the intensity and frequency of the behaviors. These children are much more likely to also engage in these behaviors while awake.

Because rhythmic movement behaviors are considered to be benign in typically developing children, the *International Classification of Sleep Disorders (3rd ed.*) does not classify these behaviors as pathologic unless there are clinical consequences, such as interference with normal sleep, resulting in significant impairment in daytime function, or resulting in injury. For the practitioner, however, note that these behaviors are often of significant concern to parents, even though they may not be considered a “disorder” per se.

**EPIDEMIOLOGY**

- **Prevalence and age of onset:** Studies indicate that approximately two-thirds (59%) of all infants engage in some type of rhythmic behavior. Body rocking is most common (43%), followed by head rolling (24%) and head banging (22%). By 18 months of age, the prevalence has decreased to 33% and to only 5% at 5 years of age; rarely do these behaviors persist into adulthood. Onset of rhythmic movement behaviors is typically prior to 1 year of age, with body rocking starting at an earlier age than head banging.

- **Gender:** Some studies report sleep related rhythmic movements to be found equally in boys and girls, whereas others report a higher prevalence in boys.

**ETIOLOGY AND RISK FACTORS**
Vestibular stimulation: An increased need for kinesthetic stimulation may be related to rhythmic behaviors, as these rhythmic behaviors are reported to be soothing.

Nighttime arousals: Any factor that increases nighttime arousals or awakenings, such as sleep-disordered breathing, pain, or gastroesophageal reflux, may provide increased opportunities for the behavior to occur. Environmental sleep disrupters, such as noise from within or outside the household, can also lead to increased arousals.

Parental attention: Caregivers can inadvertently reinforce rhythmic behaviors by providing attention.

Developmental disabilities: It should be emphasized that the majority of children who engage in rhythmic behaviors are developmentally normal and healthy. However, sleep related rhythmic movements, especially when persistent or also occurring during the day, can be associated with intellectual disabilities, pervasive developmental disorders (including autism spectrum disorders), and psychopathology.

RLS: Rhythmic movement disorders have been associated with RLS.

Additional factors: Other associated conditions include environmental stress, lack of environmental stimulation, and self-stimulation. Some studies have suggested that there is a familial predisposition.

PRESENTATION AND SYMPTOMS
Children and adolescents with rhythmic movements typically present with one of the following:

- **Body rocking** ("shuttling") presents as rocking forward and back, without head banging, usually while on the hands and knees. Body rocking usually begins earliest, at about 6 months of age.

- **Head banging**, starting around 9 months, can occur in multiple forms:
  - Lying prone and lifting the head to bang down into a pillow or the mattress.
  - Rocking on hands or knees and banging head into the headboard or wall.
  - Sitting upright and banging head back into the headboard or wall.

- **Head rolling** involves side-to-side movements of the head, usually in the supine position, with an average age onset of 10 months.

- **Body rolling** is less common than the other behaviors, and involves rolling of the entire body in a lateral manner (side to side).

Diagnostic Criteria
See Table 12.1.

Associated Features

- **Rhythmic humming or chanting**: These and other sounds may accompany the movements.

- **Insomnia**: Some report insomnia, either at sleep onset or prolonged nighttime awakenings; however, it is not clear whether the insomnia is induced by the movements or simply accompanies it.

- **Disruption to parental sleep (and that of other family members)**: This may occur if the rhythmic behavior is loud and especially if it occurs throughout the night.

- **Parental anxiety**: Parents may feel anxiety because they are commonly concerned about the risk of injury, especially head trauma. They may also associate these behaviors with intellectual disabilities and/or autism, and, thus, may have concerns about their child's development.
TABLE 12.1. Diagnostic Criteria: Sleep Related Rhythmic Movement Disorder

A. The patient exhibits repetitive, stereotyped, and rhythmic motor behaviors involving large muscle groups.
B. The movements are predominantly sleep related, occurring near nap or bedtime, or when the individual appears drowsy or asleep.
C. The behaviors result in a significant complaint as manifest by at least one of the following:
   1. Interference with normal sleep.
   2. Significant impairment in daytime function.
   3. Self-inflicted bodily injury that requires medical treatment if preventive measures are not used.
D. The rhythmic movements are not better explained by another movement disorder or epilepsy.

*ICD-9-CM: 327.59; ICD-10-CM: G47.69

Note: When there are no clinical consequences of the rhythmic movements, the rhythmic movements are simply noted but the term rhythmic movement disorder is not employed.


EVALUATION

- **Medical history:** The history is generally unremarkable. It should include a complete review for additional factors that may result in increased arousals, including sleep-disordered breathing, reflux, and pain (e.g., ear infection, headaches).

- **Developmental history:** The history is usually normal. However, further evaluation for developmental delays in a child with persistent rhythmic behaviors, especially if they also occur during the day, may be warranted. Because sensory deprivation can contribute to the etiology of rhythmic movement behaviors, in severe or persistent cases the possibility of child neglect or abuse should be considered.

- **Family history:** The history may be positive, as there appears to be a genetic component.

- **Behavioral assessment:** An assessment usually does not indicate the existence of behavioral problems, but parental response to the behavior (reinforcement) should be explored. Children who have other self-stimulatory behaviors (e.g., rumination) may be more likely to have experienced neglect.

- **Physical examination:** The examination is usually unremarkable. Occasionally, children may have minor contusions or calluses at the point of impact, but serious injury is rare.

DIFFERENTIAL DIAGNOSIS

- **Developmental delay,** as children with developmental delay may engage in self-injurious behavior or self-stimulatory behaviors, usually also seen during the day.

- **Medical disorders,** including neurologic disorders, blindness, ear infections, pain, and gastroesophageal reflux, may result in head banging or body rocking.

- **Seizures,** although usually these can be easily differentiated by clinical history. One distinguishing
feature is that children have voluntary control over rhythmic movements.

- **Other movement disorders**, including sleep starts (hypnic jerks), excessive fragmentary myoclonus, RLS or PLMD (see Chapter 16), and benign sleep myoclonus of infancy.

### MANAGEMENT

#### When to Refer

If symptoms of other underlying sleep disturbances (e.g., obstructive sleep apnea, RLS) are present, the patient should be referred to a sleep specialist. Where there are concerns regarding similar behaviors during the daytime, a referral for a developmental assessment is warranted. Referral to a pediatric neurologist is seldom warranted except in the case of persistent or severe behaviors or if there is a concern regarding possible seizure disorder.

#### Treatment

Typically, little needs to be done if a child engages in rhythmic movements at sleep times, although parents should be instructed about safety, behavioral management, and, occasionally, alternative treatments. Usually, the most important aspect in management of head banging or body rocking is reassurance to the family that this behavior is normal, common, benign, and self-limited; most children outgrow it by age 2 or 3 years.

**Safety**

All parents should be instructed to regularly tighten all screws and bolts on their child's crib or bed and to install guardrails on beds.

**Behavioral Management**

- **Discontinue attempts to protect the child**: Even if the child engages in vigorous head banging, it is unlikely that injury will occur. Installing extra bumpers on the crib or placing pillows in strategic places is usually not needed and is rarely effective, although parents may be reassured by these measures.

- **Avoid reinforcement of the behavior**: If the parents respond to the child, they may be inadvertently reinforcing the behavior. In these cases, it may be best for the parents to ignore the behavior both at bedtime and throughout the night.

- **Dampen the noise**: Moving the crib or bed away from the wall and oiling screws and bolts can dampen noise.

- **Increase sleep**: Increasing naptimes and moving bedtime earlier may result in diminished sleep deprivation, minimizing the rhythmic behaviors related to increased nighttime arousals.

**Additional Treatments**

- **Treat underlying sleep disrupters** (e.g., sleep-disordered breathing, RLS), which may significantly decrease the frequency and duration of the rhythmic behavior.

- **Treat concurrent medical problems**, e.g., ear infections, reflux.

- **Reduce environmental sleep disrupters**, such as household noise that may arouse the child during the night. Installing a noisy fan or white noise machine to mask household noise in the child's room can be helpful.

- **Consider pharmacologic treatment**, as, in severe or extremely persistent cases, treatment with a
benzodiazepine may be appropriate, especially in combination with extinction if there is an attentional component. Studies have found clonazepam to be effective. Hydroxyzine and tricyclic antidepressants have also been used for severe cases. At times, a short course of 2 to 3 weeks may be enough to disrupt the habit basis of these behaviors.

- **Sleep restriction**, as one study found that a 3-week mild sleep restriction regimen, in combination with a hypnotic, was effective for school-aged children. Patients were sleep restricted by 1 hour at night for 1 week in combination, with the use of a low-dose hypnotic, followed by 1 week of just 1 hour of sleep restriction, and then sleep time was gradually returned to baseline by 10-minute increments nightly.

**PROGNOSIS**

Most young children outgrow rhythmic behaviors by age 3 years (they disappear in 90% of children by age 4 years), although some children continue to engage in these behaviors throughout childhood, adolescence, and even into adulthood.

**Tips for Talking to Parents**

- **Explain the nature of rhythmic behaviors**, emphasizing that these are soothing behaviors for the child, similar to thumsucking.

- **Emphasize the benign nature of these behaviors**, which neither indicate an underlying neurodevelopmental or psychological condition nor result in injury (“brain damage”) to the child.

- **Review any factors that may be increasing difficulty falling asleep and/or nighttime arousals**, including parental attention, sleep disrupters (e.g., sleep-disordered breathing, reflux, pain), or environmental factors (e.g., noise, room temperature).

- **Encourage parents to ignore the behavior**, so that the behavior is not reinforced and perpetuated by parental attention.

- **Review safety issues**, including tightening of crib or bed bolts.

- **Discuss measures to reduce impact on other family members’ sleep**, such as moving the crib or bed away from the wall and using white noise in family members’ rooms to mask sound.

- **Discuss the risks and benefits of treatment options**, if medication is being considered.

*See Appendix D7 for a parent handout on head banging and body rocking.*
Bruxism, a sleep related movement disorder, is defined as the involuntary nonfunctional repetitive grinding or clenching of the teeth during sleep. The sound of the teeth grinding, although not always audible, can be disturbing to others and/or is often the reason for parental concern. Bruxism can eventually lead to dental erosion, jaw and/or facial pain, and tissue damage over time, although these are unlikely to be the presenting complaints in children and adolescents. Bruxism typically occurs during stages 1 and 2 non-REM sleep and infrequently in REM sleep. It typically occurs in association with sleep arousals, although it infrequently leads to prolonged awakenings or significant sleep disruption. Bruxism persists into adulthood in two-thirds of cases.

**Bruxism**

Bruxism is the repetitive grinding or clenching of teeth during sleep.

**Epidemiology**

Although occasional teeth grinding is common in the general population, the repetitive and persistent teeth grinding characteristic of bruxism occurs with a much lower frequency. Several studies have been conducted on the prevalence of bruxism in children based on parental report. Prevalence rates range from as low as 3.5% to as high as 49.6% in children and adolescents, with the majority of studies finding prevalence rates of 14% to 17% in children and 12% in adolescents. All studies report a decrease in prevalence with age. The majority of studies find no differences across gender, with equal prevalence rates in boys and girls.

In addition, bruxism can begin at a very young age, as soon as the upper and lower teeth have erupted.

**Etiology and Risk Factors**

Recent studies have shed light on the underlying pathophysiology of bruxism. Voluntary chewing appears to involve fairly complex serotonergic and dopaminergic pathways in the midbrain and prefrontal cortex; bruxism has been linked in several studies to dysfunction in the dopaminergic system in particular. During sleep, these jaw muscle contractions, termed rhythmic masticatory muscle activity (RMMA), can be a series of repetitive activity (phasic muscle contractions) or isolated and sustained jaw clenching (tonic contractions).

- **Prolonged breast-feeding or bottle-feeding** has been found to be associated with bruxism in preschoolers, as well as a habit of biting on things.
- **Decreased nighttime sleep** has been found to be associated with bruxism.
- **Occlusal misalignments** due to malocclusion, dental crowding, or dental trauma may increase the risk of bruxism.
- **Light and noise** in the bedroom may disrupt sleep and increase the likelihood of bruxism.
- **Allergies and nasal obstruction** have been reported to be associated with bruxism.
- **Gastroesophageal reflux** has been linked to bruxism in adults.
- **Patients with cerebral palsy and intellectual disabilities** show increased risk for bruxism.
- **Alcohol and stimulant medications** (e.g., amphetamines) can also exacerbate bruxism.
- **Nicotine** has also been reported to exacerbate symptoms in adults.

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Selective serotonin reuptake inhibitors (SSRIs) have been reported to increase the risk of bruxism.

Primary sleep disturbances (e.g., obstructive sleep apnea) may trigger bruxism.

Personality types, including being highly motivated and those who are highly vigilant have an increased risk for bruxism.

Family history increases the risk of bruxism in a child. Sleep bruxism tends to occur in family members, with 20% to 50% of individuals with bruxism having at least one family member with a history of teeth grinding. Children with a parent with a history of bruxism are almost twice as likely to grind their teeth.

Attention-deficit/hyperactivity disorder, autism spectrum disorders, and Down syndrome have all been associated with increased rates of bruxism.

PRESENTATION AND SYMPTOMS

Bruxism is characterized by clenching, gnashing, or grinding of the teeth during the night, which may be observed and/or heard by parents or others, especially those who share a bedroom with the child or adolescent. There is significant night-to-night variability and most children/adolescents with bruxism are not aware of the behavior. Bruxism can be primary or secondary (associated with use of psychoactive medications, recreational drugs, or medical disorders such as Down syndrome).

Diagnostic Criteria
See Table 13.1.

Associated Features

- **Muscle pain:** Children may complain of painful and swollen jaw (masseter and temporal) muscles, limited jaw opening, or a "clicking" jaw. Bruxism can also lead to temporomandibular joint (TMJ) dysfunction.

- **Headache:** Children or adolescents with bruxism may complain of neck and shoulder pain, or bitemporal headache in the morning ("TMJ syndrome"). Headaches are 4 times more likely in individuals with bruxism.

- **Sensitive teeth:** Patients may complain of tooth discomfort associated with extremes in food temperature.

- **Daytime bruxism:** Children and adolescents may also grind their teeth during the day, although these two behaviors appear to be etiologically different.

- **Dental damage:** Dental damage can include erosion of the teeth, damage to the tissues surrounding the teeth (recession and inflammation of the gums), and resorption of the alveolar bone.

TABLE 13.1. Diagnostic Criteria: Sleep related Bruxism

Diagnostic criteria:

A. The presence of regular or frequent tooth grinding sounds occurring during sleep.

B. The presence of one or more of the following clinical signs:
   1. Abnormal tooth wear consistent with above reports of tooth grinding during sleep.
   2. Transient morning jaw-muscle pain or fatigue; temporal headache; and/or jaw locking upon
awakening consistent with above reports of tooth grinding during sleep.

ICD-9-CM: 327.53; ICD-10-CM: G47.83


- **Daytime behavior problems**: Bruxism can be associated with increased arousals from sleep, resulting in insufficient sleep and concomitant daytime behavior problems, such as inattention and oppositional behavior.
- **Mental health issues**: Studies indicate that bruxism can be associated with stress and anxiety, although not with depression.

**EVALUATION**

- **Medical history**: The history is generally unremarkable, but may include symptoms of head and jaw pain and dental problems.
- **Developmental and school history**: The history is usually normal.
- **Family history**: The history may be positive for other family members with bruxism.
- **Behavioral assessment**: An assessment may show possible contributory factors, such as anxiety and stress.
- **Physical examination**: Evaluation for dental erosion and tissue damage should be conducted. There may be tenderness over the maxillary and temporal areas.
- **Diagnostic tests**: Tests are not indicated.

**Overnight Sleep Study (Polysomnography) is not Warranted**

According to practice parameters published by the American Academy of Sleep Medicine, polysomnography is not routinely indicated for evaluation of children with sleep-related bruxism.

**DIFFERENTIAL DIAGNOSIS**

Diagnosis of bruxism is usually straightforward. Differential diagnosis includes the following:

- **Dental disorders and other TMJ disorders**.
- **Other faciomanibular activities**, including faciomanibular myoclonus, gastroesophageal reflux, and abnormal swallowing.
- **Seizure disorder**: In rare cases, abnormal jaw movements may be associated with partial complex or generalized seizures.

**MANAGEMENT**
When to Refer
Children or adolescents with bruxism who also have significant psychiatric or behavioral issues should be referred to a mental health professional. In addition, referral to a dentist is appropriate if there are any dental concerns, including damage to dentition.

Treatment
Because bruxism is usually self-limited, treatment in children and adolescents is rarely warranted. However, since stress is a major contributing factor in many cases, possible sources of stress should be explored and eliminated if possible. Specific stress management techniques that have been shown to be helpful include the introduction of relaxing bedtime rituals, progressive relaxation exercises, hypnotherapy, and biofeedback. In more problematic cases, additional management strategies may include the following:

- **Sleeping position:** Lying in the supine position with support for the neck may alleviate muscle strain on the jaw and neck.
- **Pain relief:** Nonsteroidal anti-inflammatory medications are occasionally indicated to relieve jaw pain. Local application of heat may be helpful.
- **Dental appliances:** For children and adolescents with dental damage or persistent complaints of jaw pain, referral to a dentist for a nighttime intraoral appliance may be warranted.
- **Pharmacotherapy:** Although REM-suppressing medications, such as benzodiazepines and muscle relaxants, have been shown to be effective for severe bruxism in adults, their use is rarely indicated in the pediatric population. Some recent studies have provided some support for use of hydroxyzine. It should be noted that serotonergic drugs such as SSRIs may cause or worsen symptoms of bruxism.
- **Psychological treatment:** If an underlying anxiety disorder is suspected, psychological treatment may be warranted.

PROGNOSIS
Bruxism, although most often self-limited in children, may be chronic and is likely to be exacerbated by stress.

Tips for Talking to Parents
- **Explain what bruxism is** in simple terms, including that this behavior is usually self-limited and benign in infants and children, although there is a possibility of dental damage in older children and adolescents.
- **Explain risk factors** for bruxism, primarily stress.
- **Discuss treatment options** (if warranted), including stress management and/or psychological treatment.
- **Refer for a dental examination** in older children and adolescents.

*See Appendix D8 for a parent handout on bruxism.*
Sleep Enuresis

Sleep enuresis (bedwetting) is characterized by recurrent involuntary voiding during sleep. For a diagnosis, it must occur a minimum of twice a week after the age of 5 years. Enuresis is considered primary if the child has never had a dry period of 6 months, and secondary if the child had previously been consistently dry for 6 months but bedwetting is now occurring at least twice a week. Both primary and secondary enuresis must be present for at least 3 months. Monosymptomatic enuresis (uncomplicated enuresis) involves normal voiding during the night without any other lower urinary tract problems. Non-monosymptomatic enuresis occurs in children with any other lower urinary tract symptoms and with a history of bladder dysfunction. Enuresis can occur in any stage of sleep, with most episodes occurring in the first half of the night.

**Enuresis**

Enuresis involves involuntary voiding during sleep, at least twice a week for at least 3 months beyond the age of 5 years.

It is important to note that becoming consistently dry at night is a developmental maturation process. Children with primary enuresis typically fail to wake from sleep in response to bladder sensations or fail to inhibit bladder contractions during sleep. These skills are acquired with development, with some children taking much longer to achieve the skill of being dry at night compared to daytime continence. Thus, enuresis is not even diagnosed until age 5 years and typically not treated until after age 7 years.

Although enuresis is not problematic per se, it can have a significant impact on children and their families. Many children feel extremely embarrassed by their bedwetting and parents often punish children for something that they are unable to control.

**EPIDEMIOLOGY**

Primary enuresis steadily decreases by age with a spontaneous remission rate of approximately 5% to 10% per year. It is experienced by about 30% of 4-year-olds, 10% of 6-year-olds, 7% of 7-year-olds, 5% of 10-year-olds, 3% of 12-year-olds, and 1% to 2% of 18-year-olds. Boys are 3 times more likely to experience enuresis than do girls. Secondary enuresis accounts for less than 25% of cases. Cross-cultural studies indicate quite consistent findings.

Infrequent bedwetting, ranging from less than twice a week to less than once a month, is even more common. The more severe the bedwetting, in terms of frequency, the more likely it is to persist.

**ETIOLOGY AND RISK FACTORS**

**Primary Enuresis**

Previously, enuresis was considered to be the result of nocturnal polyuria, detrusor overactivity, and an increased arousal threshold. However, more recent studies have found that although nocturnal polyuria is common in children with enuresis, not all of them have polyuria. Furthermore, not all children with polyuria have vasopressin deficiency, and in some children nocturnal detrusor overactivity has been indicated.
Maturational delay: Maintaining bladder control throughout the night is a maturational process, with enuresis related to central nervous system maturational delays.

Lowered arousal threshold: A child with enuresis may have decreased arousability and fail to awaken in response to a full bladder. This may be associated with sleep-disordered breathing, possibly due to fragmented sleep or another mechanism.

Positive family history: There is commonly a genetic component in primary enuresis. Studies indicate a 74% prevalence rate if both parents were enuretic during childhood and 44% if one parent had enuresis as a child, in comparison to 15% if neither parent had a history of enuresis. Furthermore, the risk is 10.1 times higher in the presence of a history of paternal enuresis and only 3.6 times higher in the presence of maternal enuresis. Genetic linkage of sleep enuresis is autosomal dominant and has been noted for chromosomes 22q, 13q, and 12q.

Psychiatric and neurodevelopmental conditions: Enuresis is more prevalent in children with attention-deficit/hyperactivity disorder (ADHD; with an almost three-fold increase in prevalence) and neurodevelopmental disorders, such as developmental delays and mental retardation.

Reduced nocturnal bladder capacity: Enuresis can be due to decreased functional bladder capacity and an inability to hold urine at night. Bladder capacity increases with age from approximately 6 ounces at age 5 years to 11 ounces at age 10 years. Any condition that further reduces bladder capacity, such as constipation or cystitis, will result in increased enuresis.

Decreased vasopressin production during sleep: Some children do not have the typical increase in vasopressin during sleep, resulting in polyuria (high urinary volume) that exceeds bladder capacity. If the child does not arouse in response to the sensation of a full bladder, sleep enuresis will occur.

Secondary Enuresis

Inability to concentrate urine: Enuresis can be the result of diabetes mellitus, diabetes insipidus, or sickle cell disease.

Increased urinary production: Enuresis can result from intake of caffeine or diuretics.

Urinary tract pathology: A child with enuresis may have other symptoms, including urinary tract infections (UTIs), irritable bladder, or a malformation of the genitourinary tract.

Chronic constipation and encopresis: Constipation occurs in over 50% of children with secondary nocturnal enuresis.

Neurologic pathology: There may be a neurologic component to enuresis in some children, including nocturnal epilepsy.

Sleep disorders: Sleep disorders, primarily obstructive sleep apnea (OSA), have been associated with both primary and secondary enuresis. Studies indicate that 8% to 47% of children with OSA have sleep enuresis; the mechanism has been postulated to involve disruption of the normal nocturnal pattern of secretion of the antidiuretic hormone vasopressin by OSA-related sleep fragmentation. Resolution of enuresis following adenotonsillectomy in these children occurs in 44% to 77% of cases. Children with severe OSA and those with increased stage 2 sleep are more likely to have resolution of enuresis. Children with enuresis have also been found to have increased periodic limb movements and arousals during sleep.

Psychosocial stressor: Enuresis may result from a psychosocial stressor, such as parental divorce or death in the family.
PRESENTATION AND SYMPTOMS

Bedwetting occurs at least twice a week for at least 3 months. Some children have enuresis on a nightly basis and may even have multiple episodes per night, whereas in other children it is much less frequent.

Diagnostic Criteria

See Tables 14.1 and 14.2.

Associated Features

- **Daytime incontinence**: Involuntary voiding during wakefulness, together with nighttime enuresis, is more likely to be associated with an organic cause.
- **Constipation**: About 30% of children with enuresis report constipation, and enuresis resolves in about two-thirds when the constipation is treated.
- **Impact on social functioning**: Because of potential embarrassment, many children and adolescents with sleep enuresis avoid social situations, such as overnight visits to friends or summer camps. Studies also indicate increased vulnerability to victimization.
- **Emotional toll**: Children and adolescents with enuresis may have negative self-thoughts, including lowered self-esteem, guilt, shame, and embarrassment. There is often a fear of being “found out.”

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**TABLE 14.1. Diagnostic Criteria (ICSD-3): Sleep Enuresis**

<table>
<thead>
<tr>
<th>Primary Sleep Enuresis</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. The patient is older than 5 years.</td>
</tr>
<tr>
<td>B. The patient exhibits recurrent involuntary voiding during sleep, occurring at least twice a week.</td>
</tr>
<tr>
<td>C. The condition has been present for at least 3 months.</td>
</tr>
<tr>
<td>D. The patient has never been consistently dry during sleep.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Secondary Sleep Enuresis</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. The patient is older than 5 years.</td>
</tr>
<tr>
<td>B. The patient exhibits recurrent involuntary voiding during sleep, occurring at least twice a week.</td>
</tr>
<tr>
<td>C. The condition has been present for at least 3 months.</td>
</tr>
<tr>
<td>D. The patient has previously been consistently dry during sleep for at least 6 months.</td>
</tr>
</tbody>
</table>

*ICD-9-CM: 788.36; ICD-10-CM: N39.44


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**TABLE 14.2. Diagnostic Criteria (DSM-5): Enuresis (307.6)**

*http://obgynebooks.com*
A. Repeated voiding of urine into bed or clothes, whether involuntary or intentional.
B. The behavior is clinically significant as manifested by either a frequency of twice a week for at least 3 consecutive months or the presence of clinically significant distress or impairment in social, academic (occupational), or other important areas of functioning.
C. Chronological age is at least 5 years (or equivalent developmental level).
D. The behavior is not due exclusively to the direct physiological effect of a substance (e.g., a diuretic, an antipsychotic medication) or another medical condition (e.g., diabetes, spina bifida, a seizure disorder).

Specify whether:
- Nocturnal only
- Diurnal only
- Nocturnal and diurnal


- **Increased psychological problems:** Children who wet the bed are more likely to have both internalizing and externalizing problems, especially oppositional behaviors and conduct problems.

- **Increased prevalence of sleep disorders:** In particular, children with enuresis often have sleep-disordered breathing (8%-47%), and vice versa. A recent study reported that about 25% of a sample of children undergoing adenotonsillectomy for OSA had nocturnal enuresis, which subsequently resolved in about half within 1 month after surgery. While the exact mechanism is not clear, elevated nocturnal levels of atrial natriuretic peptide have been found in patients with OSA. Other sleep disturbances, such as snoring, periodic limb movements in sleep, sleep terrors, and restless sleep, have also been reported in association with enuresis. Snoring and restless sleep are also common.

- **Increased psychosocial stress:** Secondary enuresis is more common in children with a recent psychosocial stressor, such as parental divorce, physical or sexual abuse, or neglect.

**Enuresis is Expensive**

The cost of coping with a child with enuresis should not be underestimated; neither should its financial impact on families, especially low-income families. Overnight diapers are expensive ($125-$300 per year), as is the cost of extra loads of laundry, especially for those who need to go to a laundromat ($500-$700 per year). Some families literally cannot afford the expense of having a child who wets the bed.

**EVALUATION**

- **Enuresis history:** The age of onset, duration, and severity of enuresis and the duration of continence should be determined for secondary enuresis.

- **Medical history:** The history is generally benign. Presence of daytime incontinence, constipation, and genitourinary symptoms should be assessed, as well as other symptoms related to secondary causes.
**Developmental and school history:** The history is usually normal, although enuresis can be related to developmental delays.

**Family history:** The history is commonly positive for childhood enuresis.

**Behavioral assessment:** Behavioral concerns are uncommon in children with primary enuresis, but are common in children with secondary enuresis. Behavioral concerns include depression, anxiety, conduct disorders, and ADHD.

**Physical examination:** The examination is generally unremarkable, although should include a urinalysis. If daytime incontinence is also present, then evaluation of urinary abnormalities should be considered. Evaluation for diabetes, UTI, seizure disorder, sickle cell disease, and neurologic disorders may be considered.

**Diagnostic tests:**
- **Urinalysis** and urine culture to assess for glucosuria and UTI, particularly in secondary enuresis.
- **Enuresis diary** that documents frequency of bedwetting events.
- **Overnight polysomnography** may be warranted if there is a suspicion of OSA or periodic limb movement disorder, but is not a routine part of the evaluation for enuresis.

**Overnight Sleep Study (Polysomnography)**

According to practice parameters published by the American Academy of Sleep Medicine, children with nocturnal enuresis should be clinically screened for the presence of comorbid sleep disorders and polysomnography should be performed if there is a suspicion for sleep-disordered breathing or periodic limb movement disorder.

**DIFFERENTIAL DIAGNOSIS**

- **Secondary causes of enuresis** need to be considered (e.g., UTI, diabetes).
- **Nocturnal seizures** may result in incontinence, but typically are accompanied by other features suggestive of a seizure disorder (e.g., stereotypical movements, altered consciousness).

**MANAGEMENT**

**When to Refer**

Children or adolescents with persistent enuresis, daytime incontinence, genital abnormalities, or a history of recurrent UTIs should be referred for a urology consult. If other potentially contributing medical disorders exist, such as diabetes, a seizure disorder, or OSA, a referral is warranted. A specialist in treating behaviorally based issues can be beneficial in the implementation of behavioral treatments. Where there are concerns regarding stress or other psychological issues, a referral to a mental health specialist is warranted. Finally, a referral is appropriate for those who have not responded to treatment after 6 months.

**Treatment**

**Reassurance and education** of the child and family is the first step in the management of sleep enuresis,
followed by treatment of secondary causes, including urologic and neurologic issues, but especially constipation and OSA. Treatment of the contributing cause may cure or reduce the incidence. Furthermore, the prevalence of this condition and the fact that it often resolves spontaneously should be reviewed.

If the enuresis is not distressing to the child, treatment is not warranted, especially given the high spontaneous remission rate. For young children (ages 5-7 years), treatment is typically not warranted or recommended. Normalization of the behavior (as well as use of overnight diapers or training pants) is often the best option.

In cases in which the enuresis is distressing (e.g., in older children and adolescents) or is impacting functioning (e.g., social functioning), the following treatments should be considered. Overall, treatment is considered successful with 14 consecutive dry nights. Lack of success is defined as less than a 50% decrease in enuresis, with partial success indicated by a 50% to 90% decrease.

- **Overall good bladder health**: Bladder health can improve sleep enuresis.
  - **Void regularly**. Children should be encouraged to void regularly (every 2-3 hours) and avoid urgency. Many children avoid urinating at school, and thus voiding should be encouraged. Children should also use the bathroom at bedtime and every time they wake up during the night.
  - **Increased fluid intake during the day**. Increased fluid intake during the day can also be beneficial, both for bladder health and to reduce constipation. Fluid intake should occur primarily in the morning and early afternoon.
  - **Reduced evening fluid intake**. In the evenings, however, fluids should be limited.

- **Behavioral treatments**: The following treatments can be effective.
  - **Enuresis alarms** are the first-line treatment and the most effective for sleep enuresis, although it is important to note that they require significant effort and motivation by the child and family as well as persistence over time. There are many different types of enuresis alarms, including pads that go on the bed or alarms that attach to a child's underwear, but all are based on the premise of a moisture sensor that gets triggered by wetting. It eventually conditions a child to respond to the sensation of a full bladder. Treatment may take months before success is attained; typical treatment is 16 weeks or until 14 consecutive dry nights are achieved. The initial response rate is approximately 60%, with a cure rate of approximately 40%. The benefits of alarm treatment are the short-term and long-term efficacy and that it is noninvasive. The negatives are the relative cost of treatment and the amount of motivation required by the family.
  - **Awakening the child** at times when enuresis typically occurs or prior to parents going to bed can decrease events. Parents, however, may find that it is difficult to waken their child or that their child resists middle-of-the night trips to the bathroom. Thus, parents may find this treatment frustrating.
  - **Implement sticker charts** and other reward systems for dry nights.
  - **Minimize fluids** in the evening after dinner.
  - **Avoid caffeine**.
  - **Bladder training**, which involves having the child drink increasing amounts of fluid and progressively waiting longer to void.
  - **Involve the child in changing the bed** by having him or her help remove wet sheets and remake the bed.
  - **Use of rewards**. Families can institute reward systems for behaviors that are under the child's control,
such as drinking fluids during the day and voiding frequently. It is typically not recommended to provide rewards for dry nights, which the child is typically not able to control.

- **Pharmacologic treatment**: Using drugs is not curative but can temporarily resolve enuretic events. Medication is typically not given to children under the age of 7 years, and should be used if behavioral treatments fail. Some children benefit from nightly medication use, whereas others use it on a night-to-night basis, such as for sleepovers or overnight camp.

- **Desmopressin (DDAVP)** is the only other medication that is FDA-approved for enuresis. Desmopressin, an analogue of vasopressin, reduces urine volume. Oral preparations should be used. **Note that desmopressin intranasal formulations are no longer indicated by the FDA for the treatment of primary nocturnal enuresis due to hyponatremia that may result in seizures and death. Studies indicate that 60% to 70% of children respond to treatment (30% are full responders and 40% have a partial response), with 80% relapse following discontinuation. Desmopressin tablets (0.2-0.4 mg) should be taken at least 1 hour before bedtime and oral melt tablets (120-140 μg) 30 to 60 minutes before lights out. Desmopressin is most effective in children with normal bladder capacity and requires nightly administration. It is up to the family to decide whether to use nightly administration or only on specific nights (e.g., sleepovers). If nightly use is chosen, drug holidays should be scheduled to assess whether the medication is still needed.

- **Imipramine (Tofranil)** is FDA-approved for the treatment of enuresis in children. Doses range from 25 mg for children older than 6 years (20 kg-25 kg) to 50 mg to 75 mg for children older than age 11 years. A starting dose of 25 mg 1 hour prior to bedtime for 1 week is recommended, increasing the dose as needed. About 20% of children become dry while on treatment. Relapse typically occurs with discontinuation. The mode of action is unclear but can be effective with refractory enuresis. Side effects can include lethargy, drowsiness, agitation, and gastrointestinal upset. Because of the risk of cardiotoxicity in accidental overdose, parents should be cautioned to carefully monitor medication. Drug holidays of approximately 2 weeks should also be regularly scheduled to decrease the risk of tolerance, as well as to assess for necessity.

- **Anticholinergics (Detrol, Ditropan)** reduce muscle spasms in the bladder and are most effective for those children and adolescents with an overactive bladder and small functional bladder capacity. They are typically not considered first-line therapy and used mainly in nonmonosymptomatic nocturnal enuresis.

**Alarm Training or Desmopression: Which Should be the First-Line Treatment?**

Alarm training is typically recommended to try first if the child is motivated to become dry and there is significant family support. Desmopressin should be tried first if a short-term solution is needed or there is insufficient motivation and family support for alarm training.

**PROGNOSIS**

Most children naturally stop bedwetting, with a spontaneous remittance rate of 15% per year.

**Tips for Talking to Parents**

- Educate parents and child about enuresis, including lack of volition of the behavior and eliminating
punishment and negative consequences of nighttime episodes.

- **Discuss whether treatment is necessary**, given that most children outgrow enuresis at a rate of 15% per year.

- **Discuss good bladder health**, including frequent voiding during the day and increased fluid intake during the day (to prevent constipation), while decreasing fluid intake in the evening.

- **Consider bedwetting alarms**, for those children over the age of 7 years and for those for whom enuresis is having a significant impact on functioning or family stress.

- **Review medication alternatives**, especially for occasional use such as sleepovers or overnight camp.

*See Appendix D9 for a parent handout on enuresis.*
Sleep related breathing disorders (SRBD) in children are characterized by an abnormal respiratory pattern during sleep and encompass a broad spectrum of respiratory disorders that occur in children of all ages, from neonates through adolescents. **Obstructive SRBD**, which is the focus of this chapter, includes pathology ranging from mouth breathing during sleep to primary snoring to obstructive hypoventilation to hypopneas accompanied by reduced airflow to frank apneas with complete cessation of airflow.

**Obstructive Sleep Apnea**

Obstructive sleep apnea (OSA) is characterized by repeated episodes of prolonged upper airway obstruction during sleep in the face of continued or increased respiratory effort, resulting in complete or partial cessation of airflow at the nose and/or mouth, and often in hypoxia, hypercapnia, and sleep fragmentation. Etiology in children is commonly related to adenotonsillar hypertrophy, but other factors such as chronic nasal obstruction due to environmental allergies, craniofacial abnormalities, and, increasingly, obesity also play an important role. Both gas-exchange abnormalities and multiple arousals resulting from obstructive events contribute to significant metabolic, cardiovascular, and neurocognitive/neurobehavioral morbidity.

**Definition of Terms**

- **Apnea**: discrete pauses in breathing with complete or almost complete (≥90% drop) cessation of airflow at the nose and mouth, preferably as measured by an oronasal thermal sensor, with a duration ≥10 seconds (or in children at least 2 baseline breaths).

- **Obstructive apnea**: ≥90% cessation of airflow accompanied by continued or increased respiratory effort and evidence of increased work of breathing (e.g., paradoxical movement of the chest and abdominal muscles). Figure 15.1 depicts an obstructive apneic event with a consequent decrease in oxygen saturation. The obstructive apnea index (OAI) is the mean number of obstructive apneas per hour.

- **Central apnea**: cessation of airflow with no respiratory effort for at least 20 seconds or for at least 2 baseline breaths and associated with an arousal/awakening or ≥3% oxygen desaturation. In most healthy typically developing children, this latter type of event typically occurs during rapid eye movement (REM) sleep, at the transition from wake to sleep or from one sleep stage to another, or following a body movement or sigh that is associated with a brief increase in respiratory rate, and thus the subsequent respiratory pause represents a
normal physiologic response to a mild decrease in CO\(_2\); these events are not considered pathologic unless of longer duration or increased frequency. Figure 15.1 depicts a central apneic event with a consequent decrease in oxygen saturation. The central apnea index (CAI) is the mean number of central apneas per hour.

- **Mixed apnea**: an apnea episode lasting for at least 2 baseline breaths and with both absent respiratory effort in one portion and the presence of inspiratory effort in another portion of the event.

- **Hypopnea**: ≥30% reduction in airflow for a duration of 10 seconds or at least 2 breaths, preferably as measured by a nasal pressure sensor, and associated with an arousal or awakening or ≥3% oxygen desaturation.

- **Obstructive hypopnea**: ≥30% reduction of airflow with continued respiratory effort accompanied by snoring and/or increased inspiratory flattening of the nasal pressure and/or paradoxical breathing. Figure 15.1 depicts an obstructive hypopneic event with a consequent decrease in oxygen saturation. The obstructive apnea-hypopnea index (OAHI) is the mean number of obstructive apneas and hypopneas per hour.

- **Apnea-hypopnea index (AHI)**: typically includes both obstructive and central events unless specified otherwise. Thus, the OAHI may be a more accurate reflection of obstructive SRBD if there are multiple central events.

- **Obstructive sleep related hypoventilation**: >25% of total sleep time spent with PaCO\(_2\) >50 mm Hg as measured by end-tidal or transcutaneous CO\(_2\) levels. Obstructive hypoventilation in association with snoring, paradoxing, and/or inspiratory nasal pressure flattening on PSG may be used as diagnostic criteria for OSA in children.

- **Arousal**: an abrupt shift in EEG frequency lasting at least 3 seconds, following at least 10 seconds of sleep. The arousal index (AI) is the mean number of arousals per hour.

- **Respiratory effort-related arousal (RERA)**: an event lasting at least 2 baseline breaths which does not meet criteria for an apnea or hypopnea but is characterized by flattening of the nasal pressure waveform, snoring, increased CO\(_2\) or evidence of increased work of breathing and leads to an arousal. Although it is not mandatory to score RERAs, they may be indicative of more subtle SRBD. The respiratory disturbance index (RDI) is defined as the sum of the AHI and RERA index.

- **Periodic breathing**: ≥3 episodes of central apnea lasting >3 seconds separated by ≤20 seconds of normal breathing. Periodic breathing is considered a normal phenomenon in premature infants; it should comprise less than 3% of total sleep time by 3 months of age.

*Definitions involving specific polysomnographic variables are taken from the 2012 American Academy of Sleep Medicine scoring manual.

**CLINICAL PATTERNS OF SLEEP RELATED BREATHING DISORDERS**

It is important to recognize that OSA is at one end of a clinical spectrum of SRBDs that includes the following clinical conditions:

- **Snoring**, the most common symptom of SRBD, is a manifestation of the vibrations of the oropharyngeal soft tissue walls that occur when an individual attempts to breathe against increased upper airway resistance during sleep.
Taking Snoring Seriously

While clearly the hallmark indicator of increased upper airway resistance during sleep and the most common symptom of OSA, snoring in and of itself may be associated with adverse neurobehavioral/neurocognitive outcomes in children. Furthermore, chronic nasal obstruction and mouth breathing in the first 4 years of life may itself lead to impairments in maxilla-mandibular growth, further increasing the risk of SRBD.

![Image of schematic example of obstructive apnea and hypopneas](http://obgynebooks.com)

**FIG 15.1.** (A) Schematic example of an obstructive apnea and two obstructive hypopneas. (B) Schematic example of two central apneas.

- **Primary snoring** is typically defined as snoring without significant associated ventilatory abnormalities, such as apneas or hypopneas or gas-exchange abnormalities, or arousals as documented by nocturnal PSG.
However, habitual snoring indicates the presence of heightened upper airway resistance and there is evidence that even habitual snoring alone, in the absence of any other gas-exchange or sleep architecture abnormalities, may be associated with increased risk for adverse neurodevelopmental outcomes.

- **Obstructive hypoventilation** is characterized by snoring, increased respiratory effort, and hypercapnia, without apneas/hypopneas or respiratory arousals.

- **Upper airway resistance syndrome (UARS)** is characterized by increasing negative inspiratory intrathoracic pressure and can only be definitively diagnosed by monitoring esophageal pressure (as a proxy measure for pleural pressure). Brief, repetitive respiratory arousals terminate the negative pressure swings, which results in sleep fragmentation and often in evidence of daytime sleepiness. Symptoms of UARS include snoring and increased respiratory effort (including paradoxical chest and abdominal movements). The PSG typically demonstrates RERAs without a significant decrease in airflow or with ventilatory abnormalities (hypoxia, hypercapnia). Most pediatric labs do not measure esophageal pressure to detect UARS. This diagnosis should be considered particularly in adolescents with SRBD symptoms, unexplained daytime sleepiness, and a "normal" PSG.

- **OSA** consists of a constellation of nocturnal and diurnal symptoms, risk factors, and ventilatory abnormalities. OSA occurs when the patency of the upper airway is compromised (by functional or anatomic obstruction) or collapses during inspiration in a dynamic and interactive process that involves mechanics of breathing, ventilatory drive, and characteristics of breathing during the sleeping state.

The PSG parameters that are primarily used in evaluating OSA are the obstructive apnea-hypopnea index (OAHI), lowest (nadir) oxygen saturation, and percentage of sleep time with an elevated end-tidal CO$_2$ level. An OAHI of $\geq 1.0$ is often cited as the cutoff value in children for OSA versus primary snoring; however, it should be noted that there is no universal agreement about this criteria, and researchers have used a variety of definitions of pediatric OSA (e.g., an OAHI $\geq 5$ or an OAI $\geq 1$). While pediatric criteria may be used for patients up to 18 years old, at what specific age (e.g., $>13$ years) adult criteria are applied varies across labs and is also somewhat dependent on the discretion of the interpreting physician (i.e., adult criteria may be appropriate for a 100-kg 13-year-old). In older adolescents, the adult cutoff of an OAHI $\geq 5$ is generally used. In addition, the sleep stage distribution of obstructive events (often significantly increased during REM sleep) and relationship to sleeping position (frequently increased in the supine position) should also be noted. For example, if a school-aged child has a less-than-expected amount of REM sleep for age (typically 20%-25%) on the night of the study and does not spend much time sleeping in the supine position, the results may underestimate the severity of OSA.

An oxygen level $<92\%$ is also generally considered abnormal in children, as is $\geq 25\%$ of total sleep time spent with CO$_2$ $>50$ mmHg (obstructive hypoventilation). Other PSG variables that provide supporting evidence for the presence of OSA include an elevated AI for age and the presence of snoring and paradoxical breathing during the sleep study.

From a clinical standpoint, however, PSG parameters should not be considered "absolutes" and should always be interpreted within the context of the presenting complaints, risk factors for SRBD, and identified sequelae such as daytime sleepiness or elevated systemic blood pressure. Finally, the technical quality of the overnight sleep study should be considered (e.g., presence of REM and supine sleep, adequate total sleep time), and in some cases, it may be prudent to consider repeating the sleep study if the clinical index of suspicion remains high in the face of a negative study (a more detailed discussion of interpreting a sleep study report is included in Chapter 4).
Data regarding the prevalence of pediatric SRBD are available largely for primary snoring and OSA. Given the very small number of pediatric sleep labs currently routinely measuring esophageal pressure, the prevalence rates of UARS, for example, are not known. It should also be noted that SRBD prevalence rates may vary according to the diagnostic definition used (e.g., apnea-hypopnea threshold for OSA, “habitual” snoring as occurring “often” versus specified number of times per week) and the methods employed (e.g., parent report of symptoms versus portable ambulatory monitoring versus in-lab PSG-documented).

- **Primary snoring.** Overall, it is estimated that about 11% of children in the United States between ages 1 and 9 years snore. However, the prevalence of parent-reported snoring in the pediatric population varies depending on definition; for example, one study reported that 4.2% of children “always snored”; 10.9% “almost always” snored; 11.7% snored “greater than or equal to 3 times per week”; and 27% snored “sometimes.” Thus the reported prevalence of habitual snoring varies widely, depending on the study and definition used (e.g., loud snoring recognized by parents 3 times or more per week), from 1.5% to 27.6%. It is estimated that 3% to 12% of children have primary snoring, but given that the diagnosis of “primary snoring” is based on the absence of polysomnographically defined abnormalities, large-scale epidemiologic studies are for the most part lacking.

- **OSA.** The preponderance of evidence suggests a prevalence of OSA in the range of 1% to 5%. When defined by parent-reported symptoms, the prevalence of OSA is between 4% and 11%. The prevalence of pediatric OSA as documented by overnight sleep studies utilizing ventilatory monitoring procedures (e.g., in-lab PSG, home studies) is 1% to 4% overall, with a reported range of 0.1% to 13%, but with a prevalence of 2% to 3% using more stringent definition criteria (e.g., an OAHI >5). Most studies report a figure of between 1% and 4%. Prevalence is also affected by the following demographic characteristics.

**ETIOLOGY AND RISK FACTORS**

There are a number of factors that put a child at risk for OSA. In general terms, OSA results from an anatomically or functionally narrowed upper airway; this typically involves some combination of *decreased upper airway patency* (upper airway obstruction such as adenotonsillar hypertrophy or intraluminal fat deposits and/or decreased upper airway diameter), *increased upper airway collapsibility* (reduced pharyngeal muscle tone), and *decreased drive to breathe* in the face of reduced upper airway patency (reduced central ventilatory drive). Upper airway muscle tone is also influenced by sleep state; for example, the activity of the pharyngeal dilator muscles is diminished in REM sleep. Children with OSA also have narrower pharyngeal airways and increased nasal resistance compared with control children. The stability of the upper airway is also linked to arousal threshold; both children and adults with OSA have a blunted perception of upper airway occlusion and children with OSA will arouse at a significantly higher inspiratory resistive load compared to controls, particularly during REM sleep. At the same time, there exist several factors that may make children less susceptible to complete airway closure than adults, primarily a relatively less collapsible upper airway.

**Demographic Factors**

- **Age.** It is important to note that OSA occurs in all ages, including infants. Older studies suggested a relatively higher prevalence between the ages of 2 and 8 years, coinciding with the peak age of lymphoid hyperplasia and adenotonsillar hypertrophy. But, as overweight and obesity have become more prominent risk factors for OSA in the pediatric population, there is less evidence of consistent with age.

- **Gender.** OSA is more common in boys, especially after puberty.

- **Race/Ethnicity.** There are now considerable data suggesting that African American children have a higher risk for OSA, with an approximately 20% increase in AHI, compared to Caucasians, even after controlling for...
Specific etiological and risk factors related to the underlying mechanisms of SRBD include the following:

**Upper Airway Obstruction**

Upper airway obstruction often reflects an interaction between anatomically reduced upper airway size and soft tissue structures encroaching on the upper airway. Underlying obstruction varies in degree and level (i.e., nose, naso/oropharynx, hypopharynx) and may be due to:

- **Adenotonsillar hypertrophy substantially** raises pharyngeal resistance and ultimately results in episodic airway collapse and complex interactions between the anatomical features and other elements such as upper airway tone and ventilatory drive. Tonsillar size does not necessarily correlate with degree of obstruction, especially in older children. The adenoids, located on the roof of the nasopharynx, normally enlarge in size during infancy and childhood and subsequently progressively reduce in size during adolescence and adulthood. In normal children, the airway size grows proportionately with the soft tissues surrounding it. Disproportionate proliferation of the adenoids and tonsils occurs in some groups of children, including those with allergic rhinitis, asthma, recurrent upper airway respiratory infections, and those exposure to environmental tobacco smoke.

Adenotonsillar Hypertrophy

Adenotonsillar hypertrophy remains the most common cause of OSA in children.

- **Environmental allergies**, associated with chronic rhinitis/nasal obstruction
- **Craniofacial abnormalities**, including hypoplasia/displacement of the maxilla and mandible
- **Gastroesophageal reflux** (due to pharyngeal reactive edema)
- **Nasal septal deviation**
- **Laryngomalacia** may contributing to SRBD; improvements in PSG parameters have been demonstrated after supraglottoplasty as a treatment of clinically significant laryngomalacia.
- **Obesity or being overweight**
- **Velopharyngeal flap cleft palate repair**
- **Chronic nasal obstruction and mouth breathing**, especially in the first 4 years of life, possibly leading to impairments in maxilla-mandibular growth, further increasing the risk of SRBD. Conversely, these skeletal abnormalities may be at least partially reversible with treatment.

**Upper Airway Reduced Muscle Tone**

Upper airway reduced muscle tone (i.e., a “floppy” airway) also has an important role in the underlying pathophysiology of OSA. Factors that may influence patency of the upper airway include the following:

- **Neuromuscular disease**, including hypotonic cerebral palsy and muscular dystrophies
- **Hypothyroidism**, as it may be related to reduced upper airway patency or reduced central ventilatory drive

**Reduced Central Ventilatory Drive**

Reduced central ventilatory drive may be present in some children with OSA; an elevated number and longer duration of central apneas are also very common in these children. Conditions that may be associated with...
reduction in ventilatory drive include the following:

- **Arnold-Chiari malformation**, types I and II
- **Myelomeningocele**
- **Brainstem injury or masses**

### TABLE 15.1. Medical Conditions Associated with Obstructive Sleep Apnea in Children

#### Craniofacial Syndromes
- Apert syndrome
- Crouzon syndrome
- Pfeiffer syndrome
- Pierre Robin syndrome
- Treacher Collins syndrome

#### Neurological Disorders
- Arnold-Chiari malformation (I and II)
- Cerebral palsy
- Meningomyelocele
- Mobius syndrome
- Myasthenia gravis

#### Miscellaneous Disorders/Syndromes
- Achondroplasia
- Beckwith-Wiedemann syndrome
- Choanal stenosis
- Down syndrome
- Goldenhar syndrome
- Hallerman-Streiff syndrome
- Hypothyroidism
- Klippel-Feil syndrome
- Laryngo- and tracheomalacia
- Marfan syndrome
- Mucopolysaccharidosis
- Obesity
- Prader-Willi syndrome
- Sickle cell disease
- Subglottic stenosis

### Medical Conditions

A number of specific medical conditions significantly increase the risk for OSA in children. In many of these disorders, a combination of risk factors is present. For example, children with Down syndrome often have multiple risk factors for OSA, including hypotonia, glossoptosis (posterior tongue displacement), obesity, midface hypoplasia, and hypothyroidism. **Table 15.1** lists the most common medical conditions in children that are
associated with increased risk for OSA.

**Prematurity**
Premature birth increases future risks for SRBD including snoring and OSA. Children born preterm are twice as likely to habitually snore compared to those born full term and are 3 to 5 times more likely to have OSA. A history of prematurity may also increase the risk of neurocognitive deficits.

**Environment**
Passive exposure to cigarette smoking has been associated with both snoring and OSA in children, including infants as young as 8 months old. Studies suggest that environmental tobacco exposure in infants may be associated with snoring-related sleep fragmentation, which in turn may be negatively correlated with developmental measures. In the Childhood Adenotonsillectomy Trial (CHAT) study, environmental tobacco smoke exposure was associated with an approximately 20% increase in AHI in children with OSA.

**Obesity**
Although many children with OSA are of normal weight, multiple studies (although not all) have found a significant relationship between weight and SRBD, including habitual snoring, OSA, and central apneas. In one large study, the risk of OSA among obese children was increased four- to five-fold. Another study concluded that for each increase of 1 kg/m² of BMI above the mean in children, the risk of OSA increased by 12%. Mechanical factors that account for this relationship include reduced diameter and increased collapsibility of the upper airway related to an increase in the amount of fatty infiltration in the throat (pharyngeal fat pads), neck (increased neck circumference), and chest wall and abdomen, creating increased upper airway resistance, worsening gas exchange, and increased work of breathing, particularly in the supine position and during REM sleep. Thus, obese children may develop OSA in conjunction with relatively lesser increases in adenotonsillar volume compared to nonobese children. Obesity reduces the intrathoracic volume and restricts diaphragmatic descent during inspiration, particularly in the supine position, resulting in lower oxygen reserves and increased work of breathing during sleep. There may be a component of blunted central ventilatory drive in response to hypoxia/hypercapnia and hypoventilation as well, particularly in children with morbid or syndrome-based (e.g., Prader-Willi) obesity. Furthermore, it is observed that the distribution of body fat may be more important in predicting SRBD than BMI alone; thus, is recommended that clinicians consider patterns of fat distribution (e.g., waist circumference) and not just BMI in their assessment of the risk for SRBD.

Overweight and obese children and adolescents are also at a particularly high risk for metabolic consequences of SRBD such as insulin resistance, dyslipidemia and the metabolic syndrome, and for cardiovascular complications such as systemic hypertension. Given the evidence that obesity itself is a risk factor for a variety of negative outcomes such as mood disturbances and social problems, even after controlling for OSA, these children may also have a heightened risk for neurobehavioral dysfunction. Finally, obese children are not only at increased risk for postoperative complications following adenotonsillectomy (AT) but are also more likely to have residual disease after surgery.

**The Obesity Epidemic and Obstructive Sleep Apnea**
As the prevalence of childhood obesity increases, weight has become more of a relative risk factor for pediatric OSA. Both overweight and obese children and adolescents are at a particularly high risk for metabolic and cardiovascular complications of OSA, such as insulin resistance and systemic hypertension. Practitioners should systematically screen for SRBD symptoms in children who are overweight or obese according to CDC guidelines for age and gender BMI percentiles.
Genetics
A positive family history of OSA or disruptive snoring is found in a significant percentage of children with OSA symptoms.

Underlying Pathophysiology
Recent studies have yielded important new information regarding the underlying pathophysiology of OSA in children, including mechanisms through which OSA may increase cardiovascular, metabolic, and neurocognitive morbidity. These include (but are not limited to) systemic inflammation, alterations in autonomic regulation, vascular endothelial changes, and metabolic dysregulation. While these physiologic changes are extremely complex and highly interrelated, it is useful for the clinician to have some basic understanding of mechanisms that may lead to important clinical consequences.

There is mounting evidence that OSA is associated with a wide variety of inflammatory responses in the body. For example, adenotonsillar tissue harvested from children with OSA in comparison with adenotonsillar tissue from children with recurrent tonsillitis has revealed significant increases in inflammatory cell proliferation, and increased expression of proinflammatory cytokines and other inflammatory mediators, such as tumor necrosis factor TNF-α and interleukins IL-6 and IL-1α. In particular, OSA in children has been reported in a number of studies to be associated with increased C-reactive protein levels, independent of obesity; these studies also suggest that C-reactive protein may increase with increasing severity of OSA and that CRP levels may decrease following AT. OSA in children is also associated with a host of other inflammatory changes that have been shown to impact vascular inflammation and to contribute to the response to vascular injury. These inflammatory changes affect vascular endothelium and stimulate atheromatous plaque formation via mechanisms that are similar to those involved in atherogenesis. These inflammatory alterations may be further exacerbated by comorbid obesity.

Sleep disruption and gas-exchange abnormalities also presumably result in alterations in vasomotor tone, vascular remodeling, and eventually cardiovascular morbidity. In addition, upper airway obstruction appears to lead to changes in intrathoracic pressure, which when combined with recurrent arousals results in alterations in autonomic nervous system function. Preliminary studies in children with OSA suggesting increased sympathetic activity include the presence of OSA severity-dependent increases in urinary catecholamines, particularly norepinephrine. Disruption of the endothelium, resulting in endothelial dysfunction, may result from alterations in the autonomic nervous system and vasomotor tone, in combination with systemic inflammatory processes and atherogenesis associated with OSA as described earlier. Moreover, significant improvements in endothelial function have been shown 6 months after AT.

Finally, there are a number of mechanisms postulated to result in metabolic derangements in children with OSA. For example, the adipokine leptin, which plays an important role in the regulation of appetite, sleep, metabolic homeostasis, and respiration, is modified by OSA. Leptin also stimulates production of proinflammatory cytokines. The autonomic nervous system is also an important regulator of metabolic function in the liver, adipose tissue, muscle, and pancreas, and alterations in autonomic function further contribute to metabolic dysfunction and obesity. Importantly, even low levels of SRBD (e.g., primary snoring) may be associated with some of these changes in metabolic regulation.

Update on Causes and Consequences of OSA
New evidence published in the last few years has expanded our understanding of the physiologic
impact of OSA on multiple systems in the body and supports the far-reaching consequences of OSA on health. Systemic inflammatory changes and alterations in the autonomic nervous system linked to OSA result in damage to the vascular endothelium, which may result in increased risk of atheromatous plaque formation; in combination with OSA-induced elevations in blood pressure and ventricular dysfunction, the risk of development of cardiovascular disease is increased. Profound metabolic changes associated with OSA, including alterations in glucose metabolism and neurohormones such as leptin, contribute to an increased risk of type 2 diabetes. Inflammatory changes in brain vasculature associated with intermittent hypoxia contribute to neurocognitive and neurobehavioral sequelae. Many of these changes are exacerbated by comorbid obesity. They have also been shown to be reversible with treatment, underscoring the importance of early detection of OSA.

CONSEQUENCES AND SEQUELAE

There are a number of sequelae of OSA and SRBDs, including cardiovascular, metabolic, and neurocognitive consequences.

Cardiovascular Sequelae

Two of the most serious potential cardiovascular consequences related to OSA are systemic hypertension and right and left ventricular hypertrophy. A number of studies have documented dose-dependent relationship of AHI with increased blood pressure in children, and several studies have shown significant improvement in both systolic and diastolic ambulatory blood pressure after treatment of OSA with AT. Furthermore, recurrent hypoxic and hypercapnic episodes of OSA increase pulmonary vascular resistance, leading to pulmonary hypertension and right ventricular dysfunction. A decline in left ventricular diastolic function, an independent risk factor for future cardiovascular disease, has also been shown to occur in children with OSA, and to improve with treatment.

Metabolic Sequelae

A substantial number of studies have suggested that there are important associations between SRBD in children and insulin resistance, alterations in lipid homeostasis and metabolic syndrome (a condition characterized by co-occurrence of abdominal/central obesity, elevated blood pressure, elevated fasting plasma glucose, high serum triglycerides, and low high-density cholesterol levels and a known risk factor for cardiovascular disease in adults). The role that OSA plays is still not entirely clear, but it is likely more important in older (puberty also appears to play an important role) and obese children. Comorbid obesity in OSA increases the risk for lipid disturbances and insulin resistance. For example, adolescents with OSA were found to have a six-fold increase in the odds of developing the metabolic syndrome compared to those without OSA. Obese children with OSA are at increased risk for nonalcoholic hepatic steatosis (fatty liver disease), and treatment of OSA is associated with significant improvements in liver enzyme levels in the majority of these patients.

Neurocognitive Sequelae

To date, over 60 published studies have assessed the relationship between neurocognitive dysfunction and OSA in children and the vast majority have reported significant deficits. Although yet to be fully elucidated, one of the primary mechanisms by which OSA is believed to exert negative influences on cognitive function appears to involve repeated episodic arousals from sleep, leading to sleep fragmentation and resulting sleepiness. In addition, a number of more recent studies have posited at least an equally, if not, more important role for intermittent hypoxia leading directly to systemic inflammatory vascular changes in the brain.
Increased Healthcare Use

Children with OSA are high consumers of healthcare resources. The total number of hospital visits are 40% higher in children with OSA, often related to lower respiratory airway diseases, and they are more likely to require repeated hospital visits. OSA in children is also associated with increased referrals to otolaryngologists and pediatric pulmonologists, as well as increased prescription drug use (primarily antibiotics and respiratory medications). Healthcare use and morbidity are increased several years prior to diagnosis.

PRESENTATION AND SYMPTOMS

The most common presenting complaints in childhood OSA are those associated with nocturnal symptoms—frequent loud snoring, observed breathing pauses, restless sleep, and chronic mouth breathing with nasal obstruction. OSA is much less likely in the absence of at least occasional snoring, although obviously many children who snore do not have OSA. It should be noted that parents may not recognize, and thus may not spontaneously volunteer, information about OSA symptoms, such as snoring, and the history may only be elicited on direct questioning (a screening questionnaire for OSA is provided in Appendix B4). Behavior and academic problems, and concerns about inattention, hyperactivity, impulsivity, and irritability may also be the primary presenting complaints in children with OSA. Because parents do not necessarily associate these problems with OSA, a high index of suspicion should be maintained by the clinician, and children presenting with behavioral, mood, attentional, or academic concerns should be systematically screened for symptoms of and risk factors for OSA.

Common symptoms of pediatric OSA include the following:

Symptoms of Obstructive Sleep Apnea Vary by Age

The presenting symptoms of OSA can vary by age. For example, OSA has been increasingly recognized as also occurring in infants, especially those with a history of prematurity. For these children, symptoms may include “noisy” breathing, nocturnal sweating, disturbed/restless sleep, poor suck, and poor growth. OSA presenting during adolescence tends to more closely resemble “adult” OSA in terms of risk factors (e.g., obesity), as well as clinical presentation (e.g., snoring, apnea, hypersomnolence).

Nocturnal Symptoms

- **Loud, continuous nightly snoring**, although volume does not necessarily correlate with the degree of obstruction. Not every child with OSA has a history of snoring; however, these children will have other symptoms as described below and risk factors that should raise clinical suspicion.

- **Apneic pauses**, although these occur less frequently in children compared to adults. Parents commonly describe episodic choking, gasping, and snorting during the night. One of the best predictors of OSA severity is parental anxiety about sleep respirations. However, it should be noted that, because symptoms of OSA may be worse during REM sleep, which is concentrated in the last third of the night, parents may not be awake to observe the most severe symptoms. Parents of older children and adolescents may also be less likely to observe and note snoring and disturbed sleep.

- **Paradoxical movement** of chest wall and abdomen during breathing; parents may also note that their child is “working hard to breathe.”

http://obgynebooks.com
- Restless sleep, thrashing, and increased body movement
- Sweating during sleep, related to increased work of breathing
- Abnormal sleeping position, such as propped on pillows or sleeping with the neck hyperextended in order to maintain a patent airway
- Mouth breathing, while asleep

Daytime symptoms (physical)
- Mouth breathing and dry mouth
- Chronic nasal congestion/rhinorrhea
- Hyponasal speech
- Morning headaches that may be related to CO\textsubscript{2} retention
- Frequent infections, especially otitis media and sinusitis
- Difficulty swallowing, related to tonsillar hypertrophy
- Poor appetite, which may be related to dysphagia and chronic nasal obstruction

Daytime symptoms (cognitive and behavioral)
- Excessive daytime sleepiness, which may be manifested as more “classic” symptoms of hypersonomolence (e.g., difficulty waking in the morning, falling asleep in school or at inappropriate times, or increased napping in younger children) or with more subtle neurobehavioral signs. Evidence of frank hypersonomolence tends to be less common in children (estimated to occur in 7%-10%) compared to adults with OSA.
- Mood changes, such as irritability, and, in particular, mood instability and emotional dysregulation; low frustration tolerance, depression/anxiety, and social withdrawal
- “Internalizing” behaviors, including increased somatic complaints and social withdrawal
- “Externalizing” behaviors, including aggression, impulsivity, hyperactivity, oppositional behavior, and conduct problems. Hyperactivity, particularly in younger children, is one of the most consistently reported symptoms in a number of sleep disorders, including OSA, and may be a behavioral reaction to an internal sense of sleepiness compounded by behavioral dysregulation.
- “AD/HD”-like symptoms, such as inattentiveness, poor concentration, and distractibility. There is a substantial overlap between the clinical impairments associated with OSA and the diagnostic criteria for AD/HD. There also appears to be a selective impact of OSA, specifically on “executive functions” (a hallmark of AD/HD), which include cognitive flexibility, task initiation, self-monitoring, planning, organization, and self-regulation of affect and arousal. However, it should be noted that results of the CHAT study did not find baseline differences in measures of attention and executive function in school-aged children with moderate to severe OSA.
- Learning problems, especially for tasks involving reaction time, vigilance, and sustained and selective attention. Other cognitive deficits, such as language delays and impairments in visual perception, motor functions, and memory, have been less consistently found. Cognitive deficits have also been identified in very young children and those with very mild SRBD. For example, infants with persistent habitual snoring have been found to have lower scores on a standardized measure of infant/toddler development (Bayley scores). Other factors that appear to increase the risk of cognitive dysfunction in children with OSA include premature birth, lower socioeconomic status, asthma, obesity, short sleep duration, and African
American race.

**Academic problems.** An association between low levels of academic achievement and SRBD likely represents the combined influence of a number of these learning and attention-based impairments.

**Playing Detective**

SRBD in children may present primarily with parental complaints of behavior problems, inattentiveness ("ADHD"), and academic failure. Parents may not volunteer information about sleep and symptoms of SRBD (snoring) unless these are directly elicited by the primary care provider. Because treatment of OSA can substantially mitigate these effects, it is key to identify these children early.

**Diagnostic Criteria**

See Table 15.2.

**Associated Features**

- **Enuresis (especially secondary),** postulated to be due to alterations in atrial natriuretic peptide as a consequence of changes in intrathoracic pressure with obstructive events, is present in about one-fourth of children. Enuresis resolves in about half within 1 month after AT.
- **Growth failure (in severe cases, failure to thrive),** which may be related to a combination of decreased appetite, decreased intake, increased metabolic needs from increased work of breathing, and alterations in normal nocturnal growth hormone secretion patterns
- **Increase in partial arousal parasomnias** (e.g., sleepwalking, sleep terrors) in susceptible children, which are related to sleep fragmentation and compensatory increases in slow-wave sleep.
- **Increase in seizure frequency in predisposed children,** which is possibly related to increased arousals and intermittent hypoxia.
- **Other comorbid sleep problems,** such as bedtime resistance and nightwakings, restless legs syndrome/periodic limb movement disorder, and circadian rhythm disorders are common in children with primary OSA. These sleep problems may be secondary to the OSA (sleep fragmentation related to arousals resulting in more nightwakings) or exist independently. These comorbid sleep problems are important to identify as they may add significantly to the adverse consequences of OSA; alternatively, failure to address these sleep issues may compromise treatment success.

**TABLE 15.2. Diagnostic Criteria: Obstructive Sleep Apnea, Pediatric**

Criteria A and B must be met

A. The presence of one or more of the following:
   1. Snoring
   2. Labored, paradoxical, or obstructed breathing during the child's sleep
   3. Sleepiness, hyperactivity, behavioral problems or learning problems

B. PSG demonstrates one or both of the following:
   4. One or more obstructive apneas, mixed apneas or hypopneas, per hour of sleep†
   5. A pattern of obstructive hypoventilation, defined as at least 25% of total sleep time with hypercapnia (PaCO₂ >50 mmHg) in association with one or more of the following:
a. Snoring  
b. Flattening of the inspiratory nasal pressure waveform  
c. Paradoxical thoracoabdominal motion

*ICD-9-CM code: 327.23; ICD-10-CM code: G47.33

†Respiratory events defined according to the most recent version of the AASM Manual for the Scoring of Sleep and Associated Events

Note: UARS is subsumed under this diagnosis.


EVALUATION

Evaluation for OSA includes history of signs and symptoms, physical examination, and, in most cases, overnight PSG.

Clinical Guidelines for the Evaluation of OSA in Children

There now exist a number of sets of clinical guidelines regarding the screening and diagnosis of OSA in the pediatric population. These have been developed over the past decade by professional organizations for a variety of different practitioner audiences, and thus they differ somewhat in both the specifics of their recommendations and the strength of the supporting evidence cited. The American Academy of Pediatrics (AAP) revised its guidelines in 2012, and these form the basis of the recommendations in this chapter unless otherwise indicated. The American Academy of Sleep Medicine (AASM) included guidelines for the polysomnographic diagnosis of pediatric OSA as a set of practice parameters for respiratory indications for PSG in children in 2011. The American Academy of Otolaryngology-Head and Neck Surgery also published recommendations for evaluation of SRBD prior to AT in 2011; these are all briefly summarized below for comparison (the AASM 2012 Practice Parameters on nonrespiratory indications for PSG in children are discussed in Chapter 4).


1. As part of routine health maintenance, the clinician should inquire if the child snores. If yes, or if the child presents with signs/symptoms of OSA, the clinician should perform a more focused examination.

2. If the child snores on a regular basis (≥3 times/week) and has signs/symptoms of OSA, the clinician should either (1) obtain PSG or (2) refer the patient to a sleep specialist or otolaryngologist for more extensive evaluation.

3. OSA overnight, attended PSG remains the gold standard of care. It should be performed using specific pediatric measuring and scoring criteria.

4. If PSG is not available, the clinician may order alternative diagnostic tests, such as nocturnal video recording, nocturnal pulse oximetry, daytime nap PSG, or ambulatory PSG.

*AAP evidence-based guidelines focus specifically on uncomplicated patients with OSA associated with
either adenotonsillar hypertrophy and/or obesity in an otherwise healthy child who is being treated in a primary care setting. This guideline specifically excludes infants younger than 1 year; patients with central apnea or hypoventilation syndrome; and patients with OSA associated with other medical disorders such as craniofacial abnormalities, mucopolysaccharidosis, sickle cell anemia, neuromuscular disorders, and Down syndrome.

AASM Practice Parameters for the Respiratory Indications for Polysomnography in Children (2011)*

1. Children with mild OSA preoperatively should have clinical evaluation following AT to assess for residual symptoms. If there are residual symptoms of OSA, PSG should be performed. (Standard)
2. PSG is indicated following AT to assess for residual OSA in children with preoperative evidence for moderate to severe OSA, obesity, craniofacial anomalies that obstruct the upper airway, and neurologic disorders (e.g., Down syndrome, Prader-Willi syndrome, and myelomeningocele). (Standard)
3. PSG is indicated for positive airway pressure (PAP) titration in children with OSA syndrome. (Standard)
4. Follow-up PSG in children on chronic PAP support is indicated to determine whether pressure requirements have changed as a result of the child’s growth and development, if symptoms recur while on PAP, or if additional or alternate treatment is instituted. (Guideline)
5. PSG is indicated after treatment of children for OSA with rapid maxillary expansion to assess for the level of residual disease and to determine whether additional treatment is necessary. (Option)
6. Children with OSA treated with an oral appliance should have clinical follow-up and PSG to assess response to treatment. (Option)

*Each recommendation is followed by an assessment of the strength of supporting evidence (in descending order, Standard-Guideline-Option)


1. Before determining the need for tonsillectomy, the clinician should refer children with sleep-disordered breathing for PSG if they exhibit certain complex medical conditions such as obesity, Down syndrome, craniofacial abnormalities, neuromuscular disorders, sickle cell disease, or mucopolysaccharidosis.
2. The clinician should advocate for PSG prior to tonsillectomy for sleep-disordered breathing in children without any of the comorbidities listed in statement 1 for whom the need for surgery is uncertain or when there is discordance between tonsillar size on physical examination and the reported severity of sleep-disordered breathing.
3. In children for whom PSG is indicated to assess sleep-disordered breathing prior to tonsillectomy, clinicians should obtain laboratory-based PSG, when available.

*Italics added

Although no constellation of symptoms and physical findings has been found that reliably distinguishes OSA from primary snoring, a thorough history and physical examination is mandatory in the evaluation of SRBD in children.

- **Medical history:** Both medical risk factors for and medical sequelae of OSA may be present. The history is
often positive for both upper airway (chronic sinusitis) and lower airway (asthma) disease, allergies, and frequent upper respiratory infections. There may be a history of frequent episodes of streptococcal pharyngitis/tonsillitis. Symptoms suggestive of gastroesophageal reflux (heartburn, vomiting, avoidance of spicy foods) should be elicited.

- **Developmental/school history:** Although the developmental/school history may be normal, there is frequently a history of significant academic concerns, as well as attentional and learning problems. Certain syndromes with developmental delay as a prominent feature (e.g., Down syndrome, Prader-Willi syndrome) are associated with increased risk for OSA.

- **Family history:** A history of chronic loud snoring, as well as a diagnosis of OSA is often reported in family members.

- **Behavioral assessment:** Evaluation of behavioral and mood concerns is key in assessing the extent of daytime sleepiness-related sequelae.

- **Physical examination:** In many cases, the physical examination of children with SRBD is completely normal. However, the primary care clinician should assess for the following:
  - **Growth:** including overweight and obesity, as well as failure to thrive (especially in younger children).
  - **HEENT (head, eyes, ears, nose, throat):**
    - **Facial structure:** There are a number of craniofacial morphologic features in normal children (i.e., without craniofacial syndromes) that are consistent with pediatric OSA, including retrusive chin, steep mandibular plane, vertical direction of growth, and a tendency toward class II malocclusion. Habitual mouth breathing children lower their mandible, resulting in a high-arched palate, narrow maxilla, retrognathia, and increased lower facial height—so-called “adenoidal facies.” These children may also have midface hypoplasia. Retro- and micrognathia are best appreciated by inspection of the lateral facial profile.
    - **Signs of atopy:** including “allergic shiners,” nasal crease (“allergic salute”), and eczema.
    - **Nasal passage patency:** including asymmetry of the nares and septal deviation, mucosal thickening, edematous turbinates, and the presence of nasal polyps.
    - **Hyponasality:** ask child to repeat “Mickey Mouse,” “ninety-nine,” or “my name is money” while occluding the nose; a child with significant nasal obstruction will sound the same with occlusion as without.
    - **Mouth breathing:** may indicate enlarged adenoids and/or chronic congestion.
    - **Oropharyngeal examination:** including tongue size, palatal integrity, presence of a high-arched, narrow, elongated, or low-dependent palate, uvular size, position, and shape (e.g., bifid which may be associated with submucosal cleft palate), tonsillar size, and posterior pharyngeal space (Figure 15.2). The Mallampati score (Figure 15.3) was developed for adults to assess oropharyngeal patency and is also useful in assessing children with SRBD.
  - **Neck examination:** assess for thyromegaly, as hypothyroidism appears to be a risk factor for OSA, at least in adults. Neck circumference is one of the best predictors of OSA in adults, although similar studies have not been conducted in prepubertal children.
  - **Cardiac examination:** including systemic hypertension, signs of pulmonary hypertension (e.g., loud second pulmonary heart sound), and, in severe cases, cor pulmonale. Fortunately, these signs of severe OSA are now rarely seen.
- **Chest examination**: including the presence of expiratory wheeze, which may indicate lower airway disease as a contributing factor to SRBD.

**FIG 15.2.** Grading tonsillar size.
Diagnostic tests

For the most part, laboratory studies are unnecessary in OSA, although in severe cases evidence of polycythemia on complete blood count and/or compensatory metabolic alkalosis associated with chronic hypoventilation may be noted.

Radiologic/Imaging and Other Studies

Radiologic/imaging and other studies may be helpful in elucidating the level of obstruction.

- **Upright lateral neck radiograph** to evaluate for hypertrophy of the tonsils/adenoids, regrowth of adenoids, and the presence of lingual tonsils is frequently quite helpful.

- **Chest radiograph** may demonstrate evidence of right ventricular hypertrophy in cases of severe OSA; the electrocardiogram may also show evidence of right ventricular enlargement.

- **Other imaging tools** such as cephalometric radiographs, computerized tomography, and magnetic resonance imaging may be helpful in assessing the upper airway structure, especially in children with craniofacial anomalies. Cine MRI provides a high-resolution examination of the dynamic airway and more accurately evaluates the upper airway of obstruction in children.

- **Drug-induced sleep endoscopy (DISE)** is a relatively new and effective diagnostic tool for identifying sites of obstruction under conditions which approximate those occurring during normal sleep. It may be particularly helpful in evaluating the contribution of lingual tonsillar hypertrophy and occult laryngomalacia.

- **Flexible endoscopy** examining the nasopharynx, posterior oropharynx, the base of the tongue, lingual tonsils, and the larynx can be performed by an otolaryngologist in the office setting or in the operating room under light sedation, and is helpful in delineating the specific site(s) of obstruction. Observation of edema of the posterior oropharyngeal wall and/or granular pharyngitis may suggest gastroesophageal reflux as a contributing factor to a decrease in the airway size.

Specific Diagnostic Tools for SRBD in Children

Although the history and physical examination are important in making the diagnosis of OSA in children,
overnight attended PSG performed in an accredited sleep laboratory by technicians skilled in working with children and interpreted by a sleep medicine physician with pediatric experience remains the diagnostic gold standard for OSA in children. This is based in large part on the fact that no combination of symptoms and physical findings has been identified that reliably distinguishes OSA from primary snoring. That is, snoring and other SRBD symptoms may occur in the absence of documented respiratory abnormalities. Furthermore, the reliability of a negative history of SRBD symptoms such as snoring is poor, especially in older children and adolescents who may not be observed during sleep by adult caregivers. In addition, children are more likely to have partial rather than the more obvious complete obstruction of the airway. Finally, airway obstruction tends to be worse during REM sleep, which is concentrated in the latter third of the night when caregivers are most likely to be asleep.

The 2012 AAP recommendations, recognizing that PSG may not be readily available in all practice settings, list alternatives to in-lab overnight PSG, specifically nocturnal video recording, nocturnal pulse oximetry, daytime nap PSG, and ambulatory PSG. However, while other diagnostic assessments for OSA in children have been found to have acceptable positive predictive value, in general they have a poor negative predictive value. For example, while audiotape may be a useful screening tool, the positive predictive value “is probably too low to rely on it alone to base management decisions on positive results.” Similarly, although positive nap studies fairly reliably indicate the presence of OSA, negative studies are inconclusive and would need to be repeated. The use of ambulatory PSG in children remains controversial. A large international study concluded that in-home unattended PSG conducted under research conditions in a nonclinical sample of healthy typically developing school-aged children is “feasible, technically adequate, and well-tolerated”; however, the strict inclusion criteria of this study limits the potential generalizability to other pediatric populations. Moreover, the cost-effectiveness of this diagnostic tool, given the potential need for repeated tests due to negative results, has not been well-studied.

A recent (2013) study conducted a systematic review of available diagnostic tools for OSA in children, focusing on the correlation with PSG. In this review, many widely used tests for OSA (e.g., questionnaires, oximetry) failed to demonstrate sufficient diagnostic accuracy (sensitivity ranging from 78% to 100%, and the specificity from 0% to 62%), although the authors concluded that selected parent-report questionnaires such as the Pediatric Sleep Questionnaire (see Appendix B4) may be effective screening tools to identify children at high or low risk for OSA. Finally, no other diagnostic test reliably assesses severity of SRBD, which may impact perioperative management and is a key parameter in determining which patient’s OSA is not likely to completely resolve with AT alone.

Polysomnography: The Gold Standard

At the present time, given that the only way to make a definitive diagnosis and assess the severity of OSA is with overnight in-lab PSG, a sleep study should ideally be performed in every child with significant nocturnal and diurnal symptoms and risk factors for OSA (see Chapter 4 for more information on overnight PSG). PSG is also important as a baseline measure for children with additional risk factors in whom OSA is not likely to completely resolve with AT alone (e.g., obesity, craniofacial anomalies), and who therefore may need a follow-up postoperative sleep study.

Differential Diagnosis

- **Upper airway obstruction without sleep related ventilatory abnormalities** may be related to tracheolaryngomalacia, vascular rings, or gastroesophageal reflux.
- **Nocturnal respiratory disturbances** accompanied by ventilatory abnormalities (e.g., hypoxemia, hypercarbia) during sleep can be due to central hypoventilation syndromes or asthma.

- **Sleep related movements** mimicking OSA-related gasping and arousals may occur with nocturnal seizures; periodic limb movements during sleep may occur in association with apneas/hypopneas.

- **Excessive daytime sleepiness and neurocognitive deficits** can result from many other sleep disorders, including narcolepsy, idiopathic hypersomnia, insufficient sleep syndrome, and restless legs syndrome/periodic limb movement disorder. Daytime sleepiness can also be related to psychiatric disorders, such as depression, medical conditions, and drug therapy.

**MANAGEMENT**

**When to Refer**

When there are straightforward OSA symptoms (e.g., snoring, apneic pauses) with clear-cut risk factors (e.g., adenotonsillar hypertrophy) in an otherwise healthy child, the primary care pediatrician may be comfortable referring for and reviewing the results of the overnight sleep study. However, in cases in which there are additional risk factors (e.g., obesity, congenital syndromes), evidence of severe disease (e.g., growth failure, significant neurobehavioral sequelae), or other complicating factors (e.g., underlying medical conditions), consultation with a sleep center that has pediatric expertise is warranted. In addition, children who are candidates for continuous positive airway pressure (CPAP) treatment should be referred to a pediatric sleep center.

**Treatment**

There are presently no universally accepted guidelines regarding the indications for treatment and follow-up of pediatric SRBD (including primary snoring, UARS, and OSA), and current recommendations largely emphasize weighing what is known about the potential cardiovascular, metabolic, and neurocognitive sequelae of SRBD in children in combination with the individual healthcare professional's clinical judgment. The decision of whether and how to treat OSA specifically in children is contingent on a number of parameters, including severity (nocturnal symptoms, daytime sequelae, sleep study results), duration of disease, and individual patient variables such as age, comorbid conditions, and underlying etiologic factors.

In the case of moderate to severe disease (AHI >10), the decision to treat is usually straightforward, and most pediatric sleep experts recommend that any child with an apnea index >5 should be treated. However, it should be noted that these efforts to match treatment recommendations with OSA severity as defined by threshold levels of various polysomnographic variables have not been universally embraced, and a number of sleep researchers and clinicians have argued that they fail to account for the individual and likely genetically determined susceptibility to the neurocognitive, cardiovascular, and metabolic sequelae of OSA. Indeed, most studies have failed to find a dose-response relationship between polysomnographically derived parameters of OSA disease severity and cognitive and behavioral problems. Furthermore, several recent studies have found neurobehavioral deficits associated with primary snoring in children that are similar to those found in children with OSA. Thus the “threshold” of degree of OSA associated with adverse consequences may vary widely both across individuals and according to the outcome parameters of interest. In the future, a “hybrid” approach to the diagnosis and treatment of OSA in children, combining polysomnographic indices, demographic variables, symptoms, risk factors, and biomarkers will hopefully...
Clinical Guidelines for the Management of OSA in Children


1. If the child has OSA with adenotonsillar hypertrophy, AT is recommended as first-line treatment. If the child has OSA without adenotonsillar hypertrophy, other treatments should be considered.
2. Clinicians should monitor high-risk patients undergoing AT as inpatients postoperatively.
3. Clinicians should clinically reassess all patients with OSA for persisting signs/symptoms after therapy to determine if further therapy is required.
4. Clinicians should reevaluate high-risk patients for persistent OSA after AT, including those who had a significantly abnormal baseline PSG, have sequelae of OSA, are obese or remain symptomatic after treatment, with an objective test or referral to a sleep specialist.
5. Clinicians should refer patients for CPAP management if symptoms/signs or objective evidence of OSA persists after AT or if AT is not performed.
6. Clinicians should recommend weight loss in addition to other therapy if a child with OSA is overweight or obese.
7. Clinicians may prescribe topical intranasal corticosteroids for children with mild OSA in whom AT is contraindicated or for children with mild postoperative OSA (apnea-hypopnea index <5/hour).


1. Clinicians should communicate PSG results to the anesthesiologist prior to the induction of anesthesia for tonsillectomy in a child with sleep-disordered breathing.
2. Clinicians should admit children with OSA documented on PSG for inpatient, overnight monitoring after tonsillectomy if they are younger than age 3 or have severe OSA (apnea-hypopnea index of 10 or more obstructive events/hour, oxygen saturation nadir less than 80%, or both).

### Treatment Options

- **AT** is the most common treatment for pediatric OSA and OSA is the most common indication for tonsillectomy with or without adenoidectomy. Approximately 500,000 to 600,000 such procedures are performed annually in the United States on children younger than 15 years of age. It is the first-line treatment in any child with significant adenotonsillar hypertrophy, even in the presence of additional risk factors such as obesity.

### American Academy of Pediatrics: Contraindications for Adenotonsillectomy (2012)
• Absolute contraindications
  ■ No adenotonsillar tissue

• Relative contraindications
  ■ Very small tonsils/adenoids
  ■ Morbid obesity and small tonsils/adenoids
  ■ Bleeding disorder refractory to treatment
  ■ Submucous cleft palate

Success Rate. The results of published data on the success rate of AT in resolving OSA are highly variable, ranging from 24% to 100%. However, AT in uncomplicated cases results in significant improvement or complete resolution of symptoms in the vast majority (82%). Removal of both the tonsils and adenoids has been shown to be more effective than either alone and in most cases is recommended to avoid recurrence of symptoms, even if one appears to be the primary abnormality. Regrowth of adenoidal tissue, even after surgical removal, may also occur.

Watchful Waiting versus Adenotonsillectomy
The initial results of the CHAT study have raised some important questions about the indications for and efficacy of AT for OSA. The primary outcome measures (attention and executive functions) in the study did not differ significantly at follow-up comparing early AT (eAT) versus watchful waiting. There were greater improvements in subjective (parent and teacher report) measures of behavior, quality of life, OSA symptoms, and polysomnographic findings. Normalization of polysomnographic findings was observed in a larger proportion of children in the eAT group, although less likely in African American and obese children. However, normalization was also found in almost half (46%) of the watchful waiting group after 7 months, suggesting that careful observation without immediate surgical intervention may be warranted in some children.

Risk Factors for Residual OSA. Risk factors for residual OSA include asthma, craniofacial anomalies, Down syndrome, older age (>7 years) at time of surgery, and more severe sleep apnea (especially an AHI ≥20/hour) preoperatively. OSA may improve significantly after surgery even in obese children. However, obesity remains a major risk factor for residual OSA; reported in 54% to 76% of obese children. Note that AT for OSA may result in weight gain, even in those children who are overweight or obese at baseline. The potential mechanisms by which AT results in increased weight gain in children with OSA include increased caloric intake associated with improvements in reduced olfaction and dysphagia related to adenotonsillar hypertrophy, and decreased caloric expenditure owing to decreased work of breathing, resolution of intermittent hypoxemia, and decreased hyperactivity, as well as increased growth hormone secretion. Thus, weight monitoring, nutritional counseling, and encouragement of physical activity are imperative in overweight/obese children with OSA even after surgical intervention.

Complications. The complication rate after AT in children ranges from 0% to 32% and includes hemorrhage (3%-8% of children), respiratory decompensation, transient worsening of OSA secondary to postoperative edema and increased secretions, anesthetic complications, pain, and poor oral intake, with airway compromise representing up to 16% of complications. Return to surgery occurs in 1% to 2% of patients.
Risk Factors for Postoperative Respiratory Complications in Children with OSA Undergoing Adenotonsillectomy

- Younger than 3 years of age
- Severe OSA on PSG (apnea-hypopnea index >10 and/or oxygen saturation nadir <80%, peak PCO$_2$ ≥60 mmHg)
- Cardiac complications of OSA
- Failure to thrive
- Obesity (up to seven-fold increase)
- Craniofacial anomalies
- Neuromuscular disorders
- Current respiratory infection

Postoperative Monitoring. In all patients with OSA, both the surgeon and the anesthesiologist should be provided with information regarding the presence of apnea as well as with the results of the baseline sleep study. Careful postoperative monitoring is recommended for all children with OSA. Some centers have adopted a policy that all children undergoing AT for OSA should be monitored overnight, as the immediate postoperative period may not have adequate REM sleep in which to observe worsening of any ventilatory abnormalities. Children with severe disease or medical complications may require overnight monitoring in an intensive care setting.

Postoperative Reevaluation. Due to postoperative edema, OSA symptoms may take 6 to 8 weeks to completely resolve. All patients should be reevaluated postoperatively to determine whether additional evaluation and/or treatment are required. If there are significant residual risk factors (e.g., obesity) or continued symptoms of OSA, a follow-up sleep study at least 6 weeks post AT may be indicated. The 2012 AAP guidelines stress the importance of postoperative screening since a large proportion of high-risk children continue to have some degree of OSA postoperatively. Reevaluation may include PSG. Specific guidelines regarding postoperative testing are provided in the 2011 AASM pediatric PSG respiratory parameters.

- **Radiofrequency ablation or submucosal resection of enlarged turbinates** in appropriate patients may be helpful because nasal patency is a crucial issue that should be addressed in children with OSA. Hypertrophic inferior nasal turbinates can cause nasal airway obstruction similar to that seen with adenoid hypertrophy. Failure to address nasal obstruction from turbinate hypertrophy at the time of T&A has been found to have a negative impact on outcomes. Surgical treatment for nasal septal deviation may also be appropriate in selected cases.

- **Other surgical procedures**, such as epiglottoplasty, uvulopharyngopalatoplasty (UPPP), and maxillofacial surgery (mandibular distraction osteogenesis and maxillomandibular advancement), are seldom performed in children but may be indicated in selected cases. Palatal surgery, such as UPPP, may be used to treat complicated OSA in obese children, children with cerebral palsy, and children with Down syndrome, as well as those with other neurologic impairments or craniofacial anomalies. Radiofrequency ablation of the tongue and partial midline glossectomy may be appropriate procedures in children for whom the tongue base serves as a major source of obstruction; genioglossal advancement and lingual tonsillectomy are also surgical options that are aimed at improving oropharyngeal and hypopharyngeal obstruction with varying degrees of reported success.

- **Tracheotomy** is rarely indicated, except in the case of severe, life-threatening OSA for which other treatment modalities are contraindicated or have been ineffective.
CPAP or bilevel positive airway pressure (BiPAP) is the most common treatment for OSA in adults. PAP is a noninvasive method of providing distending pressure to maintain a patent upper airway. PAP can be used successfully with children and adolescents, even children as young as a year old. It should be considered a palliative rather than a curative therapy, and may be needed for a prolonged period of time. While the exact threshold for initiating PAP therapy may vary across patients, most pediatric sleep experts would agree that an AHI >10 indicates more severe disease and warrants at least an attempt of PAP therapy if other management strategies have failed. PAP may also be indicated in the following clinical situations:

- AT not indicated or contraindicated.
- AT fails to completely resolve symptoms, usually in children with additional risk factors such as obesity, craniofacial anomalies, or trisomy 21.
- Prior to surgery in children with severe OSA.

Pediatric patients demonstrate significant improvement in both symptoms and objective measures of OSA, including snoring, daytime sleepiness, AHI, and O₂ saturation while using PAP therapy. The minimum daily duration of PAP therapy required to mitigate the adverse effects of OSA in children is unknown. However, because adherence is typically low as measured by such parameters as high drop-out rate and limited hours of nightly use, the AAP has not recommended PAP as a first-line therapy for OSA when AT is an option.

PAP involves the use of nasal or full-face interface that is securely attached to the head. A fullface mask is often used when the patient is a mouth breather to prevent air escape; however, there may be an increased risk of aspiration in children with full-face masks. Interfaces that are approved for use in children as young as 2 years old are now commercially available. It is important to ensure a tight seal without air leaks; a chin strap may be added to prevent air leaks through the mouth. Optimal pressure settings (that abolish or significantly reduce respiratory events without increasing arousals or central apneas) are determined in the sleep lab during a full-night PAP titration. Re-titrations should be conducted periodically with long-term use (every 6 months in young children and at least yearly or with significant weight changes or recurrence of symptoms in older children and adolescents).

Side effects of CPAP use are generally minimal and include nasal congestion, dryness, and rhinorrhea; eye irritation; and facial dermatitis, although concerns about changes in craniofacial structure with long-term use, especially in young children, have been raised. Adding warmed and humidified air and slowly increasing pressure over a 10- to 20-minute period (“ramp time”) may alleviate some of these side effects. Patients may complain of feelings of claustrophobia and difficulty exhaling. In situations in which these problems prevent successful usage, BiPAP (which allows an expiratory pressure to be set at a lower level than inspiratory pressure) may be better tolerated.

Compliance with CPAP is a key factor in determining success. Objective assessment of CPAP adherence is important because parental estimates of use are often inaccurate. Most PAP devices now include a downloadable “chip” which allows such key parameters as average number of usage days, average number of hours of usage, and average AHI on PAP to be stored and then reviewed periodically by the practitioner. PAP adherence in children has been shown to be related primarily to family and demographic factors rather than severity of apnea or measures of psychosocial functioning, although for some children, the dramatic improvement in quality of life and potential improvements in attention and academic functioning observed with PAP use is an important motivating factor for continued use. Adolescents pose a particular challenge in terms of compliance. Factors such as the degree of structure in the home, social reactions, communication among family members, and perception of benefits are important issues that play a role in PAP adherence in the adolescent
population. But most children, even those with significant developmental delays such as trisomy 21, can be successful PAP users. It is extremely helpful to provide families with access to a comprehensive behavioral program utilizing such techniques as positive reinforcement, training parents to manage resistant behavior, modeling, desensitization, and shaping.

- **Weight management**, including nutritional, exercise, and behavioral components, should be strongly encouraged for all children with OSA who are overweight or obese. Children with insufficient sleep are at increased risk for obesity, so interventions targeted toward assuring sufficient sleep duration may be helpful in reducing weight-related risk for OSA. Significantly compromised obese patients may benefit from an inpatient weight loss program; bariatric surgery may be considered in morbidly obese adolescents with severe OSA. Along with many other health-related benefits, achieving weight loss and increasing exercise seem to be beneficial for OSA in overweight and obese children and adolescents. In contrast to adults, however, there is a paucity of data regarding the “dose-dependent” impact of weight loss on OSA in children and adolescents, especially in regard to modest weight loss.

- **Oral appliances** represent one of the newer advances in the treatment of OSA in children. These devices use mechanical forces to enlarge the pharyngeal airway and are of two basic types: those that result in mandibular advancement and those that expand the maxilla/palate. The latter type is a fixed oral appliance that increases the transversal diameter of the hard palate over a 6- to 12-month period and is used in children with maxillary constriction and high-arched palates, as both of these anatomic features are associated with increased nasal resistance. For the most part, the use of these oral appliances can be considered in older children (6-8 years and above) who can cooperate with use of the appliance, have all secondary dentition in place, and have mild-to-moderate residual OSA; consultation with a dentist/orthodontist experienced in fitting oral appliances in children is mandatory. Outcome studies on the use of oral appliances have been conducted mainly in adults, but recent studies in children show promising results in selected patients. In addition, since there are insufficient long-term efficacy data using either mandibular advancement devices or maxillary expansion to treat OSA in children, the 2011 AASM practice parameters recommend follow-up PSG.

- **Pharmacologic interventions** provide an important therapeutic option in the treatment of mild-to-moderate OSA in children. Data regarding the pathophysiologic mechanisms underlying pediatric OSA have led to consideration of anti-inflammatory therapies aiming to reduce adenotonsillar hypertrophy or upper airway inflammation in pediatric OSA. Thus, leukotriene inhibitors may be a therapeutic alternative in children with mild or residual OSA. Administration of leukotriene receptor antagonists, such as montelukast, has been shown to result in substantial improvements in sleep related respiratory disturbance measures, such as the apnea index, as well as reduction in adenoid size in children with mild OSA. In addition, it has been postulated that leukotriene antagonists could be used as a pre- and posttreatment modality in OSA prior to AT in an effort to improve the outcomes associated with the surgery.

Similarly, targeting glucocorticoid expression in upper airway lymphoid tissues with topical intranasal steroids may reduce residual OSA in children post AT. Fluticasone, budesonide, and dexamethasone have all been studied, with the most evidence supporting fluticasone efficacy. However, because the long-term effects of intranasal steroids are not well-studied, follow-up evaluation is recommended to ensure that the OSA does not recur and to monitor for adverse effects. Finally, the potential synergistic effect of combining intranasal steroids and oral montelukast, a common clinical practice, appears promising.

- **Avoidance of sedating agents and environmental triggers.** Sedating agents, medications containing alcohol, or medications with respiratory depressant effects should be avoided as they may exacerbate OSA (see also Chapter 20). Exposure to environmental tobacco smoke and other pollutants should also be avoided.
Positional therapy can be used as an adjunct measure in some patients with mild OSA or primary snoring in whom obstructive events are worse in the supine position. A tennis ball or other firm ball may be sewn into the pocket of a pajama top or T-shirt that is worn backwards, preventing the child from sleeping in the supine position; a fanny pack may also be used.

“Snore aids” such as external nasal dilator strips and nasal sprays have not been shown to have consistent benefit in adults with OSA and have not been studied in children.

Nasal expiratory positive airway pressure (NEPAP) devices as an alternative to PAP therapy have not been extensively studied in children. A recent small pilot study found significant overall improvement in the OAI, but of the 14 subjects, 3 did not improve and 2 worsened. Adherence was reported to be higher than typical for PAP.

Supplemental oxygen therapy is seldom warranted in children with OSA, unless there are special circumstances. In fact, oxygen therapy may worsen hypoventilation. Furthermore, oxygen therapy will not alleviate the sleep fragmentation or hypoventilation associated with OSA.

Myofunctional therapy (MT) is a program of oral facial muscle therapy that uses measurement and exercise to change the habits of abnormal swallowing patterns, build oral facial muscle strength, and train the patient to swallow correctly. The therapy is focused on correcting improper function of the tongue and facial muscles used at rest, for chewing and for swallowing. Therapists are typically speech pathologists, dental hygienists, and other oral health-related professionals. Studies suggest that MT decreases AHI by approximately 50% in adults and 62% in children. MT has promise as an adjunct to other OSA treatments in selected patients.

Prognosis
Studies that have looked at changes in behavior and neuropsychological functioning in children following treatment (usually AT) for OSA have largely documented significant improvement in outcomes, both in the short- and long-term, of OSA syndrome posttreatment, including daytime sleepiness, mood, behavior, academics, and quality of life. However, because many studies have failed to find a dose-dependent relationship between OSA in children and specific neurobehavioral/neurocognitive deficits, this has led to speculation that other factors, including individual genetic susceptibility, environmental influences such as passive smoking exposure, and comorbid conditions, such as obesity, shortened sleep duration, and the presence of other sleep disorders, may also influence neurocognitive outcomes.

Little is known about the persistence of disease in untreated or partially treated children; previous studies had suggested that children with abnormal findings on PSG are likely to continue to have these abnormalities if untreated. However, the recent results of the CHAT study trial, in which almost half of children in the “watchful waiting” control group normalized their PSG findings during the 7 month follow-up, raises some new questions about “spontaneous resolution.” Longitudinal studies also suggest that a significant percentage (40%-50%) of children with habitual snoring will continue to snore at follow-up several years later, while a much smaller percent (4%-5%) will initiate snoring during that period. Furthermore, the long-term neurobehavioral consequences of untreated SRBD have not been well-studied.

Finally, children with OSA initially treated successfully with AT may redevelop symptoms as older children or adolescents. Risk factors for recurrence of OSA after AT include obesity, gain velocity in BMI, and African American race, as well as persistent obstructive factors such as enlarged turbinates and septal deviation. Children with OSA may also be predisposed to redevelop the condition as adults, although no long-term prospective studies have been done.
Tips for Talking to Parents

- **Explain what OSA** is in simple terms, including the interaction between a child's narrow airway and obstructive elements (e.g., adenotonsillar hypertrophy) on the one hand and the upper airway muscles that work to keep the airway open on the other. Also discuss the associated sleep disruption (a good analogy for frequent arousals is like being poked in the arm every few seconds while you are asleep) and the subsequent effects of sleep fragmentation on daytime functioning.

- **Reassure parents.** Parents are often worried that their child will stop breathing during the night. Reassure parents that the brain's respiratory control centers will respond to breathing pauses by restarting the breathing process.

- **Explain risk factors** for OSA, primarily enlarged tonsils and adenoids. Discuss the potential role of other risk factors such as chronic allergies and asthma. If the child is significantly overweight, address the issue upfront.

- **Review the daytime consequences of OSA.** Parents may not attribute hyperactivity, inattention, moodiness, or poor school performance, for example, to a sleep problem.

- **Explain what to expect from the overnight sleep study.** It is particularly important to directly address any concerns the child may have (e.g., “can my Mom stay with me?” “Are there any needles?”) and to answer any parental questions regarding the procedure.

- **Review the results of the overnight sleep study.** In interpreting the study's implications, it is important to ask parents if the child's breathing was “typical” on the night of the study. Parents frequently report that their child “slept much better” in the lab, which may be associated with allergic triggers in the home. Explain the significance of “breathing pauses” and dips in O2 level, and put the numbers in context (e.g., “Adults must have at least 5 breathing pauses per hour to be diagnosed with sleep apnea, but in children we use a lower threshold of 1 to 2 pauses per hour”).

- **Discuss the risks and benefits of treatment options,** including AT, weight loss, and other treatment choices.

- **Encourage any parent who snores loudly** and has daytime symptoms to be evaluated for sleep apnea.

*See Appendix D10 for a parent handout on OSA and Appendix D11 for a handout on Your Child's Sleep Study.*
Restless legs syndrome (RLS) is a neurologic, primarily sensory disorder, characterized by an almost irresistible urge to move the legs that is often accompanied by uncomfortable sensations in the extremities. These subjective symptoms of a “need” to move and uncomfortable and unpleasant sensations (dysesthesias) occur primarily in the legs, although other body parts (e.g., arms) can also be involved. The urge to move and sensory components are usually at least partially relieved by movement, including walking, rocking, shaking, stretching, and rubbing, but only as long as the motion continues. Most episodes begin or are exacerbated by rest or inactivity, such as lying in bed to fall asleep or riding in a car for prolonged periods. Both the likelihood of having symptoms and the severity of symptoms tend to increase with the duration of inactivity. A unique feature of RLS is that the timing of symptoms also appears to have a circadian component, in that they often peak in the evening hours. Some patients with RLS, however, do not describe a significant sensory component, but instead experience the urge to move the legs and fidgetiness during periods of rest as the primary or sole presenting symptom. RLS is a clinical diagnosis that is based on the presence of these key symptoms.

Restless Legs Syndrome

RLS is a clinical diagnosis, characterized by an urge to move the legs often accompanied by uncomfortable sensations in the lower extremities. Symptoms typically occur during periods of inactivity and are worse, or exclusively present, at night. The urge to move and discomfort are temporarily relieved by increased movement of the legs. A common presenting complaint by parents of children with RLS is bedtime resistance and difficulty falling asleep.

A New Name and a New Definition

In 2013, the name of the disorder was officially changed to Willis-Ekbom disease. However, because the term RLS is at the present time much more familiar to most audiences and it is the terminology used in the International Classification of Sleep Disorders, Third Edition (2014), RLS will be used in this chapter and throughout the book. In addition, consensus definitions of pediatric RLS and of periodic limb movement disorder (PLMD) were developed and published in 2013 by a panel of pediatric sleep experts. These revised definitions and special considerations in pediatric patients are included in Tables 16.1, 16.2, and 16.3.

In RLS, the conditions under which the symptoms are most likely to occur are also the same conditions that promote sleep initiation (at night, during periods of inactivity, and rest). Conversely, the behaviors that relieve symptoms (physical activity and motion) are those most likely to interfere with sleep onset. In young children, in particular, parents may interpret these behaviors as bedtime resistance and refusal.

PLMD is characterized by a sequence (4 or more) periodic, repetitive, brief (0.5-10 seconds), and highly stereotyped limb jerks typically occurring at 20- to 40-second intervals and separated by >5- and <90-second intervals. These movements occur primarily during sleep, although some patients have periodic limb movements (PLMs) during wake periods as well. Although the upper extremities may also be affected, these movements most commonly occur in the legs and commonly consist of rhythmic extension of the big toe and dorsiflexion at the ankle. In contrast to patients with RLS, individuals with PLMs are usually unaware of these movements; however, these movements may result in arousals during sleep and consequent significant sleep disruption.
The diagnosis of PLMs requires overnight polysomnography (PSG) to document the characteristic limb movements with anterior tibialis electromyography (EMG) leads. The diagnosis of PLMD requires a minimum number of limb movements per hour (>5 in children) in the presence of a clinically significant sleep disturbance or complaint of daytime fatigue (e.g., nonrestorative sleep). Finally, it should be noted that occasional PLMs that occur primarily or exclusively in association with other conditions, such as sleep apnea, narcolepsy, or antidepressant use in adults, are considered by some to be a nonspecific EMG finding of unclear clinical significance.

TABLE 16.1. International RLS Study Group Consensus Diagnostic Criteria for RLS (2013)

RLS, a neurologic sensorimotor disorder often profoundly disturbing sleep, is diagnosed by ascertaining a syndrome that consists of all of the following features:
1. An urge to move the legs usually but not always accompanied by or felt to be caused by unpleasant and unpleasant sensations in the legs†
2. The urge to move the legs and any accompanying unpleasant sensations begin or worsen during periods of rest or inactivity such as lying down or sitting
3. The urge to move the legs and any accompanying unpleasant sensations are partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues.‡
4. The urge to move the legs and any accompanying unpleasant sensations during rest or inactivity only occur or are worse in the evening or night than during the day**
5. The occurrence of the above features are not solely accounted for as symptoms primary to another medical or a behavioral condition (e.g., myalgia, venous stasis, leg edema, arthritis, leg cramps, positional discomfort, habitual foot tapping).††

Specifier for clinical significance of RLS: The symptoms of RLS cause significant distress or impairment in social, occupational, educational, or other important areas of functioning by the impact on sleep, energy/vitality, daily activities, behavior, cognition, or mood.

Specifiers for clinical course of RLS:‡‡
A. Chronic-persistent RLS: symptoms when not treated would occur on average at least twice weekly for the past year.
B. Intermittent RLS: symptoms when not treated would occur on average <2/wk for the past year, with at least five lifetime events.

†Sometimes the urge to move the legs is present without the uncomfortable sensations and sometimes the arms or other parts of the body are involved in addition to the legs.

‡For children, the description of these symptoms should be in the child's own words.

‡When symptoms are very severe, relief by activity may not be noticeable but must have been previously present.

**When symptoms are very severe, the worsening in the evening or night may not be noticeable but must have been previously present.
††These conditions, often referred to as “RLS mimics,” have been commonly confused with RLS, particularly in surveys because they produce symptoms that meet or at least come close to meeting criteria 1-4 above. The list here gives some examples that have been noted as particularly significant in epidemiologic studies and clinical practice. However, RLS may also occur with any of these conditions, requiring a clear delineation of the RLS feelings from the other sensations.

‡‡The clinical course criteria do not apply for pediatric cases or for some special cases of provoked RLS such as pregnancy or drug-induced RLS, in which the frequency may be high but limited to the duration of the provocative condition.


Because RLS and PLMD appear to share a common underlying pathophysiology in regard to dopaminergic dysfunction in the central nervous system (CNS), they often occur concomitantly and result in sleep disruption (sleep-onset delay in RLS and sleep fragmentation in PLMD). Although similar studies have not been conducted with children or adolescents, findings in adults indicate that 70% to 90% of adults with RLS have PLMs. Recent genetic evidence suggests that comorbid RLS with PLMs may be a distinct phenotype from RLS alone. Conversely, the presence of PLMs on overnight PSG is supportive of (although not necessary for) the diagnosis of RLS in both adults and children. Thus, although they are clinically distinct entities, these two related sleep disorders are typically discussed together.

**TABLE 16.2. Special Considerations for the Diagnosis of Pediatric RLS**

- The child must describe the RLS symptoms in his or her own words.
- The diagnostician should be aware of the typical words children and adolescents use to describe RLS.
- Language and cognitive development determine the applicability of the RLS diagnostic criteria, rather than age.
- It is not known if the adult specifiers for clinical course apply to pediatric RLS. As in adults, a significant impact on sleep, mood, cognition, and function is found. However, impairment is manifest more often in behavioral and educational domains.
- PLMD may precede the diagnosis of RLS in some cases.
- Clinical features support the diagnosis of pediatric RLS.
- The following features, though not essential for diagnosis, are closely associated with pediatric RLS and should be noted when present: (1) PLMs >5/h; (2) Family history of RLS among first-degree relatives; (3) Family history of PLMs >5/h; (4) Family history of PLMD among first-degree relatives.

TABLE 16.3. Criteria for the Diagnosis of Pediatric PLMs During Sleep (2013)

1. PSG shows repetitive stereotyped limb movements that are:
   a. 0.5-10 s in duration
   b. minimum amplitude of 8 V above resting electromyogram (EMG)
   c. in a sequence of four or more movements,
   d. separated by an interval of more than 5 s (from limb movement onset to limb movement onset) and
      less than 90 s (inter-movement intervals often are short and variable in children)
2. The PLMI exceeds 5/h in pediatric cases.
3. The PLMs cause clinically significant sleep disturbance or impairment in mental, physical, social,
   occupational, educational, behavioral, or other important areas of functioning.
4. The PLMs are not better explained by another current sleep disorder, medical or neurologic disorder,
   mental disorder, medication use, or substance use disorder (e.g., exclude from PLMs counts the
   movements at the termination of cyclically occurring apneas).

Adapted from Picchietti DL, Bruni O, de Weerd A, et al. Pediatric restless legs syndrome diagnostic
criteria: an update by the International Restless Legs Syndrome Study Group. Sleep Med

Although there has been substantial progress in our understanding of the biologic basis, epidemiology, genetics,
and clinical management of RLS and PLMD over the past decade, many questions still remain. Furthermore, it is
important to note that these two disorders have only been recently recognized in children and adolescents. For
example, there are currently no approved medications for the treatment of pediatric RLS and PLMs. Recent
investigations indicate that both of these disorders are much more common in adults than previously thought,
and the same is likely true in children and adolescents. Therefore, pediatric practitioners should consider RLS
and PLMD in the differential diagnosis for any sleep problem presentation that includes significant difficulties
falling asleep, as well as nighttime awakenings with restless sleep or unexplained symptoms of daytime
somnolence, including neurobehavioral symptoms (e.g., inattentiveness, hyperactivity, irritability).

EPIDEMIOLOGY

Restless Legs Syndrome

- **Adults:** Both RLS and PLMD are common disorders in adults, although significantly underdiagnosed. In
  adults, the prevalence of RLS is estimated to be 5% to 10%, with about 3% of patients having frequent and/or
  moderate to severe symptoms. Furthermore, a history of childhood onset (before the age of 10 years) is
  present in approximately 18% of adults with RLS, while 25% of adults recall the onset of symptoms in the
  second decade of life.

- **Pediatrics:** Previous studies have examined the prevalence of RLS symptoms in a number of different
  pediatric populations, using a variety of RLS definitions, with prevalence rates ranging from 1% to 6%. A large
  telephone survey study (pediatric “REST” study) of over 10,000 families in the United States and the United
  Kingdom, using the 2003 National Institutes of Health pediatric RLS consensus criteria, found a prevalence of
  “definite” RLS in about 2% in both 8- to 11-year-olds and 12- to 17-year-olds; this represents some 980,000
  school-aged children in the United States. One-quarter to one-half of these patients had moderate to severe
  symptoms. Another much smaller study in 12- to 20-year-olds found a prevalence of RLS of 8.4%. In contrast
to a female preponderance (2:1) in adult RLS, there does not appear to be a gender difference in pediatric RLS.

**Periodic Limb Movement Disorder**

- **Adults:** The prevalence of PLMs in adults appears to be positively correlated with age (4%-11% of adults aged 30-50 years, and 25%-58% in the elderly).
- **Pediatrics:** Because a definitive diagnosis requires PSG, there are fewer data regarding the prevalence of PLMs in childhood.
  - Prevalence rates of PLMs greater than 5 per hour in clinical populations of children referred for sleep studies range from 5% to 27%; in survey studies of PLM symptoms, rates are between 8% and 12%.
  - Several studies in referral populations have found that PLMs occur in as much as one-fourth of children diagnosed with attention-deficit hyperactivity disorder (ADHD). Other recent studies have found an association between symptoms of hyperactivity and the presence of PLMs; however, the prevalence of PLMD in the general population of children with ADHD is unknown.
  - PLMs appear to be more prevalent in Caucasian children than in African American children.

**ETIOLOGY AND RISK FACTORS**

**Restless Legs Syndrome**

- **Genetic link:** Overall, it is estimated that 50% to 60% of adult RLS patients have a positive family history. “Early-onset” RLS (i.e., onset of chronic, typically mild to moderate symptoms before 35-40 years of age), also termed “primary” or “familial” RLS, appears to have a particularly strong genetic component. It is estimated that 40% to 92% of these cases are familial, and the prevalence of RLS in first-degree relatives of patients with early-onset RLS appears to be 6 to 7 times that of the general population. Data from one population study showed that RLS aggregates in families with a familial rate of 77%, a sibling relative risk of 3.6%, and an offspring relative risk of 1.8%. In the population-based “REST” study, between 70% and 80% of children with definite RLS had at least one biologic parent with RLS and 16% had both parents affected. The mode of inheritance was previously postulated to involve predominantly autosomal-dominant or pseudodominant autosomal recessive patterns, but a more complex genetic schema is likely. Although at least five genetic variants have been linked to RLS and a number of genetic loci, such as the BTBD9 locus on chromosome 6p, have been identified in large population studies, no “candidate gene” has been discovered, and there may be different genotypes for various phenotypic manifestations of RLS and/or PLMs. Environmental factors such as iron deficiency (see below) may also play a role in familial RLS.
- **Iron deficiency**, as a number of studies have implicated low iron in both adults and children as an important etiologic factor for the presence and severity of both RLS symptoms and PLMs. For example, magnetic resonance imaging studies in adults with RLS have demonstrated decreased iron stores in a number of regions in the CNS, including the basal ganglia, putamen, and substantia nigra, irrespective of body iron stores. The postulated underlying mechanism is related to the role of iron as a cofactor of tyrosine hydroxylase in a rate-limiting step of the synthesis of dopamine. In turn, dopaminergic dysfunction has been implicated as playing a key role in the genesis of RLS. In addition, there are multiple other possible mechanisms for the effect of low iron on dopamine. Recent studies have also explored potential roles for abnormalities in a number of proteins and pathways involved in iron metabolism and storage, including transferrin receptor expression, ferritin subunits, hepcidin, and ferroportin. In addition to dopamine and iron, inflammatory pathways are

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considered important to the development of RLS. For example, there is preliminary evidence of a genetic association between interleukin genes and RLS.

As a marker of decreased iron stores, serum ferritin levels in both children and adults with RLS are commonly low. Several pediatric case series have suggested that lower-than-normal ferritin levels (defined as <50 ng/mL) are found in some 70% to 75% of children with RLS. A ferritin level below 50 ng/mL increases the risk of RLS and is typically the threshold used to determine treatment with iron supplementation. It should be noted, however, that low iron is not universally present in patients with RLS; conversely, many children with iron deficiency do not have RLS symptoms.

The threshold at which low iron stores appear to have a significant impact seems to be different for peripheral versus CNS effects; for example, a reduction in hemoglobin synthesis typically occurs only when ferritin levels drop below 12 ng/mL. Thus, low iron can exacerbate RLS symptoms in the face of normal hemoglobin and hematocrit levels. In addition, because ferritin is an acute phase reactant, elevated serum levels may occur with systemic inflammatory and metabolic processes, such as diabetes, and with concurrent minor infectious illnesses. Therefore, a ferritin level within or above the normal range in the face of an upper respiratory infection in a child, for example, does not necessarily indicate normal iron stores, and the level should be repeated once the infection has resolved.

Children with ADHD may also be more likely to have low serum ferritin levels, and children with RLS symptoms and ADHD may be at particularly high risk for very low ferritin levels (<12 ng/mL). In addition, iron deficiency might decrease the effectiveness of psychostimulant treatment for ADHD.

**Serum Ferritin Levels**

Although it may still be considered in the normal range, studies suggest that a ferritin level below 50 ng/mL in a child or adolescent with RLS or PLMD warrants treatment with iron supplementation.

- **Medical disorders**, such as diabetes mellitus, end-stage renal disease, cancer, peripheral vascular disease, rheumatoid arthritis, liver transplantation, and hypothyroidism. RLS appears to be more common in pediatric patients on dialysis. RLS symptoms also may occur concurrently in children with a variety of infectious processes (e.g., mycoplasma, Group A streptococcus).

- **Sickle cell disease**, as symptoms of RLS are common, especially in children with elevated PLMs.

- **Migraine headaches**, as there is an increased frequency of RLS in migraine patients compared to controls (22% versus 5%). Furthermore, the treatment of sleep disorders, such as sleep apnea and RLS, either with behavioral or pharmacologic approaches including serotonergic and dopaminergic compounds, can result in an improvement in migraines. Both sleep related movement disorders, such as RLS, and migraine may be related to serotonergic system abnormalities. Alternatively, prodromal symptoms of migraine (e.g., drowsiness, irritability, mood changes, hyperactivity) suggest a key role for the same dopaminergic system involved in sleep related movement disorders.

- **Other pain syndromes**, including nonmigraine headaches and recurrent abdominal pain, are associated with RLS.

- **Pregnancy**, as RLS occurs in up to 15% of pregnant women, most commonly after the 20th week in association with decreased iron levels. Some women (approximately 1 in 7) who develop RLS in pregnancy continue to have symptoms postpartum.
Insufficient sleep, as any underlying sleep disturbance contributing to chronic sleep loss may exacerbate RLS.

Medications, especially antihistamines, such as diphenhydramine. Other medications that may exacerbate RLS include antidepressants (selective serotonin reuptake inhibitors [SSRIs], mirtazapine, and tricyclic antidepressants [TCAs]); neuroleptics; sedatives or narcotics (withdrawal from); lithium; calcium channel blockers; H2 blockers, such as cimetidine; phenytoin; and dopamine receptor blockers (antiemetics such as prochlorperazine, metoclopramide).

Caffeine, as the additional stimulation can exacerbate underlying RLS.

Drugs and chemicals, as even sedating substances, such as alcohol, may increase RLS symptoms.

Periodic Limb Movement Disorder

Iron deficiency and low serum ferritin levels: Both altered iron metabolism and storage, as well as central dopaminergic dysfunction, may be involved in the pathophysiology of PLMD. In addition, a relationship between an elevated PLM index (PLMI) and low ferritin levels in children has been demonstrated.

Medical conditions, including metabolic disorders, uremia, childhood leukemia, liver transplantation, and spinal cord injury.

Sickle cell disease, as elevated PLMs are common in children with sickle cell disease. In one study, 23% of the sample had a PLMI ≥5 per hour, with decreased sleep efficiency and increased arousals from sleep.

Williams syndrome, as both symptoms and PSG documentation of PLMs appear to be more common in children with this developmental disorder.

Nocturnal enuresis, as children with bedwetting have more sleep fragmentation and increased PLMs.

Medications, most notably SSRIs, as well as TCAs and atypical antidepressants such as venlafaxine. Withdrawal from other medications, including anticonvulsants, benzodiazepines, and hypnotics, can exacerbate PLMs.

Obstructive sleep apnea (OSA), and associated arousals may be associated with PLMs in both adults and children. In one pediatric study, 50% of prepubertal children with PLMs also had OSA documented by overnight PSG; treatment of the sleep apnea (adenotonsillectomy) resulted in resolution of the PLMs in 52% of these cases.

Narcolepsy, may also be accompanied by PLMs during sleep.

PRESENTATION AND SYMPTOMS

The cardinal features of RLS in children are listed below (a screening questionnaire for RLS is provided in Appendix B5), but it should be kept in mind that the initial complaint in the primary care setting may be frequently delayed sleep onset and/or daytime behavior or attention problems, rather than RLS symptoms per se. Similarly, patients with PLMs rarely present with a chief complaint of “kicking movements during the night,” but parents may report restless sleep and increased body movements during sleep. Table 16.4 presents common presenting symptoms for RLS and PLMD.
Sleep Symptoms
- Bedtime resistance and behavior problems
- Difficulty falling asleep
- Restless sleep and nighttime awakenings

Motor Symptoms
- Walking, pacing, or running about at bedtime
- Increased leg movements and/or restlessness at bedtime, during sleep, and with long periods of inactivity

Sensory Symptoms
- Leg pain or discomfort in the evening or during the night with periods of prolonged inactivity

Daytime Symptoms
- Excessive daytime sleepiness or fatigue, including difficulty waking in the morning, falling asleep in school or at inappropriate times, and increased need for naps
- Mood changes, such as irritability, low frustration tolerance, mood swings, and depression and anxiety
- Acting out behaviors, including aggression, hyperactivity, oppositional or defiant behavior, and impulsivity
- ADHD symptoms: inattention, poor concentration, distractibility, academic problems

Restless Legs Syndrome

Motor-Sensory Complaints

- **Urge to move:** Many children with RLS describe a subjective need to move their legs (e.g., stretching, kicking) or their whole body, which may be distinct from perceiving that the movements relieve the sensory symptoms. This urge is typically experienced as both unpleasant and under-limited voluntary control, somewhat analogous to the drive children may describe immediately preceding motor tics. Some children simply run around without indicating the need to move their legs. In addition to movement, some children and parents report that the sensory symptoms may also be relieved by counter-stimulation, such as rubbing or application of hot or cold stimuli.

- **Sensory symptoms:** Although symptoms of RLS may start as early as 6 to 12 months of age and thus are very difficult to identify, even relatively young children are often able to articulate and describe the sensory symptoms of RLS. In clinical interviews, children may use colorful descriptors such as “soda bubbling through my veins,” “goose-bumps on the inside,” “tiny nails poking my legs,” and “people wrestling in my legs.” They may use terms like squeezing, tingling, wiggling, itching, popping, “funny” feelings, shaking, tiredness, aching, or pulling and tugging of the legs. The symptoms are sometimes described as specifically “painful,” and younger children may use more nonspecific descriptors like “hurting.” It should be noted that in the office setting children may not volunteer these symptoms without some (nondirective) prompting from the clinician. Parents may be able to offer descriptions that their child has used at home, although at times parents are surprised by their child's responses following questioning in the office.

Although no specific body position is associated with RLS symptoms, the conditions most likely to provoke symptoms are long periods of motor inactivity combined with decreased mental activity, such as lying in bed or long car rides. In milder cases, symptoms may only be precipitated by long periods of inactivity (e.g., plane flights). Some children may experience RLS symptoms during prolonged periods of sitting in school.
Several recent studies in the United States and Europe have specifically examined presenting symptoms in children who meet diagnostic criteria for RLS. While many children described symptoms similar to those in established diagnostic criteria, others used idiosyncratic or colorful terminology such as “ants or spiders in the legs, legs want to kick, need to stretch.” All children reported feeling tired or sleepy during the day and insomnia was mentioned by 61% of the patients. One study found that pediatric patients used 16 different categories of descriptors for restless legs sensations, with “need to move/kick,” “pain/hurts,” “uncomfortable/cannot get comfortable,” and “like bugs or ants/crawling” the most common descriptors. Two-thirds reported daytime sensations, and nearly half had arm involvement. These children were also quite articulate in describing the subjective negative impact on sleep, cognitive function, and mood. Another pediatric study found that children's drawings of their RLS symptoms may also provide useful diagnostic information (Figure 16.1).

**Clinical Prompts for RLS**

While most children with RLS experience an “urge” to move their legs when sitting still and/or at bedtime, they may not spontaneously express it as such. A useful question to elicit this symptom is: “If you had to lie perfectly still while you were falling asleep, would you be able to do it?” Similarly, one of the best ways to elicit RLS sensory symptoms is to ask if anything “bothers” the child or adolescent at bedtime. Other ways to find out include asking the nondirective question, “When you are trying to fall asleep at bedtime, does anything make it harder? Does anything feel funny or hurt, like your tummy, your head, your arms, your legs?” Children and adolescents with RLS will usually positively respond to the “legs” aspect of the question.
"Growing pains": Growing pains may be described as recurrent limb discomfort or pain often occurring at bedtime and/or resulting in nightwakings. These are a common complaint of children. Symptoms may be quite dramatic such as awakening after a few hours of sleep with screaming, crying, kicking, or hitting the legs. The relationship between growing pains and RLS/PLMD is still not clear; while there is considerable overlap in the diagnostic criteria for childhood RLS and growing pains, walking to obtain relief, for example, seems unique to RLS. The literature also indicates that RLS and growing pains more commonly co-occur than would be expected based on chance alone, and the family histories of RLS and growing pains often overlap.

Although this childhood symptom has been shown to be associated both with concurrent pediatric RLS and PLMD and with later development of RLS symptoms in adulthood, some studies have suggested this relationship is fairly nonspecific. For example, in the pediatric "REST" study, a history of "growing pains" was reported in 77% to 85% of children and adolescents meeting diagnostic criteria for RLS, but it was also found in 61% to 64% of subjects without RLS. On the other hand, children with growing pains were reported to be 3 times more likely to have a PLMI ≥5 per hour than children without growing pains on PSG, suggesting that growing pains might lie on the phenotypic spectrum of RLS. A recent twin family study of growing pains provides evidence for a genetic etiology and for a genetic relationship to RLS.

Problems initiating and maintaining sleep: Because RLS symptoms are usually worse in the evening, bedtime struggles and difficulty falling asleep are two of the most common presenting complaints. In particular, children with RLS may attempt to avoid going to bed because lying still exacerbates their symptoms, and this may be interpreted as bedtime resistance. RLS may also occur during nightwakings. Sleep disturbance was reported in significantly more RLS subjects (66%-70%) in the pediatric "REST" study compared to 34% to 44% of those without RLS. Children with PLMs also have frequent sleep-onset and maintenance problems.

Restlessness and increased motor movements during sleep: Parents of children with RLS and PLMD may complain that their child is a restless sleeper, moves around or even falls out of bed during the night, or that the child's bedcovers are always disheveled in the morning. Kicking, "jerking," or "twitching" leg movements may be observed during sleep. Children with PLMs may also complain of leg pain/discomfort at night and are more likely to get out of bed during the night. PSG supports these reports of disrupted sleep: more awakenings, stage shifts, and spontaneous arousals, as well as an increased percentage of "light" stage 1 sleep.

Other sleep issues: Children with PLMs have been reported to have a higher prevalence of parasomnias, specifically disorders of arousal (see Chapter 11). OSA has also been reported to be more common in these children (see Chapter 15).

Diagnostic Criteria
In addition to the 2013 consensus definitions developed for pediatric RLS and PLMD (Tables 16.1, 16.2, 16.3), the ICSD-3 diagnostic criteria are presented in Tables 16.5 and 16.6.

Diagnosis
The diagnosis of RLS is based solely on clinical history, whereas PLMD requires an objective documentation by an overnight sleep study (PSG). However, because a high percentage of RLS
patients also have PLMD, the finding of PLMs on PSG is also supportive of the diagnosis of RLS.

Associated Features

- **Daytime sleepiness and daytime behavior problems**: Sleep-onset and maintenance difficulties related to RLS and/or PLMs that lead to reduced sleep time may result in daytime sleepiness. However, explicit symptoms of excessive daytime sleepiness (e.g., difficulty waking in the morning, drowsiness) are relatively uncommon. More commonly, neurobehavioral symptoms indicative of daytime sleepiness (e.g., hyperactivity, inattentiveness, poor focusing) are the predominant daytime symptom described in association with RLS and PLMD. Behavior problems, including mood problems and oppositional defiant behavior, may also result from the frequent nighttime arousals.

### TABLE 16.5. Diagnostic Criteria: RLS*

A. An urge to move the legs, usually accompanied by or thought to be caused by uncomfortable and unpleasant sensations in the legs. These symptoms must:
   1. Begin or worsen during periods of rest or inactivity such as lying down or sitting;
   2. Be partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues; and
   3. Occur exclusively or predominantly in the evening or night rather than during the day.

B. The above features are not solely accounted for as symptoms of another medical or behavioral disorder (e.g., leg cramps, positional discomfort, myalgia, venous stasis, leg edema, arthritis, habitual foot tapping).

C. The symptoms of RLS cause concern, distress, sleep disturbance, or impairment in mental, behavioral, or other important areas of functioning.

For children, the description of these symptoms should be in the child’s own words.

*ICD-9-CM code: 333.94; ICD-10-CM code: G25.81


### TABLE 16.6. Diagnostic Criteria: PLMD*

A. PSG demonstrates PLMs, as defined in the most recent version of the American Academy of Sleep Medicine Manual for the Scoring of Sleep and Associated Events.

B. The frequency is >5/h in children or >15/h in adults.

C. The PLMs cause clinically significant sleep disturbance or impairment in mental, physical, social, occupational, educational, behavioral, or other important areas of functioning.

D. The PLMs and the symptoms are not better explained by another current sleep disorder, medical or neurologic disorder, or mental disorder (e.g., PLMs occurring with apneas or hypopneas should not be scored).
Neuropsychiatric conditions:

ADHD. RLS symptoms and PSG-documented PLMs are significantly more common in children diagnosed with ADHD or with symptoms of hyperactivity and impulsivity. Up to 44% of children with ADHD have RLS symptoms and up to 26% of patients with RLS have ADHD symptoms. RLS and ADHD may also coexist. In this situation, ADHD symptoms in this situation are typically more severe than with ADHD alone. Treatment of RLS with either supplemental iron or dopaminergic agents, may reduce or eliminate ADHD symptoms. A meta-analysis of PSG studies in children with ADHD concluded that the only significant polysomnographic difference between ADHD and control children was an increase in PLMs.

Depression and anxiety. There is an increased risk for depression and anxiety symptoms in those with RLS. A recent study found that 64% of pediatric patients with RLS had one or more comorbid psychiatric disorders; mood disturbances in 29% patients, anxiety disorders in 12% patients, and behavioral disturbances in 11% patients. As a part of clinical care, 15 of these patients underwent pharmacogenomic testing, and metabolic abnormalities were predicted by genotyping in 12 of 15 (80%) patients. Thus, an important relationship might exist between psychotropic medication, and possibly pharmacogenomic factors, in children and adolescents with symptoms of RLS. Treatment of RLS symptoms may result in improved mood. In situations in which depression and anxiety appear to be comorbid with RLS, it is important to avoid the use of antidepressants known to exacerbate RLS (e.g., SSRIs, TCAs).

Decreased quality of life: Decrement in overall quality of life, and decreased psychosocial health, in children and adolescents can be associated with RLS.

Increased cardiovascular morbidity: Studies in adults have reported evidence of cardiovascular abnormalities associated with RLS and/or PLMD, including systemic hypertension, potentially due to RLS-induced sleep loss and/or RLS and PLMD-associated alterations in sympathetic tone. Epidemiologic studies in adults have also found an association between RLS and increased risk of coronary artery disease, heart failure, and stroke. In addition, the presence of RLS and/or PLMD comorbid with ADHD may further increase cardiovascular risk via imbalance in activity of the autonomic nervous system.

ADHD and RLS/PLMD

There is an association between symptoms of ADHD and both RLS and PLMD. Although the mechanism underlying this relationship is not fully understood, several possibilities exist. First, inadequate and disrupted sleep resulting from RLS or PLMD may cause “ADHD-like” symptoms. Second, daytime symptoms of RLS (e.g., fidgeting during periods of inactivity) may be interpreted as ADHD symptoms. Conversely, ADHD-associated evening hyperactivity may be labeled as RLS (but there is typically not a sensory component with ADHD). Finally, there may be a common underlying metabolic (e.g., iron deficiency) or genetic predisposition for both disorders.
**Medical history:** This may include a history of iron-deficiency anemia. Children with the medical conditions listed above are at increased risk for secondary RLS and PLMD. Concurrent medications (e.g., SSRIs, sedating antihistamines) should be reviewed as possible contributory factors. Caffeine intake may also exacerbate symptoms of RLS.

**Developmental history:** The history is generally noncontributory except for specific neurodevelopmental disorders that may have an increased risk of PLMs (e.g., Williams syndrome).

**Family history:** In childhood-onset RLS, a positive family history (first-degree relative) is very common. However, due to the under-recognition of this disorder, it should be kept in mind that adult family members with RLS may not have been diagnosed, and instead there may be a strong family history of insomnia, restless sleep, or growing pains. It is worth asking parents about their own symptoms at bedtime.

**Behavioral assessment:** This may reveal significant behavior problems, mood disturbances, and ADHD.

**Physical examination:** The examination is generally normal (including the neurologic examination) in children with primary RLS or PLMD and in fact, the presence of abnormal physical findings (e.g., pain, limitation of extremity movement) suggests an alternative diagnosis (Table 16.4). However, the primary care clinician may note from behavioral observation that many children with RLS are not able to sit still for long periods and often jiggle their legs or move around while sitting for prolonged periods.

**Diagnostic tests:**

**PSG:**

- **RLS:** RLS is a clinical diagnosis and, thus, PSG is not strictly required to make the diagnosis. However, documentation of PLMs on an overnight sleep study is often helpful for the diagnosis because the presence of PLMs is part of the supportive diagnostic criteria for RLS in children (see Table 16.2), particularly in situations in which the child's description of RLS symptoms may be difficult to elicit due to limited language skills. In addition, the co-occurrence of RLS and PLMD may affect pharmacologic management strategies, and reduction of PLMs on PSG may serve as a more objective “marker” of treatment response.

- **PLMD:** PSG is required for the diagnosis of PLMD. The anterior tibialis EMG used to monitor PLMs is part of the standard PSG montage in all sleep laboratories for both adults and children. PLMs are defined as four or more consecutive leg movements of minimal amplitude of 8 V above baseline EMG, lasting 0.5 to 10 seconds, separated by at least 5-second and no more than 90-second intervals. To assess sleep disturbance, leg movements that are associated with an arousal (within 3 seconds of leg movement) or awakening are scored, although in general children are less likely than adults to have associated arousals. An overall index of PLMs per hour is provided, as is an index of PLMs associated with arousals per hour. In adults, the PLMI usually exceeds 15 per hour, while in children, a PLMI >5 is generally considered significant (see Figure 16.2). Finally, it should be noted that there may be considerable night-to-night variability in PLMs, and thus a single overnight sleep study may either under- or overestimate “typical” severity.

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**American Academy of Sleep Medicine Practice Parameters for the Nonrespiratory Indications for Polysomnography in Children (2013)**

PSG is indicated for children suspected of having PLMD for diagnosing PLMD. (Standard)

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Laboratory tests: Because of the association between RLS and PLMD and iron deficiency, especially low ferritin, a complete blood count and serum ferritin level (hemoglobin and hematocrit and indices may be normal) should be checked, particularly in children who may be at high risk for iron deficiency (e.g., toddlers, adolescent girls). A serum ferritin <50 ng/mL is associated with RLS symptoms.

There are also now several studies in adults which show an association between low serum 25-hydroxyvitamin D levels (deficiency defined as <50 nmol/L) and the presence and severity of RLS symptoms. Children with growing pains may also have low vitamin D levels. Furthermore, treatment with vitamin D oral or intravenous supplementation has been reported to result in significant symptomatic improvement in adults with RLS.

Differential Diagnosis
A summary of common pediatric RLS “mimics” is presented in Table 16.7.

Nocturnal leg discomfort or pain is frequently associated with musculoskeletal and neuromuscular complaints in children, and may be confused with RLS or PLMD. Many of these conditions have specific signs and symptoms that distinguish them from RLS; these include physical findings (e.g., tibial tuberosity tenderness with Osgood-Schlatter syndrome or erythema, edema, and joint tenderness with juvenile rheumatoid arthritis), exacerbation (rather than relief) with movement and improvement with rest, lack of a temporal (evening) association, and sporadic occurrence. These conditions include (listed from relatively more common to less common) the following:
FIG 16.2. Periodic limb movements.

**TABLE 16.7. Differential Diagnosis of Pediatric RLS**

<table>
<thead>
<tr>
<th>Common Mimics</th>
<th>Less Common Mimics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positional discomfort</td>
<td>Leg cramps</td>
</tr>
<tr>
<td>Sore leg muscles</td>
<td>Arthritis</td>
</tr>
<tr>
<td>Ligament sprain/tendon strain</td>
<td>Other orthopedic disorders</td>
</tr>
<tr>
<td>Positional ischemia (numbness)</td>
<td>Peripheral neuropathy</td>
</tr>
<tr>
<td>Dermatitis</td>
<td>Radiculopathy</td>
</tr>
<tr>
<td>Bruises</td>
<td>Myelopathy</td>
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<tr>
<td>Growing pains</td>
<td>Myopathy</td>
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<td></td>
<td>Complex regional pain syndrome</td>
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<td></td>
<td>Fibromyalgia</td>
</tr>
<tr>
<td></td>
<td>Drug-induced akathisia</td>
</tr>
<tr>
<td></td>
<td>Sickle cell disease</td>
</tr>
</tbody>
</table>

- Exercise-related muscular pain
- Transient nerve compression (“pins and needles”) following prolonged inactivity
- “Growing pains,” nonspecific limb discomfort that frequently wakes children from sleep; may occur in
association with RLS but are also common in children without RLS.

- Nocturnal leg cramps, which may be associated with electrolyte disturbance or neuromuscular disorders

- Dermatitis and associated irritation or pruritus
- Orthopedic conditions, such as Osgood-Schlatter syndrome and chondromalacia patella
- Chronic rheumatologic conditions such as juvenile rheumatoid arthritis
- Peripheral neuropathy, radiculopathy, and myopathy

**Sleep-related motor restlessness** at bedtime or during the night may be due to:

- Hypnic jerks ("sleep starts"), which typically occur at wake-sleep transition only
- Myoclonic seizures, in which movements are more prominent in wakefulness
- Sleep apnea-related movement arousals
- Parasomnias

- Movements associated with ADHD, as children with ADHD may manifest increased late-day hyperactivity associated with inadequate control of ADHD in the evening or rebound from psychostimulants. Children with ADHD have also been shown to have more motoric activity during sleep.
- Akathisia (inner restlessness or urge to move), which is often associated with antipsychotics and other medications.

**Prolonged sleep onset and nighttime awakenings** may result from multiple other causes, including behavioral issues (see Chapters 7 and 8), OSA (see Chapter 15), or delayed sleep-wake phase disorder (see Chapter 18).

**Excessive daytime sleepiness** and associated neurobehavioral impairments can result from any other sleep disorder that impacts on sleep quality and quantity, including OSA and behaviorally induced insufficient sleep.

**MANAGEMENT**

**When to Refer**

It is possible to diagnose and treat children presenting with symptoms of RLS and PLMD in the primary care setting, although overnight PSG to detect PLMs requires referral to a sleep laboratory. In particular, assessment and replenishment of iron stores (when appropriate) as a first-line treatment is a reasonable strategy before referral to a sleep specialist for additional management is considered.

**Treatment**

The decision of whether and how to treat RLS and/or PLMD depends on (1) the level of severity (intensity, frequency, and periodicity) of sensory symptoms; (2) the degree of interference with sleep; and (3) the impact of daytime sequelae in a particular child or adolescent. For example, if the symptoms of RLS result in prolonged sleep-onset latency, severe complaints of leg discomfort, or attentional problems, treatment is generally warranted.

There is less of a consensus about the treatment threshold for PLMs in children. Sleep study results can be helpful from the standpoint of quantifying the number of PLMs and associated arousals and sleep fragmentation although, as noted above, PLM-induced EEG arousals in general are seen with much less
frequency in children compared with adults. For a PLMI less than 5, usually no treatment is recommended. For an index over 5, the decision to specifically treat PLMs should be based on the presence or absence of nocturnal symptoms (restless or nonrestorative sleep) and clinical sequelae (e.g., excessive daytime sleepiness, neurobehavioral complications). In addition, comorbid sleep disorders (e.g., OSA) may be associated with PLMs, and thus treatment of the underlying sleep disorder may result in resolution of PLMs.

**Treatment Strategies**

- **Healthy sleep practices:** It is recommended that children with RLS maintain consistent bedtimes and waketimes on weekdays and weekends, have a bedtime routine, and obtain adequate nighttime sleep, especially since fatigue may exacerbate the symptoms of RLS.

- **Nonpharmacologic treatments:** Moderate exercise up to a few hours before bedtime may suppress symptoms. Walking, stretching, massaging the affected area, and applying hot or cold packs may be helpful. Biofeedback and relaxation techniques may alleviate symptoms as well as reduce stress. Keeping mentally occupied, especially during long periods of inactivity, should also be encouraged, although this may be counterproductive at bedtime in terms of sleep onset.

- **Substances to avoid:** Caffeine, alcohol, antihistamines, cold or sinus preparations, and antiemetics are known to exacerbate symptoms of RLS. In adults, drugs that worsen the symptoms of RLS include those that increase thyroid hormone activity. In addition, drugs that alleviate RLS symptoms inhibit thyroid hormone activity, possibly through alteration of the CYP4503A4 isoform.

- **Iron supplementation:** Oral iron supplements are a reasonable choice as a first-line treatment for RLS and PLMs if serum ferritin levels are low (<50 ng/mL). Although data are limited, the recommended dose is typically in the range of 3 to 6 mg/kg/day for a duration of at least 3 months, followed by increased dietary iron intake once an appropriate ferritin level is reached. In a recent study of almost a hundred children diagnosed with RLS, the majority of whom were between the ages of 5 and 11 years and 70% of whom had a ferritin level less than 30 ng/mL, approximately 80% of the children who received iron and had follow-up had improvement or resolution of their symptoms. The median time to improvement or resolution of symptoms was 3.8 months. However, there are a number of caveats in regard to iron supplementation for RLS: (1) oral iron is poorly absorbed in the gastrointestinal tract; (2) compliance with daily treatment over a 3- to 6-month period is often poor; (3) tolerance may be problematic (i.e., constipation); and (4) oral iron supplementation may only be effective at lower ferritin levels. Concomitant use of ascorbic acid (vitamin C) appears to improve absorption, while iron absorption is reduced when combined with calcium. Adult studies have found that intravenous iron is significantly more likely to be effective in raising ferritin levels and reducing RLS symptoms, although this delivery system is not commonly used in the pediatric population. However, recent data suggest that intravenous iron sucrose appears to be a relatively effective therapy for pediatric patients who do not tolerate or respond to oral iron supplements. While concerns about anaphylactic reactions have been raised, at least one study in children found side effects were transient and largely related to difficulty with intravenous line placement and gastrointestinal disturbance. A very rare complication of iron supplementation is iron overload in individuals with iron metabolism disorders such as hemochromatosis.

- **Medication:** There have been very few empirical studies on the efficacy of specific medications for RLS and PLMD in children. Thus, the following recommendations are based largely on clinical experience. Similar to other drugs used for sleep, governing principles in the use of medications for RLS in children include (1) using the lowest possible dose and (2) starting at a minimal dose and titrating up according to efficacy and tolerance. Medication should be combined with nonpharmacologic interventions as outlined...
The major medications used for RLS and PLMD in adults are listed below; dosages listed are usual ranges for adults. Medications that increase dopamine levels in the CNS have overall been found to be the most effective drugs in relieving RLS and PLMD symptoms. Currently, ropinirole and pramipexole are the only medications approved by the Food and Drug Administration specifically for the treatment of RLS and PLMD in adults. Overall, 70% to 80% of adult patients experience some relief of symptoms with medication; effects seem to be most robust in relieving RLS symptoms and improving sleep quality subjectively and in decreasing PLMs. In choosing a medication to treat significant RLS or PLMD, consultation with a pediatric sleep specialist or neurologist is recommended.

- **Dopaminergic agents**: Nonergot dopamine D2/D3 agonists are considered first-line treatment for both RLS and PLMs in adults. A recent meta-analysis of all randomized controlled trials for the pharmacologic treatment of RLS supported the well-defined efficacy of dopaminergic treatment.

- **Levodopa and carbidopa (Sinemet)**: These are given regular or slow release in 100 to 125 mg or 200 to 250 mg dosages at bedtime. An additional dose of the regular preparation may be needed during the night to avoid rebound PLMs. In the only double-blind placebocontrolled pediatric trial of a dopaminergic agent, l-dopa improved RLS/PLMS symptoms in all patients with those disorders. All of the subjects were also diagnosed ADHD; however, l-dopa did not reduce ADHD symptoms compared to placebo. Problematic side effects, which may include nausea, orthostatic hypotension, insomnia, daytime fatigue and somnolence, augmentation in early evening (see below), and morning rebound, have tended to limit the use of levodopa and carbidopa for RLS and PLMs in both adults and children.

- **Dopamine agonists**: Used to treat both RLS and PLMD, dopamine agonists are now generally considered the first-line medications in adults. They typically result in a predictable, rapid, and nearly complete resolution of symptoms, although some recent data in adults suggest that these medications may be relatively less effective in treating the sensory (pain) component of RLS and more effective in treating PLMs. A number of published case reports and small-case series have suggested that these medications may also be effective and generally well-tolerated in the pediatric population. Potential issues associated with the use of dopamine agonists include augmentation (although less common than with levodopa and carbidopa), development of tolerance, and rebound symptoms on discontinuation. Side effects reported in association with dopamine agonists include nausea, headaches, constipation, and fatigue; orthostatic hypotension may occur. Rarely, behavioral changes, including compulsive behaviors such as compulsive gambling, have been reported in adults.

- **Pramipexole (Mirapex)**: This drug has been shown in a number of adult studies to be effective in reducing symptoms in moderate or severe RLS and in decreasing PLMs as well as improving sleep quality. The dose is 0.125 to 0.75 mg at bedtime.

- **Ropinirole (Requip)**: This drug has also been reported to be effective for treating RLS in adults. The dose is 0.5 to 4 mg.

- **Other medications for RLS and PLMD**: Other classes of medications have been used in adults, and to a much lesser extent in children, to treat RLS and PLMD; these include -agonists, benzodiazepines, anticonvulsants, and opioids. A meta-analysis of pharmacologic treatment of RLS in adults suggested that other medications show efficacy in small samples and may be well-tolerated alternatives for the treatment of RLS.

- **Clonidine**: This short-acting -agonist is largely used to treat RLS symptoms; side effects include hypotension. The dose is 0.05 to 0.2 mg at bedtime.
Clonazepam (Klonopin): This is a benzodiazepine used to treat both RLS and PLMD; side effects include daytime sedation. It may exacerbate OSA symptoms and tolerance may develop. The dose is 0.5 to 2.0 mg at bedtime.

Codeine and other opiates (oxycodone, propoxyphene): Opiates are used to treat both RLS and PLMD. They have been shown to demonstrate efficacy in open-label trials in adults, possibly related to effects on dopaminergic pathways. Side effects include constipation. Dependency and addiction issues do not seem to occur in adults treated with opioids specifically for RLS. The dose for codeine is 15 to 60 mg at bedtime; oxycodone (Percodan), 5 mg at bedtime; propoxyphene (Darvon, Darvocet), 200 mg at bedtime.

Gabapentin (Neurontin): This is an anticonvulsant that has been used for RLS and is reported to improve both sensory and motor symptoms and improve sleep quality; side effects include daytime somnolence. The dose is 100 to 400 mg at bedtime. In the meta-analysis cited above, the only anticonvulsants showing good efficacy in clinical trials included the α2β-ligands such as gabapentin, gabapentin enacarbil, and pregabalin.

Many adult patients on medications, particularly those on dopaminergic agents, experience a phenomenon called augmentation. Augmentation is defined as an earlier temporal onset of RLS symptoms (i.e., afternoon), more rapid onset of symptoms with inactivity, and/or spread of symptoms to other parts of the body, occurring as a direct result of a specific therapeutic intervention.

Augmentation generally occurs within 6 weeks to 6 months after initiation of therapy or dosage change and is often responsive to a change in pharmacologic agent. It appears to be more common in the face of low serum ferritin levels.

PROGNOSIS

No long-term studies have been conducted on the course of RLS or PLMD in children or adolescents. It appears, however, that early-onset RLS that begins in childhood or adolescence has a more chronic and progressive course and is likely to be a lifelong disorder, although patients with milder disease may have long periods of remission. Cases of secondary RLS and PLMD generally remit without recurrence when the underlying condition is resolved.

Tips for Talking to Parents

- Explain what causes RLS and PLMD in simple terms, including the fact that these are biologically based disorders that are often chronic and lifelong. In particular, parents may have heard or read media claims that RLS is “not a real disorder” and may have questions about this statement.

- Review the common sensory and motor symptoms of RLS and PLMD, as well as the associated bedtime struggles or sleep-onset delay and sleep disruption.

- Explain common risk or exacerbating factors, such as iron-deficiency anemia, caffeine consumption, and medication use, including sedating over-the-counter antihistamines.

- Discuss the familial component, and encourage any parent or family member with similar symptoms to be evaluated.

- Review the daytime consequences, as parents may not attribute moodiness or ADHD symptoms to a sleep problem.

- Explain what to expect from the overnight sleep study, and review the results of the overnight sleep
• **Emphasize the importance of good sleep hygiene**, especially ensuring adequate sleep.

• **Discuss the risks and benefits of pharmacologic treatment options**, including oral iron supplementation and any medications, such as dopaminergic agonists, if these are being considered.

• **Encourage parents to utilize reliable resources for further information**, such as the RLS Foundation (www.rls.org) and the National Sleep Foundation (www.sleepfoundation.org) websites; the RLS Foundation has an excellent brochure on pediatric RLS and PLMD that can be ordered from the website.

See Appendix D12 for parent handout on RLS and Appendix D13 on PLMD.
Excessive daytime sleepiness (EDS) is defined as “the inability to stay awake and alert during the major waking episodes of the day, resulting in periods of irrepressible need for sleep or unintended lapses into drowsiness or sleep.” EDS may be differentiated from “fatigue” by the presence of this sleep propensity, although the distinction is not always readily apparent by history. Hypersomnia is a clinical term that is used to describe a group of disorders characterized by recurrent episodes of EDS, reduced baseline alertness, and/or prolonged nighttime sleep periods that interfere with normal daily functioning. It is important to recognize that there are many potential causes of EDS, which may be broadly grouped as “extrinsic” (e.g., secondary to insufficient and/or fragmented sleep) or “intrinsic” (e.g., resulting from an increased need for sleep). The most common causes of EDS in both adults and children are primary sleep disorders that result in inadequate or disrupted sleep (i.e., obstructive sleep apnea [OSA], insomnia, restless legs syndrome (RLS), and periodic limb movement disorder [PLMD]) and curtailment of sleep (e.g., behaviorally induced insufficient sleep). The “intrinsic” disorders of EDS discussed in this chapter, which are associated with hypersomnia of central nervous system (CNS) origin, are less common, but nonetheless important causes of significant sleepiness-related functional impairments in children and adolescents.

Narcolepsy

Narcolepsy is a chronic lifelong CNS disorder typically presenting in adolescence and early adulthood that is characterized by profound daytime sleepiness and resultant significant functional impairment. Other symptoms frequently associated with narcolepsy include cataplexy, hypnagogic or hypnopompic hallucinations, sleep paralysis, automatic behaviors, and disturbed nocturnal sleep. Narcolepsy may be further delineated as (1) narcolepsy with cataplexy (type 1; hypocretin (HCRT) deficiency syndrome); (2) narcolepsy without cataplexy (type 2); and (3) secondary narcolepsy due to medical conditions. The fundamental pathophysiology of narcolepsy has been shown to involve dysfunction of the HCRT/orexin neuropeptide system in the hypothalamus.

Centrally mediated hypersomnias include both chronic and persistent (e.g., narcolepsy, idiopathic hypersomnia [IH]) and episodic and recurrent (e.g., Kleine-Levin syndrome) sleep disorders; they may be further divided into primary versus secondary (i.e., due to neurologic or medical conditions or substances such as medications and illicit drugs). The most clinically significant of the central hypersomnias is narcolepsy, a chronic, lifelong sleep disorder that involves a fundamental disturbance in the regulation of REM sleep and wakefulness. The most prominent clinical manifestation of narcolepsy is profound EDS, characterized by both an increased baseline level of daytime drowsiness and by the repeated occurrence of sudden and unpredictable sleep episodes. These “sleep attacks” are often described as “irresistible” in that the individual is unable to stay awake despite considerable effort, and they occur even in the context of normally stimulating activities (e.g., during meals, in the middle of a conversation). The sleep episodes may occur many times throughout the day, are of relatively short duration (10-20 minutes), and are often described in adults as at least temporarily “refreshing.” Individuals with narcolepsy may also experience much briefer sleep episodes (“microsleeps”) of which they are unaware, resulting in “automatic” behaviors and memory lapses. In younger children in particular, the clinical manifestations of EDS may be quite different from those seen in adolescents and adults (see Clinical
Presentation and Symptoms below).

Other symptoms commonly associated with narcolepsy, including cataplexy, hypnagogic and hypnopompic hallucinations, and sleep paralysis, may be conceptualized as representing the “intrusion” of REM sleep features into the waking state. For example, cataplexy, defined as a sudden complete or partial loss of motor control with sparing of respiratory muscles, is related to the muscle atonia characteristic of REM sleep occurrence during wakefulness. Similarly, the vivid visual, auditory, and tactile sensory components of hypnagogic hallucinations are hypothesized to result from REM-related dream mentation experienced while awake. As noted above in regards to daytime sleepiness, all of the ancillary features of narcolepsy can have unique characteristics in children.

The typical onset of symptoms of narcolepsy is in adolescence and early adulthood, although symptoms may initially present in school-aged and even younger children. However, the early manifestations of narcolepsy are often ignored, misinterpreted, or misdiagnosed as other medical, neurological, and psychiatric conditions, and the appropriate diagnosis is commonly delayed for a number of years. It is estimated, for example, that as many as 200,000 people in the United States have narcolepsy, but fewer than one-quarter of those individuals have actually been diagnosed. Because timely behavioral and pharmacologic intervention may have a profound effect on quality of life for these patients, it is important for the primary care physician to be able to recognize and assess children and adolescents with narcolepsy and other central hypersomnias, and to appropriately refer them for further diagnostic evaluation and treatment.

EPIDEMIOLOGY

■ Prevalence: The prevalence of daytime sleepiness in school-aged children and adolescents has been estimated to be between 10% and 20%, but these figures are highly dependent upon the definition of EDS used. In contrast, the prevalence of narcolepsy is reported to be between 3 and 16 per 10,000, with the incidence of narcolepsy with cataplexy recently estimated at 0.74 cases per 100,000 person-years and 1.37 for narcolepsy without cataplexy. Narcolepsy without cataplexy is estimated to comprise half or less of all narcolepsy cases (10%-50%). Prevalence also appears to vary across various ethnic and racial groups, with a six-fold increase in prevalence, for example, in Japan compared to Europe and North America. While the prevalence of narcolepsy in childhood is not known, extrapolation from adult studies suggests that it is greater than 20 to 60 per 100,000 in Western countries.

■ Gender: There is equal preponderance in males and females.

■ Age: Symptoms typically develop after puberty, with most individuals first reporting symptoms of narcolepsy between the ages of 15 and 30 years. While studies have indicated that as many as one-third of adult patients report the onset of symptoms before age 15 years and about 15% before age 10 years, only about 4% of narcoleptics are diagnosed before the age of 15 years. However, the onset of narcolepsy has been increasingly reported in prepubertal children; whether this represents a true increase in incidence or heightened awareness (or both) is unclear. The average time elapsed between onset of symptoms and diagnosis is 10 to 15 years.

■ Family history: Overall, the prevalence of familial narcolepsy is low, and most cases are sporadic. However, the risk of developing narcolepsy with cataplexy in a first-degree relative of a narcoleptic patient is estimated at 1% to 2%; this represents an increase of 10- to 40-fold compared to the general population, suggesting greater genetic loading in these families. The prevalence of isolated narcolepsy symptoms in first-degree relatives of narcoleptic patients is 4% to 6%.

ETIOLOGY AND RISK FACTORS

Narcolepsy results from a decrease in levels of the neuropeptides HRCT-1 and -2. Both animal and human
studies have now strongly implicated a specific deficit in the hypothalamic orexin/HRCT neurotransmitter system in the genesis of narcolepsy with cataplexy. The orexin/HRCT system affects monoaminergic and cholinergic activity and is known to be involved not only with maintenance of alertness and the stabilization of sleep-wake states, but also with hunger and satiety, energy metabolism, emotional processes, and locomotion. The underlying pathogenesis of narcolepsy is believed to involve the loss of ~70,000 posterior hypothalamic neurons that produce the wake-promoting neuropeptide HCRT (orexin); cerebrospinal fluid (CSF) HCRT/orexin is reduced or undetectable in most (although not all) patients with narcolepsy associated with cataplexy, especially in those patients who are human leukocyte antigen (HLA) positive (see below). It has been postulated that autoimmune mechanisms, possibly triggered by viral infections or vaccines, in combination with a genetic predisposition and environmental factors, may be involved. It is believed that familial narcolepsy is related to mutations in the genes synthesizing peptides in the HRCT system or their receptors. Thus far, however, the search for polymorphisms in the HRCT (orexin) neuropeptide precursor (HCRT) gene has provided inconclusive results.

**Genetic factors.** An important predisposing genetic factor is a specific HLA, the HLA DQB1*0602. HLA testing also shows a strong association with narcolepsy; more than 90% of European and Caucasian narcoleptics with cataplexy test positive for HLA DQ antigens DQA1*0102 and DQB1*0602. The presence of an increased number of DQB1*0602 alleles also appears to be associated with more severe symptoms. Interestingly, the presence of the DQB1*0602 has also been shown to be associated with interindividual differences in sleep drive, sleep fragmentation, sleepiness, and fatigue in healthy populations. In children, it appears that about 60% to 70% of individuals with narcolepsy are HLA positive. This compares to the general population, of which it is estimated about one-quarter (12%-38%) are positive for the DQB1*0602 antigen. Therefore, because the vast majority of individuals with this antigen do not have narcolepsy, the presence of HLA positivity has low specificity. In addition, some 60% of patients with narcolepsy without cataplexy are DR2 negative, and less than 30% of monozygotic twins have been found to be concordant for HLA subtype. Thus, the presence of this antigen alone is not likely to be sufficient for the development of narcolepsy, and environmental factors are probably involved.

**Immunological diseases.** Studies in adults have examined the relationship between immunopathologic diseases and narcolepsy. In a recent study, 17% of patients diagnosed with narcolepsy had one or more associated autoimmune diseases, such as idiopathic thrombocytopenic purpura, multiple sclerosis, systemic lupus erythematosus, psoriasis, Crohn disease, and ulcerative colitis. Furthermore, cataplexy was significantly more severe in patients with both narcolepsy and immunopathological diseases. Narcolepsy has also been reported following streptococcal infection, presumably also linked to an autoimmune mechanism. Another recent study showed some evidence for alterations in the cytokine profile in the serum and CSF of adult patients with narcolepsy-catalepsy compared to controls, implicating a potential role of IL-4 and significant TH2/Th1 serum cytokine secretion imbalance in the pathophysiology of narcolepsy. Increased tumor necrosis factor (TNF)-α levels have also been reported in association with narcolepsy, suggesting chronic inflammation.

**Brain structures.** fMRI studies have suggested that narcolepsy is accompanied by changes in the emotional and reward-related function centers (e.g., amygdala, nucleus accumbens, striatum) in the brain. These fMRI findings are in line with structural neuroimaging which have shown abnormalities in brain structures such as the amygdala, nucleus accumbens, midbrain, thalamus, hippocampus, and frontotemporal cortical areas in narcolepsy patients, emphasizing the pervasive influence that this disorder has on brain structure and function.

**Vaccines.** One of the most important recent developments in the understanding of pediatric narcolepsy was the recognition that there was a significant increase in the diagnosis of narcolepsy with cataplexy in pediatric patients (reported first in Scandinavia and subsequently in the United Kingdom, France, and Canada) who received the ASO3 adjuvanted pandemic A/H1N1 influenza vaccine in 2009. In August 2010, the Swedish
Medical Product Agency and the Finnish National Institute for Health and Welfare (THL) first reported cases of narcolepsy as possible adverse events following vaccination against influenza A specifically with Pandemrix (GlaxoSmithKline). In children in Ireland, for example, narcolepsy incidence was 5.7 per 100,000 children/adolescents vaccinated with Pandemrix and 0.4 per 100,000 unvaccinated children/adolescents (relative risk: 13.9); the relative incidence in England was 9.9. The increased risk for a diagnosis of narcolepsy was greatest in individuals ≤20 years of age. For example, in France, H1N1 vaccination was associated with narcolepsy-cataplexy with an odds ratio of 6.5 in subjects aged <18 years and 4.7 (1.6-13.9) in those aged 18 and over. In contrast, there seems to have been no increased risk of other neurological and immune-related diseases in association with the Pandemrix vaccine. In China, there was a substantial increase in narcolepsy cases specifically in association with H1N2 infection; occurrence of narcolepsy in 2010 in some areas showed an approximate three-fold difference compared to baseline levels.

The proposed mechanism for postvaccination narcolepsy involves a combination of an environmental trigger (e.g., vaccine, infection) that causes or enhances an antibody-mediated autoimmune response with a preexisting genetic susceptibility. It is unclear whether this represents the development of de novo disease versus hastening the onset in individuals who would have eventually developed narcolepsy. It is also not clear whether the association between vaccination and narcolepsy was solely due to the adjuvanted vaccine.

Do Vaccines “Cause” Narcolepsy?

A dramatic increase in childhood narcolepsy with cataplexy cases was noted in 2009 to 2010 in association with a specific H1N2 vaccine, first in Scandinavia and then in several other countries. While the exact mechanism is uncertain, the association may have involved an interaction between genetic susceptibility and an environmental trigger, resulting in an autoimmune-mediated destruction of hypothalamic HCRT-secreting cells. However, it should be kept in mind that the vast majority of children vaccinated against H1N1, even with the Pandemrix vaccine, did not develop narcolepsy; for example, the vaccine-attributable risk of developing narcolepsy in Finland was 1:16,000 in vaccinated 4- to 19-year-olds.

While the overall clinical picture in children developing narcolepsy after vaccine exposure is similar, some differences in presentation are found. Postvaccine patients appear to be somewhat older, and there are more frequent reports of cataplexy in vaccinated than in unvaccinated patients (82% versus 55%) and excessive weight gain (55% versus 20%). Facial hypotonia and tongue protrusion are also seen more frequently in vaccinated children.

Narcolepsy Without Cataplexy (Narcolepsy Type 2)

It should be noted that while the term narcolepsy often refers to narcolepsy with cataplexy, narcolepsy without cataplexy (narcolepsy type 2) appears to be a relatively distinct entity, both from the standpoint of etiology and risk factors, as well as in regards to therapeutic management. Narcolepsy type 2 is considered one of the syndromes with hypersomnolence unexplained by HRCT abnormalities (CSF HRCT levels are by definition >110 pg/mL). Although head trauma and viral illnesses have been reported to precede the onset of narcolepsy type 2 symptoms, the relationship with genetic and environmental factors is poorly understood. Narcolepsy without cataplexy is thought to represent 15% to 36% of adult patients with narcolepsy. The reported prevalence in children varies greatly (0%-100%) depending upon factors such as the target population, age, and duration of follow-up. The new ICSD III diagnostic criteria for narcolepsy type 2 (see Table 17.3) also emphasize that the patient should be reclassified as type 1 upon the
Other causes. Hypersomnias of central origin in general may also result from a variety of different processes involving injury to the CNS, including medical disorders (infection, metabolic disorders), neurologic disorders (head trauma and other causes of increased intracranial pressure), and any number of medications and illicit substances that result in significant daytime sleepiness and reduced alertness. Although the majority of cases of narcolepsy are considered idiopathic, “secondary” narcolepsy with cataplexy may also result from CNS insults. These include inherited metabolic and genetic disorders involving abnormalities of the CNS, such as Niemann-Pick disease type C, and autosomal dominant cerebellar ataxia with deafness. Secondary narcolepsy may also be associated with other disorders, such as Prader-Willi syndrome and myotonic dystrophy type 1; CNS trauma (e.g., following closed head injury); brain tumors, such as astrocytomas and craniopharyngiomas (particularly in the third ventricle, posterior thalamic, and brainstem regions) as well as other malignancies such as neuroblastoma; and various vascular and infectious insults to the hypothalamus. The age of onset in these secondary (or “symptomatic”) narcolepsy cases is often lower (i.e., school age or younger), and clinical evidence of signs and symptoms of the primary medical or neurological condition is typically readily apparent.

PRESENTATION AND SYMPTOMS (TABLE 17.1)

The classic “tetrad” of symptoms for narcolepsy includes EDS, cataplexy, hypnagogic and hypnopompic hallucinations, and sleep paralysis. It is estimated that about half of all adult patients with narcolepsy have all four of the primary symptoms of narcolepsy. However, pediatric case series suggest that the relative prevalence of symptoms other than EDS may be lower at the time of diagnosis in children compared to adults, especially in younger children who may not be able to describe these symptoms accurately.

- EDS: A mandatory component for the diagnosis of narcolepsy, EDS is most often the initial presenting complaint. The overwhelming urge to fall asleep is more pronounced under conditions of decreased activity and low-level environmental stimulation, such as watching a movie or listening to a boring lecture, but also occurs in other situations, such as during a meal or while talking on the phone. This excessive sleepiness occurs despite adequate nighttime sleep duration, and periods of sleep only temporarily alleviate the sleepiness. Very brief (several seconds) “sleep attacks” may also occur; in children, these have been reported to last up to 60 to 90 minutes and may be accompanied by “sleep drunkenness” with extreme confusion, irritability, and sometimes aggressive behavior upon forced awakening. The individual with narcolepsy is usually unaware of these “microsleeps” and may “stare off,” appear unresponsive, or continue to engage in an ongoing activity (automatic behavior); this may be interpreted by observers as “daydreaming” and inattentiveness related to attention deficit hyperactivity disorder (ADHD), or a manifestation of absence (“petit mal”) seizures. Younger children in particular may manifest an outward behavioral response to an internal sensation of drowsiness that is characterized by increased motoric activity and disinhibition; this too may be interpreted as hyperactivity or impulsivity associated with ADHD. In younger children, in particular, the onset of sleepiness is often abrupt and profound, although it may improve over time, and may manifest as extended nighttime sleep need and/or resumption of daytime naps following discontinuation of regular napping.

TABLE 17.1. Common Symptoms of Narcolepsy
Nocturnal symptoms
-- Disrupted or fragmented sleep
-- Hypnagogic and hypnopompic (sleep onset and offset) hallucinations
-- Sleep paralysis
EDS
-- Increased baseline sleepiness, prolonged nocturnal sleep, increased napping, or resumption of napping in younger children
-- Sudden, unpredictable, “irresistible” sleep episodes or “attacks”
-- Falling asleep in school, during sedentary activities, and especially if during active pursuits (e.g., in the midst of a conversation)
Other daytime symptoms
-- Cataplexy (sudden partial or complete loss of muscle tone with preservation of consciousness)
-- Inattentiveness, poor concentration, distractibility
-- Academic problems
-- Mood dysregulation
-- Automatic behaviors

**Cataplexy**: Cataplexy is considered to be the most distinctive clinical feature of narcolepsy and is virtually pathognomonic for the disease; for example, it is extremely rare outside of the context of narcolepsy. Cataplexy occurs in 60% to 100% of adult patients, and in at least one study, in as many as 80% of pediatric cases. Cataplexy is rarely the first symptom of narcolepsy, but it often develops within the first year of the onset of EDS. It is described as an abrupt, bilateral, partial, or complete loss of muscle tone, classically triggered by an intense positive emotion (e.g., laughter, surprise). The cataplectic attacks are typically brief (seconds to minutes), and fully reversible, with complete recovery of normal tone when the episode ends. The loss of muscle tone spares the diaphragm and ocular muscles, and can range from subtle, localized sagging of the face, eyelids, or jaw, to head nodding or knee buckling, to complete whole body collapse; the most common muscles affected in partial attacks are knees, head, jaw, and neck. More subtle cataplectic episodes may involve a subjective sense of weakness or unsteadiness without obvious external behavioral manifestations; eyelid ptosis, slurred speech, and gait disturbances may be observed. During the episode, the individual maintains complete consciousness and awareness of his or her surroundings; memory for the event is not impaired. Although laughter is the most common precipitant, other emotions such as anger, sadness, or even the anticipation or recollection of an emotion, as well as vigorous physical exercise, can also trigger cataplexy. In some children, there is no identifiable trigger for the attacks. The frequency of episodes can range from occurring multiple times per day to a few times per year.

Cataplectic episodes may be interpreted as “clumsiness,” seizures (“drop attacks”), or even psychosomatic phenomena (e.g., pseudoseizures, conversion reaction). In some children, closer to onset, cataplexy may co-occur with a complex movement disorder characterized by prominent facial involvement that appears to be a feature unique to childhood narcolepsy (“cataplectic facies”); this is described as a semipermanent state of facial (jaw/eyelid) weakness with superimposed occasional exacerbations. It often causes grimacing or jaw opening with tongue thrusting/protrusion. Young children may also have intermittent facial grimacing, persistent hypotonia, dyskinetic or “tic-like” movements, or dystonic movements that increase during emotional stimulation and eventually evolve into the more discrete cataplexy “attacks” seen in adults. (**Figure 17.1**) **Hypnagogic and hypnopompic hallucinations**: These hallucinations involve vivid visual, auditory, and
sometimes tactile sensory experiences, which may be described by children as "scary dreams." These occur during transitions between sleep and wakefulness, primarily at sleep onset (hypnagogic) and sleep offset (hypnopompic). They are reported by approximately 50% to 70% of adult patients with narcolepsy. They may be primarily auditory (hearing your name called), visual (seeing a stranger or animal in the room), or somesthetic (“out of body” experience). They are frequently described as frightening or threatening. These hallucinations may be accompanied by sleep paralysis or occur independently, and may also occur during daytime naps. However, it should be noted that sleep related hallucinations are also experienced by individuals without narcolepsy (25% and 18% of the general adult population report hypnagogic and hypnopompic hallucinations, respectively), especially in relation to chronic sleep loss. Thus, they are not considered pathognomonic for the disorder.

- **Sleep paralysis:** The inability to move or speak for a few seconds or minutes at sleep onset or offset. The episodes may be accompanied by eye-fluttering, moaning, autonomic symptoms (e.g., sweating), or a subjective sensation of struggling to move or breathe; stress or sleep deprivation may exacerbate the episodes. The paralysis ends spontaneously or after mild sensory stimulation (e.g., being touched or called). Sleep paralysis occurs in 40% to 80% of adults with narcolepsy and may also accompany hypnagogic hallucinations. This combination of frightening sensory phenomenon coupled with the inability to move or speak is understandably experienced as very unpleasant. As with hypnagogic hallucinations, sleep paralysis occurs in nonnarcoleptic individuals and, therefore, does not necessarily indicate the presence of narcolepsy.

Narcolepsy Without Cataplexy: Diagnostic Challenges

While narcolepsy without cataplexy (type 2) is overall more common than narcolepsy with cataplexy, at least in adults, the diagnosis is often more challenging for a number of reasons. Patients with narcolepsy without cataplexy must meet the same multiple sleep latency test (MSLT) criteria as those with cataplexy (in contrast to Idiopathic Hypersomnia (IH)), but by definition lack the most clear-cut symptom of narcolepsy. Patients with narcolepsy type 2 also have an overall increased risk for associated sleep disorders, such as OSA, periodic leg movements, and RLS, although to a lesser extent than patients with cataplexy.

Additional symptoms of narcolepsy:

- **Nocturnal sleep disturbances**: Sleep disruption and fragmentation are very common in narcolepsy and occur in up to 90% of adult patients. Sleep disturbances may also include abnormal REM sleep (i.e., preservation of muscle tone and increased muscle twitching in REM). In particular, the presence of muscle movement in REM sleep may result in the individual "acting out" dreams, a clinical entity known as REM behavior disorder (RBD), which may result in severe injury to the sleeper or bed partner.

- **Automatic behaviors**: These involve a semi-purposeful activity for which the individual has no memory, and are more frequent with increasing levels of sleepiness. These activities may last up to 30 minutes, and usually involve a repetitive or monotonous behavior, such as uttering words out of context or writing a page of nonsense in the midst of doing homework. These episodes may be misdiagnosed as complex partial seizures.

**Diagnostic Criteria**

See Tables 17.2 and 17.3.

**TABLE 17.2. Diagnostic Criteria: Narcolepsy Type 1**

A. The patient has daily periods of irrepressible need to sleep or daytime lapses into sleep occurring for at least 3 mo.

B. The presence of one or both of the following:
   1. Cataplexy and a MSL of 8 min and two or more SOREMPs on an MSLT performed according to standard techniques. A SOREMP (within 15 min of sleep onset) on the preceding nocturnal polysomnogram may replace one of the SOREMPs on the MSLT.
   2. CSF HRCT-1 concentration, measured by immunoreactivity, is either ≤110 pg/mL or <⅓ of mean values obtained in normal subjects with the same standardized assay.

*ICD-9-CM code: 347.01; ICD-10-CM code: G47.411

**Notes:**
1. In young children, narcolepsy may sometimes present as excessively long night sleep or as resumption of previously discontinued daytime napping.
2. If narcolepsy type 1 is strongly suspected clinically but the MSLT criteria of B1 are not met, a possible strategy is to repeat the MSLT.

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TABLE 17.3. Diagnostic Criteria: Narcolepsy Type 2

A. The patient has daily periods of irrepressible need to sleep or daytime lapses into sleep occurring for at least 3 mo
B. A MSL of ≤8 min and two or more SOREMPs are found on a MSLT performed according to standard techniques. A SOREM (within 15 min of sleep onset) on the preceding nocturnal polysomnogram may replace one of the SOREMPs on the MSLT.
C. Cataplexy is absent.
D. Either CSF HRCT-1 concentration has not been measured or CSF HRCT-1 concentration measured by immunoreactivity is either >110 pg/mL or >⅓ of mean values obtained in normal subjects with the same standardized assay.
E. The hypersomnolence and/or MSLT findings are not better explained by other causes such as insufficient sleep, OSA, delayed sleep phase disorder, or the effect of medication or substances or their withdrawal.

ICD-9-CM code: 347.2; ICD-10-CM code: G47.419

Notes:
1. If cataplexy develops later, then the disorder should be reclassified as narcolepsy type 1.
2. If the CSF HRCT-1 concentration is tested at a later stage and found to be either ≤110 pg/ml or <⅓ of mean values obtained in normal subjects with the same assay, then the disorder should be reclassified as narcolepsy type 1.

Associated Features

- **Mental health issues**: Many children and adolescents with narcolepsy experience significant psychological distress associated with functional impairment related to EDS and/or cataplexy, which may be exacerbated by the delay in diagnosis. They are more likely to have behavioral disturbances, depression, problematic peer relationships, and decreased overall quality of life. In a recent study of Swedish children developing narcolepsy after an H1N1 vaccination, 43% of patients had at least one psychiatric comorbid condition, with 29% having ADHD inattentive type, 20% with major depression, 10% with generalized anxiety disorder, and 7% with oppositional defiant disorder (ODD); temper tantrums were reported in 94%. High levels of depressive symptoms largely associated with fatigue have been reported to affect some 25% of children with narcolepsy, with girls older than 10 years of age being especially vulnerable.

- **Impaired academic performance**: While most studies have found that children with narcolepsy do not have significant impairments in overall cognitive functioning (i.e., IQ is within the normal range), specific learning and academic deficits have been identified. Due to the propensity for falling asleep in school and...
the attentional difficulties associated with narcolepsy despite having a normal IQ, childhood with narcolepsy with cataplexy represents a risk factor for subtle and heterogeneous cognitive impairments, potentially resulting in academic failure. In the Swedish study cited above, children with narcolepsy were found to have decreased verbal comprehension and working memory on neuropsychological tests. In addition, children and adolescents with narcolepsy are often labeled as lazy, inattentive, and difficult.

- **Decreased quality of life.** Narcolepsy seriously impacts health-related quality of life in terms of vitality, physical well-being, social relationships, and leisure activities, especially in adolescents. Depression seems to be a major contributor to the decrease in overall quality of life.

- **Safety concerns.** Children with EDS from any cause may pose significant safety risks. For example, children with narcolepsy and IH are twice as likely to be struck by a virtual vehicle in a virtual pedestrian environment compared to healthy controls, largely due to impaired decision making and "inattentional blindness"; that is, they recognized but did not process conditions that were unsafe. In patients with cataplexy, the risk for accidental injury is heightened, as there is little, if any, opportunity to anticipate or prevent a fall associated with a cataplectic attack. In addition, by-standers may interpret cataplexy events as seizure activity or other catastrophic events and intervene inappropriately. In order to avoid these types of situations, it is recommended that all patients with narcolepsy, particularly those with cataplexy, wear a "Medic-Alert" type device at all times. In addition, a management plan for cataplexy in the school setting is mandatory.

- **Driving.** A very important safety concern regards adolescent drivers with narcolepsy. All states require disclosure of medical conditions that could impact driving safety when applying for a driver's license. It is imperative that the condition of narcolepsy (with or without cataplexy) be disclosed. Most adolescents with narcolepsy can be medically cleared to drive if their symptoms are appropriately controlled, which usually involves good adherence to medication treatment. A Maintenance of Wakefulness test (MWT) (see below) may be indicated in order to objectively demonstrate fitness to drive.

- **Athletic activities.** Another issue particularly relevant to adolescents with narcolepsy is related to participation in athletic activities and the use of controlled substances such as psychostimulants for the treatment of narcolepsy, especially at the high school and college level. According to the NCAA, in order for a student-athlete to be granted a medical exception for the use of a banned substance, the student-athlete must have declared the use of the substance to his or her athletics administrator responsible for keeping medical records, present documentation of the diagnosis of the condition, and provide documentation from the prescribing physician explaining the course of treatment and the current prescription.

- **Comorbid sleep disorders:** OSA and periodic limb movements (PLMs) appear to be very common in children with narcolepsy. In one referral population clinical series, 85% of children had evidence of sleep-disordered breathing and 25% had PLMs on polysomnography (PSG). Children with narcolepsy and comorbid obesity are at increased risk for OSA.

- **Migraine headaches:** The prevalence of migraines is reportedly increased in individuals with narcolepsy.

- **Overweight and obesity:** A number of studies, as well as clinical observation, suggest that children with narcolepsy, especially younger children, are more likely to be overweight or obese. They also often have sudden and dramatic weight gain at the onset of symptoms, especially in younger children, although it may plateau over time. Studies have suggested that obesity is found in 60% to 74% of children with narcolepsy, whether with or without cataplexy. These children are also more likely to engage in abnormal...
eating behaviors such as bingeing. Associated hypothalamic dysfunction and the HRCT/orxin system's effect on hunger and satiety, as well as a decrease in physical activity related to sleepiness, may all play a role in increasing weight gain. In addition to a negative impact on energy level, mood, self-esteem, and general health, children with obesity and narcolepsy have a lower sleep efficiency (time asleep/time in bed), as well as a higher apnea hypopnea index and respiratory arousal index (and higher risk for OSA), potentially further compounding daytime sleepiness and neurocognitive deficits.

- **Precocious puberty:** Precocious puberty occurs at a significantly higher rate in children with narcolepsy; one study found that 17% of children with narcolepsy met criteria for precocious puberty and 41% had evidence of isolated signs of accelerated pubertal development (e.g., thelarche, pubic hair, advanced bone age). The onset of narcolepsy symptoms, precocious puberty, and obesity appear to be temporally related, especially in younger children. The mechanism is not known, but suggests extended hypothalamic dysfunction.

**The Great Masquerader**

The symptoms of narcolepsy (e.g., EDS, cataplexy) are frequently misdiagnosed as psychiatric or behavioral disorders, including ADHD, depression, conversion reactions, and even psychosis. In addition, patients with central hypersomnia and narcolepsy are at increased risk for emotional, attentional, and academic problems as a result of their EDS, particularly if the diagnosis is significantly delayed.

**Evaluation**

- **Medical history:** A thorough medical history should include evaluation for other possible causes of EDS, including sleep disorders such as OSA, RLS, and PLMD; neurologic conditions; psychiatric disorders, such as depression and substance use; and prescription and nonprescription sedating medications. Because narcolepsy may be secondary to underlying medical conditions (e.g., head injury, CNS tumors, demyelinating disorders), a thorough history should include screening for these disorders. It should be noted, however, that the most common cause of EDS in the general population, particularly in adolescents, is chronic sleep restriction related to environmental and lifestyle factors.

- **Developmental and school history:** The history is generally normal, although children with secondary narcolepsy associated with underlying neurologic disorders such as Niemann-Pick disease type C will have obvious developmental delays. There may be a history suggestive of attentional problems or a diagnosis of “ADHD”; older children and adolescents with narcolepsy commonly have a history of significant academic concerns.

- **Family history:** Although still a rare occurrence, there is an increased risk of developing narcolepsy with cataplexy in first-degree relative of narcoleptic patients; compared to the general population, the prevalence is estimated at 1% to 2%. It is estimated that up to 40% of individuals may have a family member with a history of EDS.

- **Behavioral assessment:** An assessment may indicate attention problems, mood issues, and behavioral concerns, such as hyperactivity and poor impulse control. Because of the functional impairment associated with EDS, older children and adolescents with undiagnosed narcolepsy may have significant social problems as well.
- Physical examination: Assessment of anthropometric parameters may reveal increased body mass index (BMI). In the majority of cases, the physical examination is completely normal, although EDS in the office setting (i.e., falling asleep during the clinical interview) is frequently noted. Patients with narcolepsy may appear to have a flat affect, may seem confused or cognitively slowed, and speech may be slurred or difficult to understand. However, an abnormal neurologic examination suggests the possibility of secondary narcolepsy, and necessitates appropriate additional diagnostic evaluation (e.g., neuroimaging).

- Diagnostic tests:
  - Sleep diary, which may be helpful for documenting EDS and napping despite adequate duration of nocturnal sleep, is recommended.
  - Sleepiness scales such as the Pediatric Daytime Sleepiness Scale in children and adolescents, the Cleveland Adolescent Sleepiness Scale, and the Modified Epworth Sleepiness Scale in older children and adolescents may assist in delineating the severity of EDS.
  - Overnight sleep study (PSG) and MSLT are strongly recommended components of the evaluation of a patient with profound unexplained daytime sleepiness or suspected narcolepsy. The purpose of the overnight PSG is to evaluate for primary sleep disorders, such as OSA, that may cause EDS. In addition, the presence of both a shortened sleep latency and shortened REM latency as well as fragmented nocturnal sleep helps to support the diagnosis of narcolepsy. PLMs are also commonly seen on PSG in patients with narcolepsy.

The MSLT provides an essential and objective quantification of daytime sleepiness and assesses the presence of sleep-onset REM periods (SOREMPs), which is a cardinal feature of narcolepsy. It comprises a series of five scheduled naps of 20 minutes' duration separated by 2-hour intervals during which the patient should be kept awake. The MSLT takes places on the day following an overnight PSG. Prior to PSG, the patient should be withdrawn, if possible, from any medications that may affect the CNS, with adequate time to prevent rebound changes in sleep architecture (in the case of REM-suppressing antidepressants, e.g., for at least 2 weeks). Drug testing on the morning of the MSLT may be warranted if there is a suspicion of illicit drug use. The patient should also be instructed to obtain sufficient sleep for age for at least 2 weeks before the MSLT, ideally documented with a sleep diary and actigraphy.

- MSLT diagnostic criteria (adult and adolescents):
  - Mean sleep latency (MSL) ≤8 minutes
  - Two or more SOREMPs during the MSLT performed according to standard techniques. A SOREMP (i.e., within 15 minutes of sleep onset) on the preceding night PSG may replace one of the SOREMPs on the MSLT.

Sleep latency is defined as the time elapsed between lights out and the onset of stage 1 sleep; sleep latency is averaged over the five naps to yield the MSL. In adults, an MSL of less than 5 minutes and between 5 and 10 minutes is considered consistent with “severe” and “moderate” hypersomnia, respectively. Although a MSL of less than 8 minutes is highly suggestive of narcolepsy, it is estimated that up to 30% of the normal adult population would also meet this criteria, and thus this finding is nonspecific. The prior night PSG is also essential in interpreting the MSLT results, as a patient with OSA or PLMs may also have significant daytime sleepiness. In addition, there are often findings on the nocturnal PSG that support the diagnosis of narcolepsy; these include the presence of an “SOREMP” (defined as REM sleep occurring within 15 minutes of lights out), which has been shown to have an overall positive predictive value of 88.5% for the diagnosis of narcolepsy with cataplexy in the pediatric population. More than two SOREMPs on the MSLT are generally considered highly specific for the
data suggest that up to 15% of the general population may have this finding. The combination of MSL of less than 5 minutes and more than 2 SOREMPs is estimated in adults to have a 70% sensitivity and 96% specificity for narcolepsy.

In children and adolescents, conducting and interpreting the results of the MSLT may be particularly challenging. Younger children may not cooperate with the lengthy daytime procedure required, especially immediately following an overnight PSG. Children will also often exhibit a "last nap effect" in which the anticipation of going home increases arousal and results in spuriously prolonged sleep onset. Because children have an increased sleep need compared to adults, the requirement for at least 6 hours of sleep before the MSLT may be inadequate. In adolescents, use of substances such as caffeine, nicotine, and drugs (such as stimulants and marijuana) may affect the MSLT results. A recent retrospective study in pediatric patients found that 10% of urine drug screens conducted on the morning before the MSLT were positive. In addition, 50% of the patients screening negative for tetrahydrocannabinol (THC) had nonpathologic MSLT results compared to only 29% of those with a positive THC drug screen. Another study that failed to find positive screens for drugs of abuse in 210 pediatric patients undergoing MSLT testing found objective evidence for caffeine exposure in 32% of tested children, underscoring the importance of considering drug screening in conducting and interpreting MSLT results.

Finally, it has been suggested that MSL value thresholds that are significantly higher than those used as the criteria for diagnosis in adults may still be pathologic in children. Normative data in prepubertal children suggest that a sleep-onset latency of 15 minutes or less may be indicative of significant daytime sleepiness. Some have recommended use of a MSL of <10 minutes be considered abnormal in prepubertal children. However, clinical data from pediatric sleep centers suggest that the MSL in children with narcolepsy is similar to that in adults, for example, less than 8 minutes. It should be noted that the MSLT itself has not been validated in children less than 8 years old. Interpretation of MSLT results in children should take these factors into consideration, and it should be noted that it might take several polysomnographic studies over a period of 6 months to several years to make a definitive diagnosis.

- **Maintenance of Wakefulness Test (MWT)**, in contrast to the MSLT, measures a patient's ability to stay awake under sleep-promoting circumstances (i.e., lying in a quiet darkened room). The test consists of four 20-minute trials conducted 4 times at 2-hour intervals commencing 2 hours after awakening from a night of sleep. A cutoff of a MSL of <8 minutes for inadequate control of hypersomnia and >20 minutes to indicate adequate control of EDS is generally used, although no norms exist for the pediatric population. This test is most often used to more objectively assess medication efficacy and to document an adequate level of alertness for driving and performance of occupational tasks.

- **Neuroimaging (magnetic resonance imaging)** is not routinely indicated but should be strongly considered if the EDS has a sudden onset, occurs in the presence of neurological symptoms and/or an abnormal neurologic examination, or if there is a history of recent head injury.

- **HLA testing** is not mandatory for the diagnosis of narcolepsy, but may be helpful in some cases. Although most patients with narcolepsy and cataplexy are HLA positive (75%-90%), it should be kept in mind that some 25% of normal individuals are also positive for the DQ antigens. Children with both DR15 and DQB1*0602 alleles present are more likely to have severe EDS and cataplexy.

- **CSF HRCT/orexin levels** are generally very low or nondetectable in patients with narcolepsy with cataplexy. Narcolepsy diagnosis can be confirmed by HRCT-1 (orexin-A) measurement in the CSF. This test has a high sensitivity and specificity in patients with typical cataplexy. Interestingly, these low levels may occur after the
appearance of EDS but before the onset of cataplexy. Although not required for the diagnosis, and at the current time not widely available, CSF levels may be helpful in select cases in which a confirmatory MSLT is difficult to conduct or interpret. This testing may become a more standard part of the evaluation in the future.

DIFFERENTIAL DIAGNOSIS (TABLE 17.4)

**Insufficient sleep** is the primary cause of EDS in children and adolescents. Short sleep latencies and SOREMPs on the MSLT can be manifestations of chronic sleep restriction and/or delayed sleep phase syndrome (see Chapter 18).

- **Idiopathic Hypersomnia (IH)** is a chronic disorder characterized by significant daytime sleepiness that shares many common features with narcolepsy. All symptoms may occur in both disorders, with the important exceptions of cataplexy and significant nocturnal sleep disruption being unique to narcolepsy. Patients with IH also frequently experience “sleep drunkenness” (extreme and prolonged confusion after awakening), and naps are typically described as “unrefreshing.” Although the MSL in IH is typically less than 8 minutes, fewer than two SOREMPs are present. IH is no longer classified as polysymptomatic (or “primary”) or monosymptomatic IH. Diagnostic criteria for IH are found in Table 17.5. The prevalence of IH is estimated to be 10% to 15% of sleep center patients evaluated for hypersomnia, and is rarely seen prior to adolescence; occasional familial clustering is observed. In select cases, IH may follow a viral illness, such as Guillain-Barré, hepatitis, and Epstein Barr. Head injury can also result in IH. Treatment for the associated EDS is similar to that for EDS in narcolepsy (e.g., modafinil, psychostimulants), although the daytime sleepiness associated with IH compared to narcolepsy is often even more refractory to treatment in IH.

- **Kleine-Levin syndrome** is a rare (estimated 1-2 cases/million) sleep disorder characterized by episodic, recurrent, prolonged bouts of hypersomnia in which the individual sleeps for as many as 16 to 18 out of 24 hours for several days up to 2 to 3 months at a time (median 10 days) that occur several times a year. The syndrome also involves cognitive, perceptual, emotional, and behavioral disturbances, including deficits in working memory and language, derealization (altered perception of the environment), hallucinations and delusions, depression and anxiety, hyperphagia with binge eating, and hypersexuality. Between periods of hypersomnolence, the patient is normal and has normal sleep patterns. Kleine-Levin is more common in males (4:1 ratio), and the usual onset is during adolescence. The syndrome is thought to be related to underlying hypothalamic dysfunction, which may be associated with structural brain lesions or CNS viral illness, but is most often idiopathic in nature; HLA typing is negative. The usual course generally involves gradual attenuation of frequency and duration of hypersomnia episodes with eventual complete resolution of symptoms over time; the mean duration is 14 years. Treatment with stimulants, modafinil, amantadine, and mood stabilizers, including lithium, has been tried with varying success. Diagnostic criteria for Kleine-Levin syndrome are presented in Table 17.6.

<table>
<thead>
<tr>
<th>TABLE 17.4. Differential Diagnosis of Hypersomnia in Children and Adolescents</th>
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<tbody>
<tr>
<td>○ Chronic insufficient sleep</td>
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<tr>
<td>○ Prolonged sleep need (&quot;long sleeper&quot;)</td>
</tr>
<tr>
<td>○ Primary sleep disorders (OSA syndrome, RLS/PLMD)</td>
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</tbody>
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Circadian rhythm disorders
Psychiatric disorders
Conversion disorder
Drug and toxin effects
Substance abuse
Nocturnal seizures
Medical conditions disrupting sleep; for example, asthma, atopic dermatitis, chronic pain
Brain tumors
Postencephalitis or post-viral
Postconcussion syndrome
Kleine-Levin syndrome
Idiopathic Hypersomnia (IH)
Narcolepsy types 1 and 2

### TABLE 17.5. Diagnostic Criteria: Idiopathic Hypersomnia (IH)

A. The patient has daily periods of irrepressible need to sleep or daytime lapses into sleep occurring for at least 3 mo.
B. Cataplexy is absent.
C. An MSLT performed according to standard techniques shows fewer than two SOREMPs or no SOREMPs if the REM latency on the preceding polysomnogram was less than or equal to 15 min.
D. The presence of at least one of the following:
   1. The MSLT shows a MSL of ≤8 min.
   2. Total 24-h sleep time is ≥660 min (typically 12-14 h) on 24-h polysomnographic monitoring (performed after correction of chronic sleep deprivation), or by write actigraphy in association with a sleep log (averaged over at least 7 d with unrestricted sleep).
E. Insufficient sleep syndrome is ruled out (if deemed necessary, by lack of improvement of sleepiness after an adequate trial of increased nocturnal time in bed, preferably confirmed by at least a wk of wrist actigraphy).
F. The hypersomnolence and/or MSLT findings are not better explained by another sleep disorder, other medical or psychiatric disorder, or use of drugs of medications.

*ICD-9-CM code: 327.11; ICD-10-CM code: G47.11

**Notes:**
1. Severe and prolonged sleep inertia, known as sleep drunkenness (defined as prolonged difficulty waking up with repeated returns to sleep, irritability, automatic behavior, and confusion), and/or long (>1 h) unrefreshing naps are additional supportive clinical features.
2. A high sleep efficiency (≥90%) on the preceding polysomnogram is a supportive finding (as long as sleep insufficiency is ruled out).
3. The total 24-h sleep time required for diagnosis may need to be adapted to account for normal changes in sleep time associated with stages of development in children and adolescents as well as for variability across cultures in all age groups.
4. Occasionally, patients fulfilling other criteria may have an MSLT mean sleep latency longer than 8 min and total 24-h sleep time shorter than 660 min. Clinical judgment should be used in
deciding if these patients should be considered to have IH. Great caution should be exercised
to exclude other conditions that might mimic the disorder. A repeat MSLT at a later date is
advisable if the clinical suspicion for IH remains high.

American Academy of Sleep Medicine. *International classification of sleep disorders: diagnostic

- **Menstrual-related Kleine-Levin syndrome** (previously known as menstrual-related periodic
hypersomnia) is another extremely rare cause of episodic hypersomnia; bouts of EDS are cyclical,
exclusively associated with menstruation, and are generally manifested shortly after menarche. The
bouts may be accompanied by compulsive eating, sexual disinhibition, and depression.

- **Psychiatric disorders and depression** should be considered in children and adolescents who present
with significant sleepiness and associated neurobehavioral symptoms. Conversion reactions may mimic
cataplectic attacks. However, it should be noted that the same pharmacologic agents that are used for
particular psychiatric conditions (e.g., stimulants for ADHD, tricyclic antidepressants (TCAs) for
depression) also improve symptoms of narcolepsy (e.g., EDS and cataplexy). Therefore, treatment
response to these agents should not be used to differentiate psychiatric disorders from narcolepsy.

- **Other neurologic causes** of sleepiness can result in daytime sleepiness, such as posttraumatic
hypersomnia, medications, and alcohol or drug use.

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**TABLE 17.6. Diagnostic Criteria: Kleine-Levin Syndrome**

| A. | The patient experiences at least two recurrent episodes of excessive sleepiness and sleep
duration, each persisting for 2 d to 5 wk. |
| B. | Episodes recur usually more than once a year and at least once every 18 mo. |
| C. | The patient has normal alertness, cognitive function, behavior, and mood between episodes. |
| D. | The patient must demonstrate at least one of the following during the episodes: |
|   | 1. Cognitive dysfunction |
|   | 2. Altered perception |
|   | 3. Eating disorder (anorexia or hyperphagia) |
|   | 4. Disinhibited behavior (such as hypersexuality) |
| E. | The hypersomnolence and related symptoms are not better explained by another sleep disorder,
other medical, neurologic, or psychiatric disorder (especially bipolar disorder), or use of drugs or
medications. |

*ICD-9-CM code: 327.13; ICD-10-CM code: G47.13

American Academy of Sleep Medicine. *International classification of sleep disorders: diagnostic
When to Refer
Children or adolescents with suspected narcolepsy should be referred to a sleep specialist or pediatric neurologist for diagnosis and management. Any pharmacologic treatment should be done in consultation with one of these specialists.

Treatment
Currently, there is no cure for narcolepsy but symptoms can usually be controlled so that a child or adolescent with narcolepsy can lead a normal life. An individualized treatment plan usually involves education, behavioral changes, and medication.

- **Patient and family education:** Narcolepsy can be a devastating disorder without appropriate education. Daytime sleepiness may be mistaken for laziness, boredom, or lack of ability. The experiences of cataplexy and dreaming during wakefulness may be wrongly interpreted as a psychiatric problem. Education should not only include all family members, but also teachers, coaches, and friends. In particular, school officials should be notified that accommodations at school may be necessary (e.g., scheduled naps, modified homework assignments to ensure adequate sleep duration); children with narcolepsy are eligible for 504 accommodation plans or individualized education plans (IEPs).

- **Healthy sleep habits.** Positive sleep habits are essential for children and adolescents with narcolepsy (see Appendices C1 and C8), as is obtaining adequate nighttime sleep.

- **Napping:** Regularly scheduled short (15-minute) naps once or twice a day can often help control the daytime sleepiness, although they are seldom sufficient as a primary therapy.

- **Behavioral changes:** Lifestyle changes can provide substantial improvement of symptoms.
  - A strict sleep-wake schedule that ensures adequate sleep is essential.
  - Increased physical activity can be helpful.

- **Close supervision of activities that can be dangerous,** such as driving, swimming, or cooking, is essential. All adolescents with narcolepsy must be well controlled prior to receiving permission to drive.

- **Weight management:** In particular, targeting abnormal eating behaviors and encouraging physical activity are important in children with elevated BMI.

- **Treatment of comorbid sleep disorders:** Appropriate interventions, especially for OSA (e.g., adenotonsillectomy) and PLMs (e.g., iron supplementation), may be helpful in reducing (although obviously not eliminating) sleepiness and improving daytime function in patients with narcolepsy.

- **Medications:** Medications are often prescribed to control the EDS (see Table 17.7). The goal should be to allow the fullest possible return of normal functioning in school, at home, and in social situations. Medication may also be used to control the REM-associated phenomena, such as cataplexy, hypnagogic hallucinations, and sleep paralysis, when these are clinically significant and affect the patient's quality of life. Pharmacologic agents are often used in combination to treat the individual's symptom constellation. As there is currently relatively little long-term experience with any of these medications specifically for the treatment of narcolepsy in children and adolescents, most of the recommendations below are based on adult studies.

| TABLE 17.7. Pharmacological Treatment of Narcolepsy in Children and Adolescents Suggested Pediatric Doses for Selected Agents* |

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<table>
<thead>
<tr>
<th>Children (&lt;12 y)</th>
<th>Adolescents/Adults (&lt;12 y)</th>
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<tbody>
<tr>
<td><strong>Medication for EDS</strong></td>
<td></td>
</tr>
<tr>
<td>Methylphenidate (Ritalin) 5 mg BID-TID†</td>
<td>OROS-methylphenidate (Concerta) 18-54 mg†</td>
</tr>
<tr>
<td>Dextroamphetamine (Dexedrine) 2.5-5 mg QD-BID†</td>
<td>Extended-release mixed amphetamine salts (Adderall XR) 5-30 mg†</td>
</tr>
<tr>
<td>Mixed amphetamine salts (Adderall) 5-10 mg BID</td>
<td>Lisdexamfetamine dimesylate (Vyvanse) 20-70 mg in a.m.</td>
</tr>
<tr>
<td>Modafinil (Provigil) 50-200 mg in morning†</td>
<td>Modafinil (Provigil) 100-400 mg in morning‡ (twice daily dosing may be needed in some patients)</td>
</tr>
<tr>
<td><strong>Medication for Cataplexy</strong></td>
<td></td>
</tr>
<tr>
<td>Clomipramine 3 mg/kg/d in morning or at bedtime;</td>
<td>Clomipramine 25-75 mg in morning or at bedtime</td>
</tr>
<tr>
<td>Imipramine 1.5 mg/kg/d (max 100 mg)</td>
<td>Imipramine</td>
</tr>
<tr>
<td>Fluoxetine (Prozac) 5-20 mg daily</td>
<td>Fluoxetine (Prozac) 10-40 mg daily</td>
</tr>
<tr>
<td>Venlafaxine (Effexor) 25-37.5 mg BID</td>
<td>Extended-release Venlafaxine (Effexor XR) 37.5-150 mg in morning (max 300 mg)</td>
</tr>
<tr>
<td>Atomoxetine (Strattera) 10-60 mg/d (max 80 mg); usually in 2 divided doses</td>
<td></td>
</tr>
<tr>
<td><strong>Medication for EDS, Cataplexy</strong></td>
<td></td>
</tr>
<tr>
<td>Sodium oxybate (Xyrem) 3-6 g at night in 2 divided doses</td>
<td>Sodium oxybate (Xyrem) 4.5-9 g/night in 2 divided doses</td>
</tr>
</tbody>
</table>

*There are no medications approved to treat either EDS or other associated symptoms (i.e., cataplexy) of narcolepsy in patients less than 16 y old; thus, the use of these medications are off-label for the indication of narcolepsy, and all suggested doses listed above are based on clinical experience.

†Stimulants listed are those most commonly used to treat EDS in children with narcolepsy, although it should be noted that any of the psychostimulants currently labeled for use in children with ADHD could also be used to treat EDS. Stimulant doses for narcolepsy are typically in the
same dose ranges as are used to treat ADHD; in general, longer-acting preparations are preferred in older children and adolescents.

Modafinil may be started as a morning dose of 50 mg in younger children with increases of 25-50 mg every 5-7 d as needed; older children may start with 100 mg in the morning and increase by 100 mg doses on a weekly basis if needed, up to 400 mg. A second dose may be added in the afternoon if duration of effect is inadequate with a single morning dose.

- **Medications for EDS:**
  - **Psychostimulants** are sympathomimetic amines and also used for the treatment of EDS in narcolepsy. These drugs in general have less effects on cataplexy, although some patients report symptomatic improvement. Their wake-promotion properties are related to increased release and inhibition of reuptake of dopamine, although norepinephrine effects also contribute to the mechanism of action for amphetamines. Medication choices include *methylphenidate* preparations (e.g., Ritalin, Metadate, Concerta, Daytrana) and *dextroamphetamine* (e.g., Dextedrine, Adderall XR, Vyvanse). The extensive clinical experience with the use of psychostimulants in ADHD in children may also make them a relatively more acceptable choice for both providers and families for treating EDS in pediatric narcolepsy. Although, in theory, any of the psychostimulants currently labeled for use in children with ADHD could also be used to treat EDS, most clinical experience thus far has been with the more established preparations, such as short-acting methylphenidate and dextroamphetamine. More recently, the use of longer-acting preparations, such as OROS-methylphenidate and lisdexamfetamine dimesylate, has reduced the need for multiple daily dosing regimens. Dosage titration typically follows a similar protocol to that used clinically for ADHD, with the goal of maximizing alertness while avoiding untoward side effects, including appetite suppression and weight loss, mood changes, increases in heart rate and blood pressure, and sleep disruption. All stimulants have a propensity for the development of tolerance, dependence, and addiction, as well as diversion (use of medication by individuals for whom it is not prescribed). These medications should be used with caution in children with a preexisting heart condition or a family history suggestive of dysrhythmias or early cardiac death. Combinations of long-acting and short-acting forms of stimulants may be necessary to achieve adequate coverage, and concomitant treatment with more than one class of medication may also be needed to control debilitating symptoms of EDS in narcolepsy.
  - **Modafinil** (Provigil) is a wakefulness-promoting drug available as a racemic mixture, which is unrelated to the CNS stimulants and is now considered the first-line pharmacologic treatment for the excessive sleepiness of narcolepsy in adults. The R-isomer **armodafinil** (Nuvigil) has a longer half-life and different dosing (twice as strong). Although the exact mechanism of action is not known, the alertness-enhancing properties of modafinil probably involve relatively selective dopamine reuptake inhibition. In general, modafinil and armodafinil are less efficacious in adults than amphetamine or methylphenidate but may be better tolerated, with fewer peripheral side effects. In particular, the incidence of cardiovascular and psychiatric side effects appears to be less than with stimulants, and abuse and dependence potential is low. The most common side effects are disturbed nocturnal sleep, gastrointestinal discomfort (nausea, diarrhea), decreased appetite, headaches, dry mouth, anxiety, and depression. However, modafinil can induce an allergic reaction, and Stevens-Johnson syndrome (SJS), a life-threatening condition, has been reported in conjunction with its use. Modafinil is also associated with decreased efficacy of hormonally based contraceptives; thus, the use of a barrier method is imperative in sexually active patients in order to avoid unwanted pregnancy. It should be [http://obgynebooks.com](http://obgynebooks.com)
noted, however, that modafinil, similar to the psychostimulants, has acquired “street value” in some settings in regard to use as a countermeasure for sleepiness and/or a “study aid”; thus, an increased risk of diversion exists. Failure to respond to psychostimulants (or modafinil) prescribed at an adequate dosage range should prompt a reevaluation for other possible causes of or contributory factors for EDS.

No randomized controlled trials of modafinil for childhood narcolepsy have been published. A recent review of clinical experience in Europe reported that modafinil was effective in more than 85% of patients, and in the remaining cases, lack of efficacy, habituation, or mild adverse effects (e.g., headache, irritability, loss of appetite) led to drug discontinuation. No severe hypersensitivity reactions were reported, and no serious skin reactions were recorded.

- **Medications for cataplexy (typically REM suppressants):**

  - Antidepressants, including **TCAs**, such as imipramine (Tofranil) and clomipramine (Anafranil), **selective serotonin reuptake inhibitors (SSRIs)**, such as fluoxetine (Prozac), and **other antidepressants** such as venlafaxine (Effexor) and duloxetine (Cymbalta), block the presynaptic reuptake of catecholamines, including serotonin. There is more evidence for their efficacy in treating cataplexy than for the other related symptoms of narcolepsy. Dosing typically starts at lower than the usual antidepressant dose and is titrated up; abrupt discontinuation may cause rebound cataplexy, sleep paralysis, and hypnagogic hallucinations. Side effects of TCAs include dry mouth, sweating, constipation, blurred vision, urinary retention, nausea, and orthostatic hypotension; TCAs are notably contraindicated in subjects with cardiovascular conduction abnormalities. Common side effects of SSRIs include nausea, vomiting, and agitation; more activating SSRIs, such as fluoxetine, may exacerbate sleep disruption. SSRIs and other antidepressants have also been linked to an increase in suicidal ideation in children and adolescents. It should be noted that both TCAs and SSRIs might exacerbate PLMs during sleep. Because of significant potential toxicity of TCAs in overdose, these medications should be used with extreme caution in situations in which accidental or deliberate ingestion may occur and their use is not recommended in prepubertal children.

  - **Atomoxetine** (Strattera) is a selective norepinephrine reuptake inhibitor indicated for the treatment of ADHD in children that appears to have some anticataplectic properties. Side effects include urinary retention, drowsiness, insomnia, and gastrointestinal disturbances.

- **Other medications for narcolepsy:**

  - **Sodium oxybate or α-hydroxybutyric acid (GHB) (Xyrem)**, AA-aminobutyric acid B receptor (GABA-B) agonist, has been approved by the Food and Drug Administration as the only medication for the treatment of both EDS and cataplexy in narcoleptic patients over the age of 16 years. The mechanism of action is not known, but sodium oxybate is known to activate the GABA-B receptor. Note that it increases slow-wave sleep. It appears to be effective for symptoms of EDS, cataplexy, and disrupted nocturnal sleep, and may be effective for the treatment of hypnagogic hallucinations and sleep paralysis. The full therapeutic effect of sodium oxybate often takes weeks to months to manifest. The typical adult dose is 6 to 9 g, and the comparable pediatric dose appears to be about 60 mg/kg. Due to the short elimination half-life (40-60 minutes), one-half of the therapeutic dose is administered at bedtime, and the second half 2.5 to 4 hours later. Side effects may include nausea and vomiting, loss of appetite, weight loss, tremor, constipation, headache, anxiety, and suicidal ideation. Sodium oxybate may also increase salt load and can be a respiratory depressant; therefore, use in patients with comorbid OSA is relatively contraindicated. Alcohol use is also contraindicated in patients on
Xyrem. It should also be noted that sodium oxybate has a significant potential for abuse. It is sometimes referred to as the “date rape drug” because of its rapid induction of profound sleepiness. It has also been used recreationally (e.g., to “cool down”) after taking abused stimulants, and with continuous use at high doses, cessation can create severe withdrawal symptoms and overdoses can be fatal. Thus, its availability is rigorously controlled, and it is only shipped from a central pharmacy; both patients and physicians must register with the pharmaceutical company that supplies the drug.

While there are no randomized controlled trials of sodium oxybate in children, a recent small retrospective study showed subjective improvements in cataplexy, EDS, and nocturnal sleep disruption in the majority of pediatric patients; 15% of the patients on sodium oxybate discontinued due to side effects (e.g., sleep loss, persistent nausea). At an average follow-up of 4 years, 35% of patients were on monotherapy with sodium oxybate and rest on combinations with stimulants, modafinil, and/or venlafaxine.

**Benzodiazepines.** Case reports and case series, some of which have included pediatric patients, have suggested that benzodiazepines, such as clonazepam, triazolam, and temazepam, might reduce symptoms of EDS and cataplexy in patient with narcolepsy. The mechanism is postulated to be related to improvements in sleep fragmentation and consolidation of nocturnal sleep in these patients following a bedtime dose.

### PROGNOSIS
Narcolepsy is a chronic, lifelong disorder that will always require management; patients with narcolepsy typically have a normal lifespan. While narcolepsy with cataplexy is believed to have a stable course, there are few longitudinal studies, especially in narcolepsy presenting in childhood. The goal of treatment is adaptation, safety, and improved quality of life.

#### Tips for Talking to Parents
- **Explain what narcolepsy is** in basic terms: a chronic neurologic disorder in which there is an imbalance in the control of sleep and wakefulness.
- **Explain what is currently understood regarding the genetic and neurological basis** of narcolepsy.
- **Emphasize** the patient's relative lack of control over EDS, especially sleep attacks. In particular, school officials should understand that students are not “lazy” or uncooperative if they exhibit signs of EDS in class.
- **Review the daytime consequences of narcolepsy** because parents may not attribute daytime sleepiness, academic problems, and apparent inattention, for example, to a sleep problem.
- **Discuss the manifestations of the other common symptoms** of narcolepsy (i.e., cataplexy, hypnagogic hallucinations, sleep paralysis), and the relative possibility that the patient may develop these feature in the future.
- **Emphasize that narcolepsy is currently viewed as a lifelong disorder but one that may be controlled with behavioral and medication treatments.** This is often a devastating diagnosis for both patients and families, and is analogous to a child or adolescent being diagnosed with another serious medical condition, such as diabetes. Families should be given time to adjust to the diagnosis and offered both ongoing support and frequent opportunities to ask questions.
• **Emphasize safety concerns**, as children with cataplexy in particular may experience injuries during cataplexy attacks. In adolescents, discussion of safety while driving is imperative.

• **Discuss the risks and benefits of treatment options**, including lifestyle changes and medication choices.

• **Encourage any parent or other family member who experiences EDS** to be evaluated for narcolepsy.

*See Appendix D14 for a parent handout on narcolepsy.*
Circadian rhythm disorders in general either represent a “mismatch” between the individual’s intrinsic sleep-wake schedule and environmental demands (delayed and advanced sleep-phase disorders, shift work disorder, jet lag disorder) or an internal failure of synchronization of the circadian clock (non-24-hour sleep-wake disorder).

By far the most commonly clinically encountered circadian sleep-wake disorder in the pediatric population is delayed sleep-wake phase disorder (DSWPD), which involves a habitual and persistent phase shift of more than 2 hours in the sleep-wake schedule (later sleep onset and waketime) that conflicts with the individual’s normal school, work, and/or lifestyle demands. It is the timing rather than the quality of sleep per se that is problematic, as individuals with DSWPD typically do not have sleep complaints if they are allowed to sleep on their preferred (later) schedule. The most common clinical presentation is sleep initiation insomnia when the individual attempts to fall asleep at a socially acceptable desired bedtime, accompanied by extreme difficulty getting up in the morning, even for desired activities, and daytime sleepiness. Insomnia disorder may also develop, often as a result of trying to cope with the inability to fall asleep.

**Delayed Sleep-Wake Phase Disorder**

DSWPD, a circadian rhythm disorder, involves a significant and persistent shift in an individual’s sleep-wake schedule that interferes with environmental demands, usually resulting in significant daytime sleepiness and academic and behavior problems.

A subtype of DSWPD is motivated delayed sleep-wake phase disorder, which is typically composed of adolescents who are not willing to change their sleep schedule nor resume a normal lifestyle, primarily related to going to school. Mood or anxiety disorders, especially social phobia or social anxiety, and family dynamics are common contributors to the genesis of this disorder. Other comorbid conditions also are often present or trigger the sleep disturbance, including pain syndromes, chronic fatigue, and postural orthostatic tachycardia syndrome (POTS).

DSWPD may occur at any age, but is most common in adolescents and young adults. Individuals with DSWPD often start out as “night owls”; that is, they have an underlying predisposition/circadian preference (possibly genetically determined) for staying up late at night and sleeping late in the morning, especially on weekends, holidays, and summer vacations. The typical sleep-wake pattern in DSWPD is a consistently preferred bedtime or sleep-onset time after midnight (typically 2:00-6:00 a.m.) and waketime after 10:00 a.m. In order to cope, many adolescents with DSWPD take lengthy afternoon naps or attempt to “catch up” by extending sleep on weekends and days off from school. With DSWPD, even highly motivated adolescents face considerable challenges in shifting their sleep back to an earlier time without assistance.

**Other Circadian Rhythm Sleep-Wake Disorders**

Although uncommon, as delineated in the ICSD-3, there are other circadian rhythm sleep-wake disorders that may occur in children:

- **Advanced sleep-wake phase disorder (ASWPD).** ASWPD is characterized by a stable advance of the major sleep disorder, in that habitual sleep onset and offset occurs 2 or more hours earlier than required or desired times. Although rare in children and adolescents,
those with ASWPD typically complain of early morning awakenings, maintenance insomnia, and excessive evening sleepiness.

- **Irregular sleep-wake rhythm disorder (ISWRD).** ISWRD is more common in children and adolescents, especially in children with neurodevelopmental disorders (e.g., autism spectrum disorders, Angelman syndrome, Williams syndrome, Smith-Magenis syndrome) and medical conditions (e.g., traumatic brain injury, brain tumors). ISWRD is characterized by lack of a clearly defined circadian rhythm of sleep and wake, with sleep occurring at variable times throughout the 24-hour cycle. Presenting complaints often include insomnia and excessive sleepiness, and parents may complain that their child sleeps too much or too little or at inappropriate times (e.g., “falls asleep too early,” “wakes too early,” has difficult waking in the morning, sleeps during the day). Total sleep time, however, may be normal for age, although the sleep-wake cycle is disorganized. The key characteristics of ISWRD are the lack of prolonged consolidated sleep periods, the seemingly random distribution of sleep across the 24-hour day, and the marked variability across days or weeks with no predictable sleep-wake pattern.

- **Non-24-hour sleep-wake rhythm disorder (N24SWD).** N24SWD is characterized by a lack of entrainment of the intrinsic circadian pacemaker to the 24-hour light-dark cycle and includes symptoms of insomnia and excessive sleepiness. Short periods of alignment may occur. N24SWD occurs primarily in children and adolescents who are totally blind, as well as those with developmental intellectual disabilities. The key characteristics of N24SWD are the predictable pattern of misalignment between the child's sleep patterns and the light-dark 24-hour cycle and periods of apparent symptom remission during transient periods of alignment.

- **Jet lag disorder.** Jet lag is characterized by a temporary misalignment of the sleep-wake cycle generated by the endogenous circadian clock as a result of a change in time zone. Sleep complaints include sleep disruption, sleepiness, fatigue, and impaired daytime functioning.

**Epidemiology**

- **Prevalence:** Studies indicate that DSWPD affects approximately 7% to 16% of adolescents. DSWPD is seen in approximately 10% of sleep clinic patients presenting with complaints of insomnia.

- **Gender:** At least one study of late adolescence/early adulthood indicated that young men are more likely to have an evening preference than young women.

- **Age of onset:** Onset is typically during adolescence, when an underlying circadian preference for late bedtime and waketime (“eveningness”) may become exaggerated during the normal pubertal shift to later sleep onset and waketimes (see Chapter 2). Younger children with a marked phase delay may also develop DSWPD.

**Etiology and Risk Factors**

In order to fully understand the sleep initiation insomnia and profound difficulties in morning waking in patients with DSWPD, it is important to understand some basic principles of circadian biology. First, in the several hours prior to the habitual preferred bedtime, there is a marked circadian-mediated increase in wake promotion, the so-called “second wind” phenomenon or “forbidden zone.” Thus, an individual with DSWPD who gets into bed at a “normal” bedtime is typically attempting to fall asleep during this period of maximum alertness. Similarly, the period 1.5 to 2 hours before the preferred waketime represents the nadir of wakefulness promotion by the circadian system, resulting in extreme difficulty in becoming alert at the desirable waketime in patients with DSWPD. This morning sleepiness is further compounded by an increased homeostatic sleep drive that results from chronically insufficient sleep in these individuals as schedule demands change (e.g., earlier school start...
times in high school). Late evening activities, such as sports practice and late-day caffeine consumption, may further delay sleep onset. Furthermore, exposure to “screens” (e.g., television, computer, e-reader) may contribute to the development and maintenance of the delayed sleep phase by suppressing melatonin release. Decreased exposure to light in the morning may also exacerbate the delayed circadian phase.

The etiology of DSWPD is still unknown, with many theories postulated. Some theorize that DSWPD involves an intrinsic abnormality in the circadian oscillators that govern the timing of the sleep period. That is, there may be a problem in the synchronization or “entrainment” of the circadian clock to environmental cues or an intrinsic circadian period that is longer than average. Evidence supporting a primarily neurobiological substrate for this disorder also includes persistence of the disorder in the face of significant academic and social consequences, high rates of relapse following successful intervention, and some evidence supporting a genetic component. Others postulate that DSWPD involves abnormalities in the circadian phase response curve to light, as mentioned above; not only are these patients less likely to be exposed to morning light because they sleep later and more likely to be exposed to evening light as a result of delayed bedtimes, but they may have an underlying increased sensitivity to even low levels of evening light. Others have postulated that individuals with DSWPD have a diminished ability to compensate for lost sleep and have reduced homeostatic drive even if they awaken early.

In addition, those children or adolescents experiencing difficulties in school or school refusal may present with a preferred or motivated phase delay—risk factors that favor persistence of symptoms and make the disorder more difficult to treat. The relationship between psychiatric disorders and DSWPD is a complex one; DSWPD may result in symptoms of depression, and the insomnia associated with primary depression may mimic DSWPD. Furthermore, studies have indicated that a strong circadian “eveningness” preference in and of itself may confer an increased risk of depressive symptoms, as well as other mental health concerns (e.g., impulsivity, aggressive and antisocial behavior, substance and alcohol use).

Finally, a positive family history has been found in 40% of individuals with DSWPD. It has been postulated that there is an autosomal dominant trait that may be a significant contributor.

**PRESENTATION AND SYMPTOMS**

The most common presenting symptoms are sleep-onset insomnia and oversleeping/daytime sleepiness. Poor school attendance is also very common.

- **Sleep onset at a consistently late time**, usually after midnight in adolescents.
- **Minimal difficulty with sleep maintenance**, with infrequent nighttime awakenings.
- **Significant difficulty waking at the required time (e.g., waking for school)**, decreased alertness in the morning, and often daytime sleepiness.
- **Persistent difficulty going to sleep at an earlier time**, rather than the preferred bedtime. Although, with effort, the individual with DSWPD may temporarily achieve sleep onset at an earlier time, there is a natural tendency to “drift” to the later preferred bedtime.
- **Complaints of “insomnia,”** if the adolescent attempts to go to sleep at an earlier time; however, no sleep-onset insomnia is experienced if bedtime coincides with the preferred sleep-onset time (e.g., on weekends, school vacations).
- **Daytime sleepiness (e.g., napping, dozing off, mood changes, inattentiveness)**, in addition to difficulty waking in the morning, may be experienced throughout the day due to chronic insufficient sleep.
Motivated DSWPD should be considered if there is a history of the following:

- **History of mood/anxiety disorder**, especially social phobia and social anxiety.
- **Ill-defined medical issues**, such as pain syndromes, chronic fatigue, and POTS.
- **Exaggerated symptoms**, such as not being able to be awakened with extreme measures (e.g., throwing cold water).
- **Failure to comply with simple treatment recommendations**, such as refusing to stop napping or lights out at the same time every night.

### Diagnostic Criteria
See Table 18.1.

### Associated Features

- **Bedtime resistance and delayed sleep onset** in younger children. Children with an evening preference may have difficulty falling asleep at an age-appropriate bedtime, leading to protests, excuses, and oppositional behavior.
- **Evening or night preference** with optimal functioning (“second wind”) during the late afternoon, evening, and night hours.
- **Weekend “oversleep”** until late morning or early afternoon in an attempt to make up for weekday insufficient sleep. Extended sleep periods are often seen during vacations as well, especially in the first few days of the vacation.
- **Poor school performance**, as adolescents with DSWPD are often chronically sleep deprived and thus, even when highly motivated, may perform poorly in school.
- **School tardiness and frequent absenteeism** are often present. The sleep problem may lead to a downward spiral of missed school due to the frequent tardiness and absences, resulting in academic failure and, subsequently, in more school avoidance. In severe cases, the child or adolescent may have stopped going to school altogether.
- **Increased alcohol and caffeine** have been reported in adolescents with DSWPD.
- **Decreased sports participation but increased other primarily solitary activities**, such as learning to play a musical instrument.

### TABLE 18.1. Diagnostic Criteria: Circadian Rhythm Sleep-Wake Disorder, Delayed Sleep-Wake Phase Disorder

**General Criteria for Circadian Rhythm Sleep-Wake Disorder**

A. A chronic or persistent pattern of sleep-wake rhythm disturbance primarily due to alteration of the endogenous circadian timekeeping system or misalignment between the endogenous circadian rhythm and the sleep-wake schedule desired or required by an individual’s physical environment or social/work schedules.

B. The circadian rhythm disruption leads to insomnia symptoms, excessive daytime sleepiness, or both.

C. The sleep and wake disturbances cause clinically significant distress or impairment in mental, physical, social, occupational, educational, or other important areas of functioning.

**Specific Criteria for Delayed Sleep-Wake Phase Disorder**

A. There is a significant delay in the phase of the major sleep period in relation to the desired or
required sleep time and wake-up time, as evidenced by a chronic or recurrent complaint by the patient or a caregiver of inability to fall asleep and difficult awakening at a desired or required clock time.

B. The symptoms are present for at least 3 mo.

C. When patients are allowed to choose their ad libitum schedule, they will exhibit improved sleep quality and duration for age and maintain a delayed phase of the 24-h sleep-wake pattern.

D. Sleep log and, whenever possible, actigraphy monitoring for at least 7 d (preferably 14 d) demonstrate a delay in the timing of the habitual sleep period. Both work/school days and free days must be included within this monitoring.

E. The sleep disturbance is not better explained by another concurrent sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

ICD-9-CM: 327.31; ICD-10-CM: G47.21


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**EVALUATION**

- **Medical history:** The history is generally benign.

- **Circadian misalignment and school history:** Parents often note that these children are frequently lifelong “night owls.” Questions regarding morningness-eveningness (“owl-lark”) preferences may reveal a pattern of alertness and sleepiness during the day that is delayed relative to normal circadian peaks and troughs. Questions about “feel best” times for going to bed, morning waking, and performing various tasks (e.g., taking tests, playing a sport) may be helpful in confirming a pattern of delayed circadian rhythms. A key differentiation is the extent of sleeping in on weekends and nonschool days, as well as weekend oversleep. Individuals with insomnia may sleep later on weekends, but it is usually minimal compared to school days, whereas those with DSWPD will have significantly different schedules on weekends. Furthermore, academic problems are common and usually a result of the sleep disorder. When academic concerns predate the sleep problem, there may be an element of school avoidance compounding the situation.

- **Family history:** The history may reveal other family members with a marked evening circadian preference. Approximately 40% of individuals with DSWPD have a positive family history.

- **Behavioral assessment:** It is extremely important to assess for possible substance use and psychiatric issues, including depression, anxiety disorders, school refusal, and school phobia. Furthermore, an assessment of motivation and secondary gains, such as school avoidance, is critical to ascertaining other factors that will likely interfere with treatment success.

- **Physical examination:** The examination is generally benign.

- **Diagnostic tests:**
  
  - **Sleep diaries:** Recording of sleep-wake patterns is a very important component of the evaluation for DSWPD and will often graphically demonstrate the phase delay and consistent fall-asleep time.

  - **Circadian phase preference questionnaires:** These include the Children's Chronotype Questionnaire [http://obgynebooks.com](http://obgynebooks.com)
Actigraphy: Use of an actigraph (a wristwatch-like device that measures and stores data regarding body movements over time; this is transformed with a computerized algorithm into sleep-wake patterns; see Chapter 3) in conjunction with a sleep diary may be a very useful adjunct to more objectively document sleep-wake patterns over a period of 1 to 2 weeks. According to recent practice parameters developed by the American Academy of Sleep Medicine, actigraphy is not only indicated for initial assessment but can also be useful as an outcome measure in evaluating the response to treatment.

Polysomnography: In general, overnight sleep studies are not indicated unless there is a concern regarding another primary sleep disorder such as obstructive sleep apnea. In fact, due to their delayed sleep schedule and consequent prolonged sleep-onset latency, it is often difficult to obtain sufficient sleep on an overnight sleep study in patients with DSWPD unless the lab can accommodate a “late” waketime in the morning.

Salivary melatonin. Sequential salivary melatonin levels under dim light conditions (dim light melatonin onset; DLMO) may be helpful in determining the underlying circadian rhythm. Although such measurement is currently used primarily in research settings, commercially available kits are expected to become available in the future.

Sample Questions from the CCTQ

1. Considering your child's “feeling best” rhythm, at what time would your child go to bed if he or she could decide by himself or herself and if he or she were entirely free to plan the next day (e.g., weekend)?
   a. Prior to 6:59 p.m.
   b. 7:00 to 7:59 p.m.
   c. 8:00 to 9:59 p.m.
   d. 10:00 to 10:59 p.m.
   e. After 11:00 p.m.

2. Let's assume that your child has to be at peak performance for a test that will be mentally exhausting for 2 hours. Considering your child's “feeling best” rhythm and that you are entirely free to plan your child's day, which one of the three time intervals would you choose for the test?
   a. 7:00 to 11:00 a.m.
   b. 11:00 am to 3:00 p.m.
   c. 3:00 to 8:00 p.m.

3. At what time in the evening does your child seem tired and in need of sleep?
   a. Prior to 6:30 p.m.
   b. 6:30 to 7:14 p.m.
   c. 7:15 to 9:29 p.m.
   d. 9:30 to 10:14 p.m.
   e. After 10:15 p.m.

DIFFERENTIAL DIAGNOSIS

It is important to confirm that the presenting complaint and current symptoms are the result of a circadian misalignment, and not the result of insomnia, insufficient sleep, or a comorbid mental health issue or substance use.

- **Insomnia:** Difficulties falling asleep at night may be the result of psychophysiologic or “learned” insomnia (see Chapter 19). One clear differentiating factor between the two is that children and adolescents with DSWPD have few or no difficulties falling asleep if they go to bed at their preferred sleep-onset time. In contrast, individuals with insomnia experience difficulties falling asleep no matter what time they go to bed and often do not exhibit a consistent fall-asleep time. However, it should be noted that, in clinical practice, patients with DSWPD frequently develop a secondary “conditioned” insomnia due to spending prolonged periods of time in bed attempting to fall asleep; thus, implementation of behavioral interventions targeted at reversing this situation (e.g., stimulus control, sleep restriction) may be necessary.

- **Restless legs syndrome (RLS):** Symptoms of RLS (e.g., an urge to move the extremities accompanied by uncomfortable sensations in the legs that is worse with inactivity and relieved by movement; see Chapter 16) may present as delayed sleep onset.

- **Poor sleep hygiene and evening screen time:** Maintenance of an erratic sleep schedule, use of caffeine or other substances, and technology use (“screen time”) in the evening may result in complaints of difficulty falling asleep. However, individuals with DSWPD will still have delayed sleep onset even after the establishment of an appropriate sleep schedule and the institution of appropriate sleep habits (see Chapter 5).

- **Circadian preference:** Although a tendency toward eveningness (“night owl”) is a risk factor for DSWPD, typically this preference does not have the same intractable quality and persistence, and does not result in significantly compromised functioning.

- **Lifestyle issues and insufficient sleep:** For example, socializing and late-night television viewing can result in insufficient sleep and difficulty awakening in the morning, but patients do not complain of “insomnia.”

- **School avoidance or refusal:** Adolescents with primary school avoidance, such as that resulting from an anxiety disorder or related to a learning disability and academic failure, may have a late bedtime and appear difficult to awaken in the morning. In this situation, the sleep schedule is not intractable, and a more appropriate sleep schedule may be maintained on weekends and holidays. However, school avoidance frequently coexists with and exacerbates DSWPD.

- **Psychiatric disorders:** Depression, bipolar disorder, and anxiety disorders are associated with difficulty falling asleep, but the set sleep-wake schedule pattern that is characteristic of DSWPD is not usually present in these situations, and the sleep disturbance will covary with the psychiatric symptoms.

- **Substance use:** Substance use, both prescription medications and alcohol/drug use, can result in insufficient sleep, daytime sleepiness, and poor school attendance.
When to Refer
Children or adolescents with DSWPD who also demonstrate symptoms of other underlying sleep disrupters (e.g., obstructive sleep apnea, RLS/periodic limb movement disorder) may require referral to a sleep specialist. When there are concerns regarding school refusal, depression, or other psychological issues, referral to a mental health specialist is warranted. Treatment programs involving sleep scheduling, including chronotherapy, light therapy, and melatonin, are best accomplished by or in consultation with a sleep specialist.

Treatment
The goal in the treatment of DSWPD is basically two-fold: first, shifting the sleep-wake schedule to an earlier time and, second, maintaining the new schedule. The choice of a target bedtime and waketime should be discussed with the patient and family in the context of balancing the primary goal of adequate sleep with lifestyle considerations. The initial treatment phase is generally more intense and requires strict adherence to the treatment protocol; the maintenance phase is necessary because of the natural tendency of these individuals to gradually shift to a later bedtime and waketime.

Motivation: Key to Successful Treatment
A highly motivated adolescent is required, as achieving success in realigning an adolescent's sleep schedule and maintaining the change can be difficult. Thus, issues such as the motivation of the child or adolescent, the resources of the family, and the existence of any psychiatric or substance abuse problems must first be addressed before success can be expected. Sufficient motivation for (and barriers to) instituting change must be explored. Similar to many other behavioral interventions, a well-developed plan is essential to achieve success. Behavioral contracts are often necessary, and psychological and family issues may need to be addressed if resistance to change is encountered, as is frequently the case.

Successful treatment requires a motivated patient and family, and a coordinated approach that includes changes in sleep habits. Often, a combination of sleep scheduling, melatonin, and/or bright-light therapy is used. However, it should be noted that there is overall little consensus, particularly in adolescents, regarding the optimal procedures to implement them although practice parameters indicate that timed light exposure and timed melatonin are guideline indications. Ideally, timing of treatment should be based on direct measurements of the “phase response curve” (e.g., core body temperature, salivary melatonin levels); however, this is clearly not yet practical in most clinical settings. Therefore, the following recommendations are based on “guesstimates” of circadian timing that are applicable to clinical practice, but consultation with a sleep specialist is usually warranted.

Shifting the Circadian Rhythm: Phase Advancement and Phase Delay (Chronotherapy)
Treatment for DSWPD involves shifting the circadian sleep-wake rhythm so that sleep onset and morning waking occur at the desired times. There have been positive case reports documenting the efficacy of phase shifting, although no controlled trials in adolescents have been conducted. There are two primary strategies to accomplish this shift:

- **Phase advancement and timed light therapy**: Phase advancement is used when the difference between the current later sleep-onset time and the target earlier sleep-onset time is less than 3 hours. At first, the schedule is stabilized with the patient maintaining a consistent sleep schedule. Waketimes are then shifted
earlier by approximately 30 to 60 minutes per day. Thus, if the patient typically wakes at noon, the first-day waking is set to 11:00 a.m., then 10:00 a.m., etc. As waketime gets earlier, usually starting around 9:00 a.m., waketimes are shifted in smaller increments, such as 30 minutes per day. Each day upon waking, bright-light treatment should be instituted whether via exposure to natural outdoor light or via a specialized light box. Bedtimes can be shifted according to the same schedule, although the shift in bedtime is usually done in just 15- to 30-minute intervals to maintain significant sleep pressure at bedtime and avoid prolonged periods of lying awake in bed. Shifting the morning waketime while more gradually advancing the bedtime over several weeks will result in relative sleep restriction and should be explained to the patient and the family. Sleep restriction assists in accomplishing the phase shift by increasing the homeostatic sleep drive during the initial period of treatment, thus increasing the likelihood that the patient will be ready to fall asleep at the earlier bedtime.

The pace (shifting the waketime and bedtime earlier every day versus every 2 or 3 days) and the time increment of the shift (advancing by 15 versus 30 versus 60 minutes) is somewhat dependent on the chronicity of the delayed sleep-phase problem (the longer the duration, the more gradual the process needed), the patient’s ongoing response to treatment, and how quickly the shift to the target bedtime and waketime must be accomplished.

---

**Sample Phase Advancement Schedule**

<table>
<thead>
<tr>
<th></th>
<th>Bedtime</th>
<th>Waketime</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline night</td>
<td>2:00 a.m.</td>
<td>11:00 a.m.</td>
</tr>
<tr>
<td>Treatment night 1</td>
<td>1:45</td>
<td>10:00 a.m. (light exposure)</td>
</tr>
<tr>
<td>Treatment night 2</td>
<td>1:30</td>
<td>9:00 a.m. (light exposure)</td>
</tr>
<tr>
<td>Treatment night 3</td>
<td>1:15</td>
<td>8:30 a.m. (light exposure)</td>
</tr>
<tr>
<td>Treatment night 4</td>
<td>1:00</td>
<td>8:00 a.m. (light exposure)</td>
</tr>
<tr>
<td>Treatment night 5</td>
<td>12:45</td>
<td>7:45 a.m. (light exposure)</td>
</tr>
</tbody>
</table>

*Continue to advance bedtime and waketime by 15 min nightly.*

Goal night          | 10:30 p.m.           | 6:30 a.m. (light exposure) |

---

**Phase delay (chronotherapy):** Phase delay is used for more severely delayed sleep-phase cases (e.g., shift
is greater than 3 hours) and involves delaying bedtime and waketime by 2 to 3 hours daily to every other day. Thus, if an adolescent usually goes to bed at 4:00 a.m., on the first day of intervention bedtime is scheduled for 6:00 a.m. (or 7:00 a.m.), the second day 9:00 a.m., and so on, until the desired bedtime is achieved. Adolescents are often compliant during the first phase of this treatment because of the perception that they are being “allowed” to stay up later each day. Since the patient is usually sleepy by the successively later scheduled sleep times, delayed sleep onset is not a problem; however, the patient may have difficulty remaining awake for an additional 2 to 3 hours each day and may need family support and supervision to accomplish this. Chronotherapy requires considerable motivation and commitment on the adolescent’s part as the treatment phase is quite disruptive, and will necessitate missing school during the period in which sleep is scheduled to occur in daytime hours. If possible, chronotherapy should be timed to coincide with a school vacation to avoid missing school during the period of daytime sleep. The disadvantages of phase delay typically make it a second-line treatment option.

### Sample Phase Delay Schedule

<table>
<thead>
<tr>
<th></th>
<th>Bedtime</th>
<th>Waketime</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>4:30 a.m.</td>
<td>12:30 p.m.</td>
</tr>
<tr>
<td>Treatment night 1</td>
<td>7:30</td>
<td>3:30</td>
</tr>
<tr>
<td>Treatment night 2</td>
<td>10:30</td>
<td>6:30</td>
</tr>
<tr>
<td>Treatment night 3</td>
<td>10:30</td>
<td>6:30</td>
</tr>
<tr>
<td>Treatment night 4</td>
<td>1:30</td>
<td>9:30</td>
</tr>
<tr>
<td>Treatment night 5</td>
<td>4:30</td>
<td>12:30</td>
</tr>
</tbody>
</table>

*Continue to advance bedtime and waketime by 3 h nightly.*

<table>
<thead>
<tr>
<th></th>
<th>Bedtime</th>
<th>Waketime</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goal night</td>
<td>7:30</td>
<td>3:30</td>
</tr>
</tbody>
</table>

### Additional behavioral strategies
Healthy sleep habits: The patient's sleep habits should be reviewed and reorganized if necessary, with an emphasis on the development of positive sleep routines and avoidance of evening electronics exposure and caffeine. Caffeine and other stimulant use can also contribute to a delayed sleep onset, especially late afternoon and evening consumption. Secondary poor sleep habits, such as lying in bed awake for long periods, also need to be addressed (see Appendices C7 and C8).

Avoid napping and maintain sleep schedule: Napping must be avoided to help consolidate sleep. In addition, the patient must maintain a consistent sleep schedule both on weekdays and weekends, even after the initial treatment phase is completed.

Bright-light therapy: Although there are no established guidelines regarding intensity, duration, and timing of light exposure, scheduled exposure to bright light is beneficial to help set the sleep-wake rhythm to an earlier time. However, light exposure at the "wrong" circadian time (i.e., before the nadir in core body temperature) may actually exacerbate the phase delay; a reasonable estimate of circadian nadir is 2 hours before the habitual (preferred) waketime. In uncomplicated or mild cases, light exposure can consist of eating breakfast in a sunny area of the home and spending time outdoors first thing in the morning. In more difficult cases, exposure to 20 to 30 minutes of bright light (2,500-10,000 lux) provided by a specially designed light box unit will help. Patients should also avoid light exposure in the evening, and dark glasses may be worn during exposure to sunlight late in the day. Commercially produced light boxes or visors are portable units, which combine high-output (2,500-10,000 lux) fluorescent tubes with reflectors, and can be obtained online at such sites as www.litebook.com, www.apolollighttherapy.com, and www.lighttherapyproducts.com. Some studies have suggested that short wave-length (blue) light may be relatively more effective in phase shifting. Light exposure therapy should be continued for at least 2 to 4 weeks.

Tips for Bright-Light Exposure Therapy

- As each manufacturer has different specifications, it is important to read instructions carefully for the individual light box or lamp unit.
- The UV filter (if needed) should be in place before use.
- The patient should not look directly at the light but make sure the light shines on the eyes; no sunglasses should be worn. Patients may read, watch television, or eat during the period of light exposure.
- Distance from the light box unit and head position determines the light intensity delivered. If engaging in other activities (reading, eating) while using the light box, the light should be positioned about 16 to 20 inches from the patient, at head or chest level.
- Some patients experience eye or skin irritation (especially patients with fair skin, light eye color), queasiness, and headaches with use of a light box. If this occurs, increasing the distance of the light box and decreasing the exposure time before gradually increasing it may help.
- Some antibiotics and acne or retinoic acid creams may increase light sensitivity.

Motivational interviewing: Motivational interviewing, an emerging technique to affect behavior change, can be critical, especially in the treatment of motivated DSWPD. Motivational interviewing helps the patient both identify that there is a problem and elicit motivation to change. To help recognition of a problem, education about what constitutes a sleep problem and questioning about current consequences of sleep difficulties (e.g., poor school performance, family stress) can help with recognition that there is an issue. A good first step is
asking questions regarding both the positive and negatives of both maintaining the status quo and making changes. Having adolescents articulate the benefits of the current situation and the negatives of change (e.g., having to return to school) can help identify underlying motivations and allow open discussion about motivation. Continued questioning will help engage patients to identify positive aspects of change and increase motivation to change. Treatment is likely to fail, and patients are likely to refuse to comply with treatment recommendations in situations where there is minimal to no motivation.

Pharmacologic approaches

- **Melatonin:** There have been several studies indicating the efficacy of melatonin to promote a corrective phase advance, although with variable results and relapse following discontinuation. These studies have found that physiologic doses of oral melatonin (0.3-0.5 mg) administered in the afternoon or early evening (i.e., 1.5-7 hours before the habitual sleep-onset time) seem to be most effective in advancing the sleep phase. Larger doses may actually maintain endogenous melatonin levels and the phase delay. Melatonin timing should really be based on DLMO, although that may be difficult to assess. In general, the earlier the melatonin is administered, the larger the phase advance; however, similar to phototherapy, inappropriately timed melatonin administration may worsen the phase delay. It should be noted that melatonin also has hypnotic (sedating) effects when given in larger doses just before bedtime. Finally, long-acting melatonin has been found to maintain and at times even worsen a phase delay. Unfortunately, there is no consensus on the best time to take melatonin to achieve a phase advance. Also note that there are some potential risks with the use of melatonin (see Chapter 20).

- **Hypnotics:** Hypnotic use has been suggested as a means to induce sleepiness at the desired bedtime. To date, there is insufficient evidence as to the efficacy of such use in the treatment of DSWPD.

**Additional issues**

- **Maintenance phase:** Treatment for DSWPD usually involves an intense treatment phase, during which there can be no deviation from the prescribed schedule. Following this phase, there will be a protracted maintenance phase, which is also quite difficult. Many find this phase to be the more difficult aspect of treatment, as there will continue to be an evening preference. All it takes is one weekend or vacation in which old habits are resumed to undo all achievements. Once the new schedule is firmly entrenched, an occasional late night is permitted, but the adolescent should not be allowed to sleep more than 1 or 2 hours later than his or her usual weekday waketime. In addition, patients should continue to avoid bright light in the evenings, whereas light exposure in the morning should be as bright as possible. Daytime napping should continue to be avoided, as sleeping during the day will decrease sleepiness at bedtime, resulting in a delay of sleep onset.

- **Other behavioral and mental health issues:** Other issues must be addressed if warranted, such as school refusal and other psychiatric problems (e.g., depression, substance use).

**PROGNOSIS**

DSWPD is often a chronic disorder, but can be changed with high motivation. However, a high rate of relapse following initial treatment success is common.

**Tips for Talking to Parents and Patients**

- Describe DSWPD and emphasize the persistent nature of the circadian clock with this disorder.
Discuss treatment options, including phase advancement or phase delay, light exposure, and melatonin use.

Encourage parents to have the child or adolescent take responsibility for the sleep schedule.

Assess motivation to change and engage in motivational interviewing with the child or adolescent.

Explain the role of sleep hygiene in treatment success.

Discuss issues of sleep schedule maintenance following the shift in the circadian rhythm.

See Appendix D14 for a parent handout on DSWPD.
Insomnia is broadly defined as subjective significant difficulty initiating and/or maintaining sleep, or early morning awakening. These sleep complaints must also result in some degree of impairment in daytime functioning, which may range from irritability, attention/concentration impairment, and behavioral disturbances to more significant effects on mood and school performance. The sleep complaint must be accompanied by distress about poor sleep and/or impairment in the family, and occur despite adequate opportunity and adequate circumstances (e.g., safe, dark, quiet environment).

Insomnia complaints may be of a short-term and transient nature (usually related to an acute, and often stressful, event), or may be characterized as long-term and chronic (at least 3 times a week for 3 months). In the updated Diagnostic and Statistical Manual, 5th ed. (DSM-5) and International Classification of Sleep Disorders, 3rd ed. (ICSD-3) nosologies, a diagnosis of insomnia should now be made without attributing causality to the presence of a comorbid condition, such as depression, anxiety, or pain (i.e., insomnia is no longer characterized as “due to” a medical or psychiatric condition). It is also important to note that difficulty falling asleep or staying asleep may be caused by another sleep disorder, such as restless legs syndrome (RLS) (Chapter 16) or obstructive sleep apnea (OSA) (Chapter 15).

However, the criteria for “significant difficulties” initiating and maintaining sleep in both adults and children is obviously quite subjective, especially when reported by caregivers rather than the patient, and dependent on a number of factors (e.g., culturally based sleep practices, child temperament, environmental conditions). In addition, the presenting complaints (e.g., bedtime resistance) and manifestations of childhood insomnia (e.g., hyperactivity, academic difficulties) may be quite different from those in adults. In response, a consensus definition of pediatric insomnia was developed a number of years ago. This definition states that pediatric insomnia involves “repeated difficulty with sleep initiation, duration, consolidation, or quality that occurs despite age-appropriate time and opportunity for sleep and results in daytime functional impairment for the child and/or family.” While DSM-5 and ICSD-3 do not use separate diagnostic criteria for adult and pediatric insomnia, a number of key features of the consensus definition (e.g., symptoms as reported by the patient or caregiver, behavioral problems as a manifestation of sleepiness) are now incorporated into the updated DSM-5 and ICSD-3 diagnoses.

Although insomnia is one of the most common sleep complaints in adults, parents (as well as children and adolescents themselves) may present with complaints of “bedtime problems” or “nightwakings" rather than “insomnia” as the chief concern. The etiology and management of the most common causes of bedtime resistance (e.g., inadequate limit setting) and nightwakings (e.g., inappropriate sleep-onset associations) are discussed in Chapters 7 and 8 (Bedtime Problems and Nightwakings). The focus of this chapter is on the more traditional definition of insomnia that was formerly termed “psychophysiologic" or “primary" insomnia; that is, difficulties in initiating or maintaining sleep that are typically seen in older children and adolescents.

With the release of the DSM-5 and the ICSD-3, there is considerably more consistency in the definition of insomnia disorder across the two nosologies, now called insomnia disorder in DSM-5 and chronic insomnia disorder in ICSD-3. The diagnostic criteria are quite similar (see Tables 19.1 and 19.2) and both require symptoms to be present at least 3 times per week for at least 3 months. The ICSD-3 also includes short-term insomnia disorder, which involves
identical diagnostic criteria except that the symptoms have been present for less than 3 months. For simplification purposes, the term **insomnia** will be used throughout this chapter to refer to the diagnosis of **insomnia disorder** or **chronic insomnia disorder**.

### TABLE 19.1. Diagnostic Criteria (ICSD-3): Chronic Insomnia Disorder

<table>
<thead>
<tr>
<th>A.</th>
<th>The patient reports, or the patient's parents or caregiver observes, one or more of the following:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Difficulty initiating sleep</td>
</tr>
<tr>
<td>2.</td>
<td>Difficulty maintaining sleep</td>
</tr>
<tr>
<td>3.</td>
<td>Waking up earlier than desired</td>
</tr>
<tr>
<td>4.</td>
<td>Resistance going to bed on appropriate schedule</td>
</tr>
<tr>
<td>5.</td>
<td>Difficulty sleeping without parent or caregiver intervention</td>
</tr>
<tr>
<td>B.</td>
<td>The patient reports, or the patient's parents or caregiver observes, one or more of the following related to the nighttime sleep difficulty:</td>
</tr>
<tr>
<td>1.</td>
<td>Fatigue/malaise</td>
</tr>
<tr>
<td>2.</td>
<td>Attention, concentration, or memory impairment</td>
</tr>
<tr>
<td>3.</td>
<td>Impaired social, family, occupational, or academic performance</td>
</tr>
<tr>
<td>4.</td>
<td>Mood disturbance/irritability</td>
</tr>
<tr>
<td>5.</td>
<td>Daytime sleepiness</td>
</tr>
<tr>
<td>6.</td>
<td>Behavioral problems (e.g., hyperactivity, impulsivity, aggression)</td>
</tr>
<tr>
<td>7.</td>
<td>Reduced motivation/energy/initiative</td>
</tr>
<tr>
<td>8.</td>
<td>Proneness for errors/accidents</td>
</tr>
<tr>
<td>9.</td>
<td>Concerns about or dissatisfaction with sleep</td>
</tr>
<tr>
<td>C.</td>
<td>The reported sleep-wake complaints cannot be explained purely by inadequate opportunity (i.e., enough time allotted for sleep) or inadequate circumstances (i.e., the environment is safe, dark, quiet, and comfortable) for sleep.</td>
</tr>
<tr>
<td>D.</td>
<td>The sleep disturbance and associated daytime symptoms occur at least 3 times per week.</td>
</tr>
<tr>
<td>E.</td>
<td>The sleep disturbance and associated daytime symptoms have been present for at least 3 months.</td>
</tr>
<tr>
<td>F.</td>
<td>The sleep-wake difficulty is not better explained by another sleep disorder.</td>
</tr>
</tbody>
</table>

*ICD-9-CM: 307.42; ICD-10-CM: F51.01


### TABLE 19.2. Diagnostic Criteria (DSM-5): Insomnia Disorder (780.52; G47.00)

<table>
<thead>
<tr>
<th>A.</th>
<th>A predominant complaint is dissatisfaction with sleep quantity or quality, associated with one or more of the following symptoms:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Difficulty initiating sleep (In children, this may manifest as difficulty initiating sleep without caregiver intervention.)</td>
</tr>
<tr>
<td>2.</td>
<td>Difficulty maintaining sleep, characterized by frequent awakenings or problems returning to sleep after awakenings (In children, this may manifest as difficulty returning to sleep without caregiver intervention.)</td>
</tr>
</tbody>
</table>

http://obgynebooks.com
A hallmark of insomnia is excessive worry about sleep and an exaggerated concern regarding the potential daytime consequences. In addition, there is physiological arousal, which can be in the form of cognitive hypervigilance such as racing thoughts; in many individuals with insomnia an increased baseline level of arousal is further intensified by this anxiety about sleeplessness.

**Insomnia**

Insomnia involves difficulty falling asleep and/or maintaining sleep, including early morning awakenings. Insomnia is typically accompanied by learned sleep-preventing associations and physiological arousal, resulting in a complaint of sleeplessness and decreased daytime functioning. In many cases, insomnia may be comorbid with another sleep or medical disorder.

**Epidemiology**

- **Prevalence:** Recent studies on insomnia in adolescents found that 9% to 24% experience chronic insomnia, with up to 35% experiencing insomnia at least several times a month. Studies in school-aged children (ages 5-12) have reported prevalence rates as high as 30% when based on parental report of difficulty falling asleep or staying asleep.

- **Gender:** Studies indicate that insomnia has an increased prevalence in girls, both in school-aged children and adolescents, consistent with adults.

- **Lower socioeconomic status:** Low socioeconomic status is associated with increased insomnia. Some studies indicate increased prevalence in Hispanic and Black youth, whereas other studies have found minimal differences in the prevalence of insomnia across ethnic or racial groups.

**Etiology and Risk Factors**

There are a number of theories as to the mechanism of insomnia. Factors that have been studied include physiologic arousal, emotional arousal, cognitive arousal, and faulty conditioning; however, empirical evidence is inconclusive. The two factors that are the most substantiated are the following:

- **Sleep behaviors (maladaptive sleep habits),** including excessive time in bed, irregular sleep-wake
schedules, and daytime napping.

- **Sleep cognitions** (beliefs and attitudes about sleep and the possible consequences), such as “I'll never be able to fall asleep tonight,” and “If I can't fall asleep, I won't be able to get up in the morning, I'll miss taking the test in my first class, and I'll fail the course”.

Insomnia most likely results from a combination of **predisposing factors** (such as genetic vulnerability and underlying medical or psychiatric conditions) with **precipitating factors** (acute stress) and **perpetuating factors** (poor sleep habits, caffeine use, maladaptive cognitions about sleep).

In adults, a number of predisposing risk factors for insomnia have been identified:

- **Personality traits:**
  - Hyperarousal (e.g., racing thoughts, increased vigilance)
  - Obsessional thinking style (“I can never fall asleep easily.”)
  - Repression of emotions (“I won't let that bother me.”)

- **Medical and psychological problems:** These include congestive heart failure, chronic obstructive pulmonary disease, anxiety disorders, and depression.

- **Gender:** Women are more at risk for developing insomnia.

- **Family history:** It is more likely that the insomnia is based on learned maladaptive sleep habits, but there may be a genetic contribution in some cases.

### PRESENTATION AND SYMPTOMS

- **Sleep complaints:** The insomnia may present as difficulty falling asleep, difficulty maintaining sleep, or, less commonly, early morning awakenings. Thus, overall sleep efficiency (time asleep divided by time in bed) is reduced. Difficulties initiating or maintaining sleep are typically defined as greater than 20 to 30 minutes.

- **Learned sleep-preventing associations:** Insomnia, by definition, involves learned sleep-preventing associations. These may present as:
  - Excessive daytime worrying about being unable to fall asleep or stay asleep.
  - Trying too hard to fall asleep at bedtime, although sleep comes easily at other times (e.g., while watching television).
  - Conditioned association of being in bed “awake” instead of “asleep.” Interestingly, individuals with insomnia may fall asleep more easily in other environments, such as in the living room or in the sleep lab, that do not have the same conditioned associations of anxiety and wakefulness with the bed and bedroom.
  - Increased somatic tension related to sleep, including agitation or muscle tension.

- **Decreased daytime functioning:** Complaints often include subjective cognitive impairment, mood changes, and poor functioning.

### Diagnostic Criteria

See Tables 19.1 and 19.2

**Short-term or Situational Insomnia**
Short-term or situational insomnia typically occurs in children and adolescents with previously normal sleep and lasts a few days to a few weeks. Short-term insomnia can be the result of sleeping in an unfamiliar or nonconducive sleep environment (e.g., too noisy, too hot), stressful life event (e.g., start of school), disruption of sleep schedule (e.g., trip, jet lag), or an illness.

Associated Features

- **Depression**, changes in mood, and decreased sense of well-being. Studies also indicate that insomnia is a significant risk factor for future depression, and that persistent insomnia may predict depression relapse. Suicidal ideation and suicide attempts are also associated with insomnia. Finally, studies indicate that insomnia is a significant predictor of current and future suicidal ideation, suicide attempts, and suicide completion.

- **Daytime fatigue**, including reduced energy, although rarely excessive daytime sleepiness.

- **Poor school performance**, related to decreased mood and fatigue.

- **Excessive caffeine use**, to maintain wakefulness and combat daytime fatigue.

- **Alcohol, marijuana, and other drug use** are associated with insomnia. Adolescents may use marijuana or alcohol as a sleep aid.

- **Hypnotic use**, including prescription or over-the-counter hypnotics (e.g., antihistamine preparation), is common in adults with insomnia and may be present in adolescent and college-aged patients.

Insomnia during adolescence is a significant risk factor for current and future development of depression and suicide.

**EVALUATION**

- **Medical history**: A thorough medical history should include evaluation of other possible causes or comorbidities of sleep-onset and maintenance difficulties, including sleep disorders (e.g., OSA, RLS), acute and chronic medical disorders (especially pain conditions), psychiatric disorders, and alcohol and drug use. Concurrent medications should be reviewed as possible contributory factors, especially psychostimulants (see Chapter 20). Caffeine use and cigarette smoking may also play a role.

- **Developmental and school history**: The history is generally normal. Children and adolescents with insomnia may be anxious or depressed. Many are academic “overachievers.” This situation may lead to a cycle of heightened anxiety about the effects of the insomnia on academic performance and even more difficulty falling asleep.

- **Family history**: Symptoms are often present in first-degree relatives, which may be an indication of genetic vulnerability and/or environmental modeling.

- **Behavioral assessment**: An assessment may reveal mood disturbances and anxiety disorders.

- **Physical examination**: The examination is usually unremarkable.

- **Diagnostic tests**: 
  - **Sleep diaries** will reveal prolonged sleep onset and/or nighttime awakenings and/or early morning
awakenings. Sleep diaries often yield important additional information on maladaptive bedtime activities and behaviors when insomnia is suspected. Sometimes, sleep diaries may indicate that the patient's subjective perception of sleep difficulty is actually significantly worse than the sleep log evidence would suggest (termed “sleep state misperception”).

- **Polysomnography** is rarely indicated in the evaluation of insomnia unless an additional underlying sleep disorder is suspected.

- **Actigraphy** can provide documentation of prolonged periods of wakefulness. Alternatively, some individuals with insomnia have “sleep state misperception”; i.e., they tend to perceive and report getting less sleep than they actually do, and having more and longer awakenings than can be objectively documented. In the latter situation, actigraphy can confirm that the patient is actually sleeping better than assumed, which may help to alleviate anxiety. Actigraphy is also beneficial when parents are unclear as to the severity of sleeplessness, either overestimating or underestimating sleep.

**DIFFERENTIAL DIAGNOSIS**

Insomnia should be differentiated from the following:

- **Delayed sleep-wake phase disorder (DSWPD):** DSWPD is characterized by few or no difficulties falling asleep at the individual's preferred (later) bedtime, in contrast to a patient with insomnia who typically complains of delayed sleep onset no matter what time he or she goes to bed. In addition, when allowed to choose their own schedule, patients with DSWPD typically fall asleep and wake at consistent times (see Chapter 18).

- **RLS and periodic limb movement disorder (PLMD):** Individuals with RLS (see Chapter 16) often present with difficulty falling asleep at bedtime, whereas middle-of-the-night wakings can be associated with PLMD. The key differentiation between insomnia and these disorders is the reporting of an almost irresistible urge to move the legs that is often accompanied by uncomfortable sensations when trying to fall asleep (RLS) and restlessness or leg movements (PLMD).

- **OSA:** Sleep-disordered breathing may result in difficulties falling asleep and maintaining sleep given the frequent arousals that result from the apneic pauses. Children and adolescents with sleep-disordered breathing will likely have additional symptoms, such as snoring, noisy breathing, breathing pauses, and restlessness (see Chapter 15).

- **Poor sleep practices:** Poor sleep practices (see Chapter 5), including maintaining an erratic sleep schedule or the use of caffeine or other substances, and inadequate sleep can contribute to difficulties falling and staying asleep. Patients with insomnia often continue to experience sleep disturbances even after maintaining healthy sleep practices. In those with insomnia, there is typically conditional arousal to the sleep setting.

- **Lifestyle issues:** Practices such as staying up late to socialize, do schoolwork, or engage in other activities (e.g., television viewing) are voluntary behaviors that may result in a child or adolescent habitually going to bed late.

- **Sleep-disruptive environmental issues:** Bedrooms that are not conducive to sleep, due to temperature, light, or noise, may result in sleep difficulties. Sharing a room with others who are awake during the night or snore may also contribute to sleep problems, as can sleeping in a place that is not considered safe or there is a threat of imminent harm.

- **Short sleepers:** Some individuals require less sleep, although the level of individual variability in
children and adolescents is unknown. Those who require less sleep do not experience impairments in daytime functioning.

COMORBID CONDITIONS
Insomnia is often comorbid with other conditions and the risk relationship is often bidirectional, with insomnia increasing the risk of medical/psychiatric conditions and medical/psychiatric conditions increasing the risk of insomnia. The direction of the relationship may not always be clear and it may change over time. For example, caregivers often are concerned that sleep problems are causing a psychiatric issue, such as depression, but it may not be that clear cut.

With the release of DSM-5 and ICSD-3, insomnia is no longer considered secondary to other medical or psychiatric disorders. Furthermore, research shows that the insomnia even if it develops as a result of another condition typically has its own course and may remain even when the comorbid condition is treated and resolves. Treatment of the insomnia may result in improvements with the sleep difficulties as well as the comorbid condition. An insomnia diagnosis would apply when the insomnia symptoms occur independently from the comorbid medical or psychiatric disorder or when the insomnia symptoms persist following resolution of the co-occurring sleep, medical, or psychiatric issue.

- **Psychiatric disorders (e.g., depression, anxiety):** Insomnia is often comorbid with depression and anxiety. In cases of depression, other symptoms including depressed mood, anhedonia, and loss of appetite will be present. In generalized anxiety disorder, which often develops in school-aged children and is associated with insomnia, the excessive anxiety and worry will be reported both at bedtime and throughout the day, and daytime functioning is impaired. Note that the anxiety that is often associated with insomnia is specifically related to the inability to sleep, whereas anxiety associated with generalized anxiety disorder is more global and diffuse. Separation anxiety disorder may also be present, with sleeplessness occurring at night when the child is separated from attachment figures.

- **Medical conditions:** Medical disorders are frequently comorbid with insomnia (see Chapter 22 for a review of sleep and medical disorders).

MANAGEMENT

When to Refer
Children or adolescents with insomnia who also present with symptoms of other underlying sleep disrupters (e.g., OSA, RLS) should be referred to a sleep specialist. Children or adolescents whose insomnia fails to respond to simple behavioral interventions may also benefit from referral to a sleep clinic or behavioral medicine specialist with expertise in treating sleep problems, particularly cognitive-behavioral therapy. If there are concerns regarding psychiatric or psychological issues, a referral to a mental health specialist is warranted.

Treatment
A thorough evaluation for possible causes of and contributing factors to insomnia is critical prior to the development of a treatment plan. The following treatment strategies can be effective with primary insomnia. These strategies can also be beneficial in cases of comorbid insomnia.

- **Healthy sleep practices:** Maintenance of healthy sleep practices (see Chapter 5 and patient handouts in Appendices C7 and C8) is critical with insomnia, including the following:

  - **Developmentally appropriate bedtime,** as a bedtime that is too early can lead to delayed sleep
onset

- **Consistent sleep schedule**, on weekdays, weekends, and school vacations
- **Avoidance of naps**, as daytime sleep can contribute to difficulties falling asleep at bedtime
- **Avoidance of caffeine**, especially in the afternoon and evening
- **Sleep-conducive environment**, including cool, dark, and quiet
- **Removal of electronics from the bedroom and avoidance of late-evening (i.e., after 9 p.m.) screen use**, including television viewing, computer use, video gaming, cell phones, and e-reader use, as these devices can be both mentally and emotionally stimulating, and suppress melatonin via input of blue light in particular to the retina
- **Bedtime routine**, that is consistent night to night and relaxing
- **Consistent morning waketime**, regardless of the prior night’s sleep, to regulate the internal clock and synchronize the sleep-wake cycle

**Cognitive-behavioral treatment:** The goal of behavioral interventions is to disrupt the negative learned associations that are often a prominent feature of insomnia. These include the following:

- **Stimulus control:** Stimulus control involves discontinuing any activities in bed that are not conducive to sleep (including watching television, using a computer, and worrying), as these activities often serve as cues for wakefulness rather than sleepiness. Rather, the bed and bedroom should be associated with sleep only and a consistent sleep-wake schedule should be established. The patient should only go to bed when sleepy, so bedtime should be delayed until he or she is sleepy (e.g., yawning, eyes drooping). Once in bed, the patient should get out of bed if not asleep within 15 to 20 minutes, and engage in a quiet nonstimulating activity (e.g., reading, folding laundry). When again drowsy, the patient should return to bed. It may be necessary to repeat this cycle several times. It is critical for the patient to leave the bed (and possibly bedroom) if unable to fall asleep or else the cycle will continue, with the bedroom and bed associated with sleeplessness. In addition, the patient should only use the bed and bedroom for sleep, should get up at the same time every day, and should avoid all napping.

- **Sleep restriction:** Restriction of the time in bed to a minimum of hours, usually 6 to 7, will increase sleep efficiency, consolidate sleep, and disrupt the learned association of sleeplessness and being in bed. Consistent and accurate recording of sleep patterns in a sleep diary is an essential component of sleep restriction therapy. To begin, the amount of time in bed should be set to the estimated amount of nighttime sleep at baseline. Once the sleep efficiency (total sleep time and time in bed) is greater than 85%, a schedule of increasing time in bed (which is gradual enough to maintain the sleep efficiency above 85%) is instituted over a period of days to weeks. However, given the potential for daytime impairment, sleep should never be restricted to less than 6 hours in children and adolescents.

- **Cognitive restructuring:** This cognitive-behavioral technique involves teaching the patient to counter inappropriate thoughts, using a three-step process: (1) identifying the inappropriate sleep cognition, (2) challenging the validity of each sleep cognition, and (3) replacing the thought with a more productive one.

- **Relaxation:** Relaxation strategies, primarily progressive muscle relaxation as well as deep breathing, visual imagery, and meditation, can be beneficial.

- **Medication:** Given that no medications are FDA-approved for insomnia in children or adolescents, hypnotics are typically not recommended as a first-line intervention in this age group for the treatment of insomnia. In specific circumstances, hypnotics may be necessary, but should always be used in combination with healthy sleep practices and behavioral interventions. A complete
Seven Rules for Beating Insomnia

1. **Choose a set wake-up time.** Wake up at the same time every day (although weekend days can be 1 to 3 hours later depending on school schedules), no matter how much sleep you got the night before.

2. **Choose a bedtime.** Choose the earliest possible bedtime that is late enough that you are sleepy but is not too early so it doesn't let you be in bed too long. You only want to spend the amount of time in bed that you actually need for sleep.

3. **Go to bed when you are sleepy, but not before your chosen bedtime.** Don't go to bed until you are sleepy. So, if you are still wide awake at your chosen bedtime, wait a while longer until you are sleepy enough to fall asleep quickly.

4. **Get out of bed when you can't sleep.** If you are lying in bed and can't sleep, get out of bed and do something relaxing out of the bedroom such as reading a book. Go back to bed when you feel sleepy enough to fall asleep quickly. Again, if you do not fall asleep quickly, get up. Keep repeating this cycle until you fall asleep. You need to get out of bed when you can't sleep both at bedtime and in the middle of the night.

5. **Don't worry or plan in bed.** When lying in bed at night, don't spend the time worrying or planning for the next day. Set aside another time of the day to do these things. If you automatically start thinking and worrying when you get in bed, get up and don't head back to bed until your thoughts won't interfere with falling asleep. Thinking in bed is a habit, and one that you can break.

6. **Only use your bed for sleep.** Don't do anything but sleep in your bed. That is, don't do other activities, such as watch television or do homework.

7. **Avoid naps.** Naps will interfere with your ability to fall asleep at bedtime, so no naps.

Adapted from Owens JA, Mindell JA. *Take charge of your child's sleep.* New York: Marlowe, 2005.

**PROGNOSIS**

Research has not been conducted on the long-term outcome of insomnia in children or adolescents. However, given the persistence of some underlying predisposing factors for insomnia, such as certain personality traits, some individuals are clearly at risk for recurrent problems. In addition, the learned nature of the disorder makes it likely that insomnia will persist if untreated.

**Tips for Talking to Parents (and Children/Adolescents)**

1. Distinguish between insomnia as a symptom and insomnia as a diagnosis.
2. Discuss the “3Ps” of insomnia: predisposing, precipitating, and perpetuating factors.
3. Explain the learned nature of insomnia and the sleep related behaviors that are often initiated to relieve insomnia (e.g., napping, excessive time in bed) but may actually exacerbate the problem.
4. Explain the conditioned association between bed and wakefulness and anxiety in individuals with insomnia.
5. Develop appropriate healthy sleep practices.
6. Discuss treatment options including behavioral interventions, cognitive therapy, and, in select cases,
hypnotic use.

See Appendix D16 for a parent handout on insomnia.
MEDICATIONS FOR SLEEP

Most sleep disturbances in children and adolescents are appropriately managed with behavioral therapy alone; however, there may be clinical situations in which pharmacologic intervention or a combination of behavioral and pharmacologic intervention is considered by the pediatric practitioner for a child or adolescent with significant difficulties in initiating and/or maintaining sleep (medication use for specific sleep disorders such as restless legs syndrome and narcolepsy are covered in their respective chapters). There are a variety of over-the-counter (OTC) and prescription medications in the category of sedatives and hypnotics that are used in clinical practice by healthcare practitioners, as well as by parents, to treat pediatric insomnia. However, it is important to note that very little empirical data regarding the efficacy, safety, and tolerability of pharmacologic interventions for insomnia exist in the pediatric population. There are currently no sleep medications including hypnotics approved for use in children under the age of 16 years by the Food and Drug Administration (FDA).

It should be emphasized in the context of the following discussion of medication use that insomnia is a descriptive rather than a diagnostic term and does not specify etiology. There are many possible causes for the same constellation of symptoms typically presenting as childhood insomnia (e.g., bedtime resistance or difficulty initiating sleep and nightwakings), including medical (e.g., drug-related or pain-induced or from primary sleep disorders such as obstructive sleep apnea [OSA]) and behavioral (e.g., associated with unhealthy sleep habits or negative sleep-onset associations) issues, or a combination of these factors. Therefore, it is imperative that the choice of therapy, medication or otherwise, be diagnostically driven; this in turn implies that the clinician has a systematic evaluation strategy to assess the nature of the presenting complaints and potential etiologies and to narrow down the potential differential diagnoses.

A host of variables impact the appropriateness of medication use in a given clinical situation. These include the following:

- **Patient variables**, such as age, presence of comorbid psychiatric and developmental conditions (e.g., attention deficit hyperactivity disorder [ADHD], autism or pervasive developmental disorder, blindness or severe visual impairment), presence of chronic medical conditions (e.g., chronic pain) or acute medical conditions, and hospitalization.

- **Provider and practice setting variables**, including provider familiarity with behavioral treatment strategies, time, and reimbursement issues.

- **Cultural and societal variables**, such as acceptance of psychotropic use in children in general and acceptance of alternative therapies.

Alternatively, there are a number of reasons why practitioners might be reluctant to prescribe or recommend medications for sleep problems in children:

- **Concerns about efficacy**, particularly given the lack of empirical data regarding effectiveness of these drugs in children.

- **Concerns about tolerance**, including short- and long-term side effects, as well as rebound insomnia once the medication is discontinued.

- **Concerns about safety**, including dependency issues, effects on sleep architecture, and risk of accidental overdose.

- **Ethical considerations**, such as medicating a “parent problem” and giving the “wrong message” to families about management of sleep issues.

Unfortunately, concerns regarding safety and efficacy are somewhat justified, as sleep disturbances remain one of the most poorly researched areas in pediatric psychopharmacology. Most of the information available regarding use of these medications is taken from adult data. There have been only a handful of studies that have examined the effectiveness of hypnotic and sedative use in children and adolescents using the “gold standard” of randomized placebo-controlled clinical trials. The empirical evidence that does exist primarily comes from case reports or small case series. Despite this, a number of studies both in the United States and Europe suggest that prescribing or recommending sedatives and hypnotics for sleep complaints is relatively common practice among pediatricians and general practitioners as well as child psychiatrists. These studies also suggest that even in infants and preschoolers, sleep complaints often dominate the presenting symptoms for which psychotropic medications in general are prescribed.

**Sleeping Pills for Sleepless Nights?**

Selection of specific intervention strategies for children and adolescents with insomnia, including behavioral treatment and pharmacologic management, must first and foremost be diagnostically driven. Specific pharmacokinetic (PK) and pharmacodynamic (PD) properties of available sedative and hypnotics should be considered in the context of the given clinical situation. Treatment goals should be clearly outlined and the emergence of side effects carefully monitored.

Faced with what is admittedly inadequate empirical support for the use of sleep medications in children and adolescents coexisting with what is sometimes a pressing clinical need, how does the pediatric practitioner help families make rational choices regarding medication use?

In addition to considering the factors listed above, some additional considerations should be kept in mind:

- **Careful evaluation of the causes of and contributing factors to a sleep disturbance** in the individual child or adolescent is key. First and foremost, selection of specific intervention strategies, including behavioral treatment as well as pharmacologic management, must address the underlying etiologies. Diagnostic classification systems, such as the International Classification of Sleep Disorders (ICSD-3), allow sleep disorders in children to be reliably described and diagnosed, which provides a rationale for the use of specific treatment strategies, including pharmacologic ones, in the clinical setting.

- **Thorough evaluation of the impact of the sleep disturbance on the child's health and daily functioning** must be combined with diagnostic assessment. In particular, possible neurobehavioral signs of daytime sleepiness (e.g., mood changes, attention problems, impulsivity, poor school performance) must be carefully assessed.

- **Thorough evaluation of the impact of the sleep disturbance on the family** must also be combined with diagnostic assessment. Parent and family variables that are important to consider include educational level, parenting skills, household composition, parental stress level and caregiver exhaustion, and previous experience with and acceptability of pharmacologic treatment. There are situations, albeit relatively rare, in which families are so exhausted and overwhelmed by a child's sleep problems that concerns about both safety and parental mental health are warranted. This situation is particularly likely to occur in the setting of other developmental and medical issues.

**When to Consider Medications for Sleep**

There may be clinical situations in which a combination of frequency, severity, chronicity, and functional impact on both child and family of the sleep problem; family variables, including stress and parental exhaustion; and characteristics of the child (including comorbid medical, psychiatric, and neurodevelopmental issues) may make it appropriate to consider the use of sedative or hypnotic medication in combination with behavioral therapy and healthy sleep practices for insomnia in children.
Characteristics of the individual clinical situation must be reviewed. These include type and severity of the sleep problem, duration, frequency, and previous failed attempts at conventional behavioral therapy.

Once the decision to include pharmacologic management has been made, additional specific issues to consider include the following:

- In almost all cases, medication is not the first treatment choice nor the sole treatment strategy for children with insomnia; medication should be viewed only within the context of a more comprehensive treatment plan. Empirically supported behavioral interventions (e.g., graduated extinction, positive routines, parent education) are the mainstay and first choice for the treatment of pediatric insomnia. Thus, medication should be used in combination with nonpharmacologic strategies. While pharmacologic interventions are likely to have a more rapid and potent effect, nonpharmacologic treatments have been shown to have more long-lasting effects (i.e., persistent after medication has been discontinued).

- Psychoeducation of the patient and family is key. While the contrary may appear self-evident, medication may be viewed by families as the “silver bullet” solution to their child's sleep problems. A brief and simple explanation of the basics of sleep regulation, including the role of the homeostatic sleep drive, circadian rhythms, and the multiple internal (e.g., hunger, boredom) and external (e.g., noise and light levels) sleep facilitating and inhibiting factors, can often help caregivers and patients to appreciate the complexity of the sleep-wake system and both emphasize the importance of behavioral strategies and minimize the relative importance of the role of medication. A sleep drug should be viewed as one component of an array of potential sleep facilitating mechanisms rather than as a substance that will “put a child to sleep.”

- The institution of appropriate and healthy sleep practices is critical. As a corollary to psychoeducation, sleep behaviors that revolve around the basic environmental (e.g., room temperature), sleep-wake scheduling, sleep practices (e.g., bedtime routine), and physiologic (e.g., caffeine use) factors promoting optimal sleep are a necessary component of the treatment approach to pediatric insomnia (see Chapter 19).

- The presence of both medically based and behaviorally based sleep disorders must be assessed and appropriately addressed. Sleeplessness in children commonly co-occurs with other primary sleep disorders (e.g., OSA, restless leg syndrome [RLS]), leading to increased daytime sequelae. Thus, pharmacologic treatment of insomnia could potentially exacerbate coexisting sleep problems. For example, sedatives and hypnotics with respiratory depressant properties and medications that may cause significant weight gain should be avoided if the insomnia occurs in the presence of OSA. In addition, selective serotonin reuptake inhibitors (SSRIs) should be used with caution in treating insomnia, as they may increase symptoms of comorbid RLS.

- Initiation of a medication trial should be accompanied by planning of an “exit strategy.” There should be definable, realistic, agreed-upon, and measurable outcomes that would provide a clear signal to both the family/patient and the healthcare professional that termination of medication should be considered. Treatment goals should be clearly outlined and measurable (e.g., sleep onset consistently less than 30 minutes, improvement in mood and attentiveness, decrease in subjective distress about the insomnia in caregiver and/or patient). The immediate goal of treatment should be to alleviate or improve, rather than eliminate, sleep problems. Monitoring of both efficacy and side effects should take place frequently and systematically.

- In addition, abrupt discontinuation of sleep medication, especially short and intermediate half-life drugs, should in general be strongly discouraged. Abrupt discontinuation may not only result in “rebound insomnia” but may also increase the exacerbation of other sleep problems. For example, sleep medications that are potent rapid eye movement (REM) suppressants (e.g., SSRIs) when withdrawn without taper may result in a compensatory increase (rebound) in REM sleep and a subsequent increase in REM-related phenomenon such as nightmares.

- Dosing should be initiated at the lowest level likely to be effective and titrated up as necessary. Furthermore, whenever possible, medications should also be used for the shortest possible duration (<1 month). The duration of therapy should be discussed and clarified with the family before initiating medication.

- Rational treatment selection should be based on the clinician's judgment of the best possible match between the clinical circumstances (e.g., type of sleep problem, patient characteristics) and the individual properties of currently available drugs (e.g., onset of action, safety, tolerability). Thus, clinicians should have some degree of familiarity with the pharmacologic profile of the sedatives and hypnotics currently available for use in the pediatric population (see Tables 20.2 and 20.3). Medication selection, particularly in terms of duration of action, should be appropriate for the presenting complaint—that is, for children with sleep-onset problems, a shorter-acting medication is generally desirable, whereas for sleep maintenance problems, longer-acting medications may be considered. Timing and type of medication should minimize “morning hangover” or persistent grogginess. In general, this means choosing an agent with the shortest possible half-life.

- Timing of drug administration is another important consideration relative both to the targeted time of sleep onset (e.g., within 30 minutes of “lights out”) and to the “second wind” phenomenon, in which circadian-mediated alertness in both adults and children typically increases in the evening hours just before sleep onset, making it more difficult to fall asleep during this 1-hour to 2-hour window. Most hypnotic medications have their onset of action within 30 minutes of administration and peak within 1 to 2 hours. Thus, giving the medication too early (e.g., 2 hours before sleep onset) is not only less likely to be effective than dosing closer to bedtime, but may in fact cause disinhibition or even induce dissociative phenomenon (i.e., hallucinations) when administered during the circadian alertness window. Timing should also reflect the individual properties of a given drug. For example, melatonin in a small (0.5 mg) physiologic dose administered 5 to 7 hours prior to habitual sleep onset has a primarily chronobiotic (i.e., sleep phase advancing) effect, while a larger dose (i.e., 3-5 mg) 30 minutes before bedtime acts more as a sedative/hypnotic. Finally, the little pediatric PK and PD data that exist for hypnotic drugs suggest that some medications (e.g., zolpidem) are metabolized differently in younger children, who may require higher dosing than adults. Thus, a dose that is not adequate to actually induce sleep could still potentially result in a “paradoxical” reaction in which the child becomes groggy, and subsequently agitated and disinhibited.

- Avoidance of activities requiring alertness following administration of hypnotic medication should be discussed. While this may seem obvious, not all adolescents, for example, may appreciate the inherent safety issues involved in activities such as driving after taking a sleep medication.

- Potential modifications in dosing and timing of administration should be addressed. It should be clarified with the family whether medication is to be used on a nightly or “as needed” basis. If the latter, clear parameters should be established regarding if and when medication should be administered (e.g., if the child is not asleep within 60 minutes of lights out, middle-of-the-night dosing should be no later than 2 a.m.). Dose escalation should also be specifically addressed to avoid inadvertent overdose, as caregivers may assume that “if one pill doesn't work, then two (or three) might.” This is particularly critical with drugs with a narrow therapeutic index, such as clonidine. In general, dosage increases are best accomplished only after direct communication and consultation with the healthcare provider.

- All medications prescribed for sleep problems should be closely monitored for the emergence of side effects. Side effects should be reviewed with the family, as well as with the child or adolescent as appropriate. Furthermore, discontinuation of these agents may result in increased sleep problems. For example, increased nightmares as a result of REM rebound may occur if a REM-suppressant medication is withdrawn abruptly.

- Medication should also be used with caution in situations in which there may be potential pharmacodynamic drug-drug interactions with concurrent medications (e.g., sedatives and hypnotics and opiates) or PK drug-drug interactions (e.g., fluoxetine, a CYP2D6 and 2C19 inhibitor, and diphenhydramine). In particular, adolescents should be screened for alcohol and drug use, as well as pregnancy, prior to initiation of therapy, as many recreational substances may have synergistic clinical effects when combined with sedatives and hypnotics. In addition, hypnotics with high toxicity levels in overdose should be used with extreme caution in...
situations in which there is any risk of accidental or intentional overdose.

Caregivers and patients should be questioned regarding concurrent use of parent- or self-initiated nonprescription sleep medications (e.g., Tylenol PM, melatonin, herbal preparations) as well as other OTC medications. It should be noted that, in many cases, the use of medication for sleep problems in children is initiated by parents (or by adolescent patients themselves) without the physician’s recommendation or knowledge. Parents may assume that OTC medications commonly utilized to induce sleepiness, such as diphenhydramine, are harmless. In addition, while generally viewed by parents as “safe,” the potential drug-drug interactions between most herbal preparations and sedatives and hypnotics, as well as other medications, are largely unknown and thus should be approached with caution. Lack of knowledge about possible side effects or about interactions of these OTC medications with other drugs may pose a safety threat. For example, parents may not be aware that the active ingredient in Tylenol PM (diphenhydramine) is the same as that in Benadryl. In some cases, OTC sleep medications may interact with other prescription or OTC drugs, and, in others, with an underlying medical condition. Because parents may be embarrassed about the home use of sedative medications, they may be reluctant to share this information with their healthcare provider. In addition, adolescent patients may fail to consider use of OTC sleep aids as “taking any medication” when queried. Thus, maintaining an open and nonjudgmental approach is key to optimal communication and allows for the development of more effective and appropriate treatment strategies in cooperation with the family.

INSOMNIA MEDICATIONS

There are currently no sedative or hypnotic medications approved for use in children by the U.S. FDA.

A summary of pharmacologic and clinical properties of medications that are currently used in the treatment of insomnia in the United States is presented below. Drugs are grouped first by type (OTC versus prescription drugs) and then by pharmacologic category (e.g., benzodiazepines (BZDs), antidepressants). As is standard in the adult insomnia literature, drugs that are FDA approved for insomnia in adults (indicated by an asterisk) are listed first within each category. Finally, prescription medications commonly used off-label for insomnia are listed in alphabetical order, in order to avoid any implied preference in rank.

Because the currently available empirical evidence regarding safety and efficacy of pharmacologic insomnia treatment in children is inadequate to rank recommendations for the use of these medications, the focus is on a description of the drug properties and any specific precautions regarding their use in the pediatric population. Additional details regarding pediatric clinical trial results are provided for certain drugs when available, but this does not imply a preference for these specific medications.

While a detailed discussion of the pharmacology of hypnotic drugs is beyond the scope of this chapter, it is nevertheless useful for the clinician to have some basic understanding of the mechanisms through which these medications work. In general, any drugs that inhibit the wake-promoting and/or increase sleep-promoting neurotransmitters can potentially act as sedative/hypnotics (see Table 20.1). For example, both first-generation “antihistamines,” such as diphenhydramine, and 5-aminobutyric acid (GABA)-aminergic drugs, such as BZDs, are sedating.

A summary of individual medication properties can be found in Tables 20.2 and 20.3. Highlighted below are mechanism of action, effects on sleep architecture, relevant pharmacodynamic and kinetic properties, relevant efficacy data, side effects likely to impact on pediatric populations, and other clinical considerations. Unless specifically indicated, all clinical trial data are from adult populations; pediatric-specific information regarding safety and efficacy is included when available. Most manufacturers do not list pediatric soporific doses for these drugs so that dosing in clinical practice is often based on a relative percentage of the adult dose.

Choosing an Insomnia Medication (American Academy of Sleep Medicine Clinical Guidelines for the Evaluation and Management of Chronic Insomnia in Adults; 2008)

When pharmacotherapy is utilized, the choice of a specific pharmacological agent within a class should be directed by (Consensus):

1. Symptom pattern
2. Treatment goals
3. Past treatment responses
4. Patient preference
5. Cost
6. Availability of other treatments
7. Comorbid conditions
8. Contraindications
9. Concurrent medication interactions
10. Side effects

OTC Medications

Antihistamines

Both prescription (e.g., hydroxyzine) and OTC (e.g., diphenhydramine) antihistamines are the most commonly prescribed and recommended sedatives in pediatric practice. First-generation drugs (diphenhydramine, hydroxyzine, chlorpheniramine) cross the blood-brain barrier and bind to H1 receptors in the central nervous system (CNS); second- and third-generation antihistamines, such as terfenadine and loratadine, are significantly less sedating. They are generally rapidly absorbed, and effects on sleep architecture are minimal. OTC sleep aids contain diphenhydramine or doxylamine, both of which have demonstrated modest efficacy in reducing sleep-onset latency (SOL) in a few clinical trials, including in adults with psychiatric disorders. A double-blind placebo-controlled pediatric study of diphenhydramine HCL (1 mg/kg) showed significant subjective improvement in sleep latency and nightwakings, although a more recent study in 6- to 15-month-olds found that diphenhydramine was no better than a placebo in reducing nightwakings. Potential side effects
effects include daytime drowsiness, anticholinergic effects (e.g., dry mouth, blurred vision, urinary retention), and paradoxical excitation; fatal overdoses with diphenhydramine have been reported in infants. It should also be noted that tolerance to antihistamines tends to develop, necessitating increasing doses. Parental and provider familiarity tend to make antihistamines a more acceptable choice for many families, and widespread clinical experience indicates that these medications are largely well tolerated in children.

### TABLE 20.1. Sleep- and Wake-Promoting Neurotransmitters

<table>
<thead>
<tr>
<th>Wake-Promoting Neurotransmitters</th>
<th>Sleep-Promoting Neurotransmitters</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Glutamate</td>
<td>• Acetylcholine (REM)</td>
</tr>
<tr>
<td>• Acetylcholine</td>
<td>• GABA</td>
</tr>
<tr>
<td>• Dopamine</td>
<td>• Galanin</td>
</tr>
<tr>
<td>• Norepinephrine</td>
<td>• Adenosine</td>
</tr>
<tr>
<td>• Serotonin</td>
<td>• Glycine</td>
</tr>
<tr>
<td>• Histamine</td>
<td>• Melatonin</td>
</tr>
<tr>
<td>• Hypocretin/Orexin</td>
<td></td>
</tr>
</tbody>
</table>

### TABLE 20.2. Pharmacology of Selected Medications Used for Pediatric Insomnia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Mechanism of Action</th>
<th>Half-Life (h)</th>
<th>Metabolic Pathways</th>
<th>Time to Maximum Plasma Concentration (min)</th>
<th>Drug-Drug Interactions (pharmacokinetic and dynamic)</th>
<th>Sleep Architecture Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diphenhydramine</td>
<td>Antihistamines</td>
<td>H1 subtype receptor agonists; first-generation drugs cross blood-brain barrier</td>
<td>4-6</td>
<td>Hepatic</td>
<td>Rapid absorption and onset of action;</td>
<td>ETOH/CNS depressants (barbiturates, opiates)</td>
<td>Decrease SOL; may impair sleep quality</td>
</tr>
<tr>
<td>Brompheniramine</td>
<td></td>
<td></td>
<td>4-6</td>
<td>Peak levels</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chlorpheniramine</td>
<td></td>
<td></td>
<td>4-6</td>
<td>2-4 h</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydroxyzine (Atarax)</td>
<td></td>
<td></td>
<td>6-24</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Melatonin7</td>
<td>Hormone analogue</td>
<td>Main effect suprachiasmatic nucleus; weak hypnotic (may have GABA-receptor effects)</td>
<td>30-50 min.</td>
<td>Hepatic</td>
<td>30-60 (sustained release peak level 4 h)</td>
<td>Largely unknown; NSAD’s ETOH, caffeine, BZD’s may interfere with normal melatonin production</td>
<td>Decreases SOL; main effect on circadian rhythms</td>
</tr>
<tr>
<td>Flurazepam (Dalmane)</td>
<td>BZD receptor agonists</td>
<td>Bind to central GABA (γ-aminobutyric acid) receptors</td>
<td>47-100</td>
<td>CYP450 3A oxidation</td>
<td>30 min-13 h</td>
<td>CYP450 3A inhibitors (fluoxetine/grapefruit juice) increases levels</td>
<td>Suppress SWS; reduce frequency of nocturnal arousals</td>
</tr>
<tr>
<td>Quazepam (Doral)</td>
<td></td>
<td></td>
<td>25-84</td>
<td>CYP450 3A oxidation/glucuronidation</td>
<td>60-180</td>
<td>ETOH/barbiturates increase CNS depression</td>
<td></td>
</tr>
<tr>
<td>Temazepam (Restoril)</td>
<td></td>
<td></td>
<td>4-18</td>
<td>CYP450 3A oxidation</td>
<td>60-90</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Estazolam (ProSom)</td>
<td></td>
<td></td>
<td>10-24</td>
<td>CYP450 3A oxidation</td>
<td>120</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Triazolam (Halcion)*

Zolpidem (Ambien/Ambien-CR)*

NonBZD receptor agonists (BzRA):

BZD-like 2.5-3 CYP450 oxidation/aldehyde oxidation 90 ETOH, CNS depressants may potentiate effects Decrease SOL, little effects sleep architecture

Zaleplon (Sonata)*

Eszopiclone (Lunesta)*

Ramelteon (Rozerem)*

Synthetic melatonin receptor agonist Selective affinity MT1, MT2 receptors 1-2.6 CYP1A2 (CYP3A4/CYP2A9) 45 Avoid use with P1A2 inhibitors (fluvoxamine) Decreases SOL; no effect NW

Doxepin (Silenor) TCA Selective H1 antagonist 15.3 Primarily metabolized by hepatic cytochrome P450 3.5 h Cimetidine, ETOH, CNS depressants Reduced WASO

Suvorexant (Belsomra) Orexin receptor antagonist 12 Primarily by CYP3A 2 h The recommended dose 5 mg when used with CYP3A inhibitors and the dose generally should not exceed 10 mg; ETOH, CNS depressants Reduced SOL/WASO; increased TST

Clonidine (Catapres) α-agonists α-adrenergic receptor agonists; (guanfacine more selective) decrease NE release 6-24 17 50%-80% of dose excreted unchanged in urine Rapid absorption; bioavailability 100%; onset action within 1 h; peak effects 2-4 h Decreases SOL; reduce REM, SWS

Trazodone (Desyrel) Atypical antidepressant 5-HT, serotonin agonist Biphasic; first T1/2 3-6 h; second T1/2 5-9 h 10-36 h post-ingestion CYP450/CYP2D6 30-120 Potentiates effects ETOH, CNS depressants, digoxin, phenytoin, antihypertensives Decreases SOL, improves sleep continuity, decreases REM, increases SWS

FDA approved as hypnotic in adults.

SWS, slow-wave sleep (stage N4); SOL, sleep-onset latency; BZD, benzodiazepine; NSAID, nonsteroidal anti-inflammatory drug; ETOH, ethyl alcohol; CNS, central nervous system; NW, nightwakings; REM, rapid eye movement.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Adult Dosing Range (mg/d)</th>
<th>Formulation</th>
<th>Side Effects</th>
<th>Development Tolerance/Withdrawal Effects</th>
<th>Safety Profile/Overdose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diphenhydramine</td>
<td>25-100 (should not exceed daily dose 300 mg)/12.5-50</td>
<td>Tablet, capsule, syrup, injectable</td>
<td>Daytime drowsiness, GI (appetite loss, nausea/vomiting, constipation, dry mouth), paradoxical excitation</td>
<td>OD: hallucinations, seizures, excessive stimulation</td>
<td>Weak soporifics; high level parental/practitioner acceptance</td>
<td></td>
</tr>
<tr>
<td>Medicine</td>
<td>Dose Range</td>
<td>Formulations</td>
<td>Side Effects</td>
<td>Usage</td>
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<tr>
<td>Brompheniramine</td>
<td>4</td>
<td></td>
<td>Largely unknown; reported hypotension, bradycardia, nausea, headache; possible exacerbation of comorbid arthritis, asthma</td>
<td>Tolerance does not appear to develop</td>
<td></td>
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<tr>
<td>Chlorpheniramine</td>
<td>4</td>
<td></td>
<td>Largely unknown; reported hypotension, bradycardia, nausea, headache; possible exacerbation of comorbid arthritis, asthma</td>
<td>Tolerance does not appear to develop</td>
<td></td>
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<tr>
<td>Hydroxyzine</td>
<td>25-100; 0.6 mg/kg (children)</td>
<td>Sublingual, liquid, capsule, tablet; various strengths</td>
<td>Tolerance does not appear to develop</td>
<td>Unknown</td>
<td></td>
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<tr>
<td>Melatonin</td>
<td>2.5-5 (range 0.3-25) 0.1-10 (children)</td>
<td>Sublingual, liquid, capsule, tablet; various strengths</td>
<td>Tolerance does not appear to develop</td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Flurazepam*    | 15-30      | Capsules, tablets                                 | Sleep maintenance; also used to reduce partial arousal parasomnias (sleep terrors, sleepwalking); use short half-life BZD for sleep onset; longer half-life for sleep maintenance | Sleep maintenance;
|                |            |                                                   | Marked abuse potential                                                                 | Sleep maintenance                                                      |
| Quazepam*      | 7.5-15     |                                                  | Marked abuse potential                                                                 | Sleep maintenance                                                      |
| Temazepam*     | 7.5-30     |                                                  | Marked abuse potential                                                                 | Sleep maintenance                                                      |
| Estazolam*     | 1-2        |                                                  | Marked abuse potential                                                                 | Sleep maintenance                                                      |
| Triazolam*     | 0.125-0.25 |                                                  | Marked abuse potential                                                                 | Sleep maintenance                                                      |
| Zolpidem*      | 5-10; 6.25-12.5; pediatric study suggests ≥0.25 mg/kg up to 20 mg in 2-18 y | Capsules | Headache, retrograde amnesia; hallucinations; few residual next-day effects | Well tolerated in adults/OD; CNS depression; hypotension |
| Zaleplon*      | 5-20       |                                                  | Headaches, dysgeusia, dizziness; well-tolerated long-term | Limited efficacy in pediatric trials                                  |
| Eszopiclone*   | 1-3        |                                                  | Headaches, dysgeusia, dizziness; well-tolerated long-term | Pediatric clinical trial showed no increased efficacy compared to placebo |
| Ramelteon*     | 8          | Tablets                                           | No significant side effects noted | No significant safety issues identified; no abuse liability |
| Doxepin        | 3 (elderly)-6 mg | Tablets | Nausea, worsening depression | No abuse potential; avoid in severe sleep apnea |
| Suvorexant     | 5, 10, 15, and 20 mg | Tablets | Next-day driving performance impairment (20 mg); risk sleep-driving, other complex behaviors; worsening depression; sleep paralysis, hypnagogic/hypnopompic hallucinations, cataplexy-like | Schedule IV controlled substance |

*Used in children with developmental disabilities, autism, neurologic impairment, blindness; ADHD; jet lag

http://obgynebooks.com
Melatonin

Melatonin is a hormone secreted by the pineal gland in response to decreased light, mediated through the suprachiasmatic nucleus. The mechanism of action of commercially available melatonin is to supplement the endogenous pineal hormone. The plasma level of exogenous melatonin peaks within about 1 hour of administration. Thus, it may be helpful in reducing SOL when taken close to bedtime. Although there is considerably less evidence to support this indication in children, sustained release (controlled release, prolonged release) preparations have been reported in adults, especially in the elderly, to assist in maintaining sleep by increasing sleep continuity and reducing arousals. Clinical uses for melatonin include chronic or acute circadian rhythm disturbances (e.g., delayed sleep-wake phase disorder, jet lag) in normal children and in children with special needs or neurodevelopmental disorders (e.g., blindness, Rett syndrome, autism).

The pediatric literature on the safety and efficacy of melatonin use is fairly robust, particularly compared with other hypnotic drugs frequently used in clinical practice. For example, a number of studies have now demonstrated that melatonin is effective in reducing SOL not only in typically developing children, but specifically in children with ADHD and autism as well. The rationale is based on the premise that at least some of these children have a circadian-mediated phase delay (i.e., delayed sleep onset and offset compared to developmental norms). In addition to effects on circadian regulation of sleep-wake cycles, melatonin has mild hypnotic properties. Relatively long-term studies (up to 4 years) have failed to demonstrate significant adverse effects of melatonin use in a variety of pediatric populations. However, although generally regarded as safe, potential side effects of melatonin include suppression of the hypothalamic-gonadal axis (triggering precocious puberty on discontinuation) and increased reactivity of the immune system in children with immune disorders or on immunosuppressants (i.e., corticosteroids). The bottom line is that although the balance of evidence suggests that melatonin appears to represent the best combination of efficacy and tolerance compared to other OTC and prescription medications for insomnia in both otherwise healthy children and special pediatric populations, without systematic longitudinal follow-up neither claims of safety concerns nor negligible risk can presently be substantiated.

<table>
<thead>
<tr>
<th>Melatonin: Conflicting Opinions</th>
</tr>
</thead>
<tbody>
<tr>
<td>• <a href="http://www.nlm.nih.gov/medlineplus/druginfo">www.nlm.nih.gov/medlineplus/druginfo</a>: “Melatonin should not be used in most children. It is possibly unsafe. Because of its effects on other hormones, melatonin might interfere with development during adolescence.”</td>
</tr>
<tr>
<td>• <a href="http://www.naturalsleep.org">www.naturalsleep.org</a>: “Melatonin, according to more than 24 studies, is safe for children and has been used with little to no side effects.”</td>
</tr>
<tr>
<td>• <a href="http://www.livestrong.com">www.livestrong.com</a>: “Although the use of low doses of melatonin to help children sleep seems to be safe and effective, more research is needed to answer lingering questions.”</td>
</tr>
</tbody>
</table>

In the late 1980s, tryptophan (a precursor of both serotonin and melatonin) supplementation was reported to be associated with eosinophilic myalgia syndrome, but subsequently a contaminant in certain production batches was identified as the underlying cause. While largely anecdotal information suggests that tryptophan seems to have mostly mild and short-term side effects, additional evidence regarding the safety and efficacy of its use in children is needed.

Melatonin is not regulated by the FDA. Therefore, the commercially available formulations may vary in strength and accuracy in regards to concentration. “Pharmaceutical Grade” melatonin, available on the Internet, is produced from pharmaceutical grade ingredients (rather than animal tissue) and may be more reliable. Reported hypnotic bedtime doses for melatonin include 1 mg in infants, 2.5 to 3 mg in older children, and 5 mg in adolescents; use of melatonin in children with special needs has reported doses ranging from 0.5 mg to 10 mg, irrespective of age. It should be noted that studies of melatonin use in adults for advancing sleep phase in delayed sleep-wake phase disorder have reported that smaller doses (e.g., 0.5 mg) 5 to 7 hours before sleep onset may be more effective in treating sleep-onset delay in the context of a circadian rhythm disorder.

Complementary and Alternative Preparations

Most herbal preparations are considered generally safe. However, this is based on only a handful of studies that have suggested that some herbal preparations may be useful for sleep in the pediatric population, and these products in general remain largely untested in children. Valerian root, St. John’s wort, and Humulus lupulus (hops) have been shown to have some evidence of efficacy in adult and/or pediatric studies. Lemon balm, chamomile, and passionflower have limited to no evidence, and kava-kava has been associated with significant safety concerns (e.g., hepatotoxicity).

Valerian root, which has BZD-like properties, has been shown in several studies in adults to have sleep-promoting effects without the “hangover” effects seen with the BZDs, although effects may not be seen for several weeks. Chamomile is reported anecdotally to have mild sedating effects. It should be noted that herbal preparations, including herbal teas and weight loss and laxative preparations, contain a significant percentage of caffeine and thus may disrupt sleep. Lavender appears to have a CNS depressive effect and as such may potentiate the effects of other CNS depressants such as alcohol, aromatherapy with the volatilized lavender oil has been reported to improve sleep quality.

Prescription Insomnia Drugs (Asterisk indicates drug is FDA-approved for insomnia in adults)

Benzodiazepine Receptor Agonists: BZDs*

The hypnotic effect of the BZD is mediated by their action at the GABA<sub>A</sub> receptors: GABA<sub>A</sub>: the major inhibitory neurotransmitter in the brain. These medications shorten SOL and increase total sleep time (TST), and improve non-REM sleep maintenance. Most disrupt slow-wave sleep (SWS). They also have

<table>
<thead>
<tr>
<th>Drug</th>
<th>Usual Doses</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clonidine</td>
<td>0.025-0.3 (up to 0.8) by 0.05 increments (children)</td>
<td>Dry mouth, bradycardia, hypotension, rebound hypertension on discontinuation</td>
</tr>
<tr>
<td>Trazodone</td>
<td>25-50 (up to 150)</td>
<td>Dizziness, CNS overstimulation, Cardiac arrhythmias, hypotension, priapism</td>
</tr>
</tbody>
</table>

ADHD, attention deficit hyperactivity disorder; BZD, benzodiazepine; CNS, central nervous system; GI, gastrointestinal; OD, overdose.
muscle relaxant, anxiolytic, and anticonvulsant properties. The shorter onset of action of some benzodiazepines has led to their use in treating sleep-onset insomnia, while agents with a longer half-life and duration of action have been more commonly used to address sleep maintenance. In general, this class of medication should only be used for short-term or transient insomnia, or in clinical situations in which their other properties (e.g., anxiolytic) are advantageous. There are five benzodiazepines that are FDA approved for the treatment of insomnia in adults (estazolam, flurazepam, quazepam, temazepam, triazolam). All are schedule IV controlled substances.

Of note, BZDs are occasionally used to treat intractable partial arousal parasomnias (e.g., sleep terrors) in children because of their SWS-suppressant effects. However, use of longer-acting BZDs may lead to morning “hangover,” daytime sleepiness, and compromised daytime functioning. Anterograde amnesia and disinhibition may also occur. There is a risk of habituation or addiction with these medications as well as withdrawal phenomena, all of which make them of very limited use in children and adolescents.

Nonbenzodiazepine Receptor Agonists

Nonbenzodiazepine receptor agonists (non-BzRAs) have a more selective profile, binding preferentially to GABAA receptor complexes containing 1 subunits. Effects on sleep architecture appear minimal, although they may increase SWS. In general, these drugs appear to have greater selectivity, an improved safety profile, and lower risk of dependence compared to the BZDs. Different properties of individual drugs make them relatively more suitable for sleep onset and/or maintenance.

- **Zaleplon (Sonata)**. Zaleplon has a very short half-life and as such is primarily used for sleep-onset insomnia. While not FDA approved for middle-of-the-night administration, the short half-life (if at least 4 hours remain before the desired wake time) makes it also potentially useful for this indication. Side effects include dizziness, anterograde amnesia, confusion, and hallucinations. The most common adverse event reported in adults is headaches.

- **Zolpidem tartrate (Ambien)**. Zolpidem also acts by binding selective GABAA receptors; half-life is 2 to 3 hours. Longer duration trials in adults suggest continued hypnotic benefit at 6 months, without the development of tolerance. Disinhibition with any of the BzRAs may occur in the period immediately after taking the medication and hallucinations have been reported. Recent reports of sleep-related events (sleep eating, sleep driving) in adults taking zolpidem have raised additional concerns about its use in children. Although rebound insomnia may occur with either of these compounds, studies in adults suggest that “as needed” administration is well tolerated.

There have been two published clinical trials of zolpidem in the pediatric population. The first was an open-label, dose-escalation, PKs/PDs study in 2- to 18-year-olds. The major findings were that PK measures were not predictive of sleep outcomes and that sleep parameters exhibited considerable variability and potential paradoxical effects. The mean SOL decreased by a modest 5 minutes and TST increased on average by 20 minutes. The recommended maximum dose by the study authors was 20 mg (compared to 10 mg in adults). A subsequent 8-week double-blind randomized placebo-controlled parallel design trial of 201 children 6 to 17 years old with ADHD-related insomnia at a dose of 0.25 mg/kg (maximum dose 10 mg) reported no significant change in mean objective SOL as measured by polysomnogram at week 4 with drug compared to placebo, although there was subjective improvement in insomnia on both the parent and child-reported Clinical Global Impression (CGI) scale in the older (12-17 year old) group at weeks 4 and 8. Although no residual sedation/rebound was reported, adverse events included dizziness and headache. More concerning was a 7.4% incidence of hallucinations, all occurring at lower doses and mostly in boys.

- **Extended release zolpidem tartrate (Ambien-CR)** has a longer half-life, which may make it more useful in sleep maintenance. Initial recommended dose is 6.25 mg in women and 6.25 to 12.5 mg in men. There is no label limitation on duration of use.

- **Zolpidem** also has several alternative delivery systems that may be useful in select clinical situations: zolpidem oral spray (Zolpimist) (5 and 10 mg) and zolpidem tartrate sublingual tablet (Intermezzo) (1.75 mg recommended and maximum dose in women, 3.5 mg in men), which recently became the first FDA-approved medication for middle-of-the-night waking followed by difficulty returning to sleep in adults. The labeling requires that at least 4 hours remain before the planned time of waking.

- **Zaleplon (Lunesta)**. Zaleplon is a GABA-ergic sleep medication that, because of its longer half-life, has been used in adults for both sleep initiation and sleep maintenance. Peak drug concentration occurs at 60 minutes; half-life and clinical effect are approximately 6 hours. Fatty foods tend to delay the absorption. Side effects include unpleasant taste and headache. Interestingly, one study in adults with OSA found that zaleplon increased the arousal threshold and lowered the apnea hypopnea index (AHI) in patients without marked overnight hypoxemia. Abrupt withdrawal with prolonged use (>2 weeks) may be associated with rebound insomnia. Studies in adults have shown no development of tolerance at 6 months.

This medication has been approved for longer-term use, and there is no label limitation on duration of use. Results of a 12-week double-blind randomized controlled trial of eszopiclone for ADHD-related insomnia in children 6 to 11 and 12 to 17 years old (n = 486) at low (1-2 mg) and high (2-3 mg) doses followed by a 12-month open-label safety trial (n = 304) were published in 2014. There was no difference compared to placebo on subjective (latency to persistent sleep or wake after sleep onset on polysomnography) or objective measures (CGI; ADHD rating scales). Adverse events reported included headache, bad taste (dysgeusia), and dizziness. Overall, the drug was well tolerated with 11% discontinuing treatment during the open-label trial due to side effects.

Melatonin Receptor Agonists

Ramelteon (Rozerem) is a synthetic melatonin receptor agonist, acting selectively at the MT1 and MT2 receptors. Its sleep-promoting effect may be related to reduction of the alerting output of the suprachiasmatic nucleus, and it is thought to reduce the circadian-mediated arousal that precedes sleep onset (the so-called “forbidden zone”). It appears to have little interaction with other neurotransmitters. Ramelteon is approved for use in sleep initiation insomnia, and shows moderate efficacy in reducing sleep onset latency (SOL) (in adults). It is absorbed rapidly (0.5-1.5 hours) and a half-life of 1.0 to 2.6 hours (up to 5 hours with the active metabolite). In clinical trials, subjective improvements are typically less consistently reported than are objective improvements in SOL. Side effects may include dizziness and fatigue, as well as mood changes. Next-day effects on functioning, including psychomotor performance, memory, and attention, appear minimal. Ramelteon has been reported to be associated with mild transient increases in prolactin in women with long-term (6 months) administration and decreases in testosterone in older men. Co-administration with fluvoxamine should be avoided. It is a non-scheduled approved hypnotic, with no abuse potential or limitations on duration of use.

Orexin Receptor Antagonists

Suvorexant (Belsomra) was approved by the FDA in August of 2014 for use as needed to treat difficulty in falling and staying asleep (insomnia). Suvorexant is the first approved orexin receptor antagonist, and alters the signaling (action) of orexin in the brain. It is available in four different tablet strengths—5, 10, 15, and 20 mg—and should be taken no more than once per night, within 30 minutes of going to bed, with at least 7 hours remaining before the planned time of waking. Side effects include next-day driving performance impairment at the 20-mg dose level. Similar to other sleep medications, there is also a risk of sleep-driving and other complex behaviors.

Histamine Receptor Agonists

Doxepin (Silenor) is a selective histamine receptor agonist. Due to its long half-life (15.3 hours), it is FDA approved for the treatment of both transient (short-term) and chronic (long-term) insomnia characterized by difficulty with sleep maintenance in both adults and elderly patients. In clinical trials, low-dose doxepin demonstrated maintenance of sleep into the 7th and 8th hours of the night, with no meaningful evidence of next-day residual effects. It appears to have a favorable safety and tolerability profile, with the overall incidence of adverse events comparable to placebo, a low discontinuation rate, and no evidence of tolerance, amnesia, or complex sleep behaviors (e.g., sleep-driving, sleep-eating). Because this drug appears to not have abuse potential, it is not designated as a controlled substance and thus could be useful in...
patients with a history of substance abuse.

Drugs Used Off-Label for Insomnia

α-Agonists

Clonidine, a central α2-agonist that decreases adrenergic tone, is one of the most widely used medications for insomnia in pediatric and child psychiatry practice, particularly in children with sleep-onset delay and ADHD. Despite its widespread use, data regarding safety and efficacy in children with ADHD and sleep problems are limited, although several descriptive and/or retrospective studies have reported adequate clinical response and a relatively low side effect profile. The drug is rapidly absorbed, with an onset of action within 1 hour and peak effects at 2 to 4 hours. Tolerance often develops, necessitating increases in dose. Mid-sleep awakening may also occur as blood levels drop during the night. Effects on sleep architecture are fairly minimal, but may include increased slow wave sleep (SWS) and decreased REM sleep. Thus, direct effects may include an increase in SWS partial arousal parasomnias (e.g., sleep terrors) and REM rebound later in the night with related increase in nightmares. Clonidine has a narrow therapeutic index. Increases in the number of exposures to clonidine over the past several decades have raised concerns about the potential for increased risk of accidental or intentional overdose. TCAs may also exacerbate RLS symptoms.

Tricyclic Antidepressants

In general, antidepressants are believed to mediate sleep promotion by influencing activity of non-GABA neurotransmitters (e.g., histamine, acetylcholine, serotonin) involved in the regulation of sleep and wakefulness. Most antidepressants, especially those with anticholinergic effects, suppress REM and increase latency to REM sleep; abrupt withdrawal may lead to increased nightmares (REM rebound). Although frequently used in clinical practice, there is overall a lack of methodologically rigorous research supporting the use of antidepressants for insomnia in adults or children. Thus, the use of antidepressants for insomnia should take into consideration the presence of concurrent mood issues and whether hypersomnia is part of the clinical picture, as the use of an antidepressant may increase daytime sleepiness. Treating the underlying mood disorder will often result in improved sleep, and successful sleep interventions often result in improvements in mood. The antidepressant dose for insomnia is typically less than the dose used to treat mood disorders.

Second-Generation Antidepressants

Atypical Antidepressants

- Mirtazapine (Remeron) is an 2-adrenergic 5-HT antagonist with a high degree of sedation at a low dose (7.5 mg). It has been shown to decrease SOL, increase sleep duration, and reduce wake after sleep onset in both healthy adults and those with major depression, with relatively little effect on REM sleep. It may, however, result in daytime somnolence.

- Nefazodone (Serzone) is a 5-HT antagonist and norepinephrine reuptake inhibitor that is associated with significant sedation. Side effects include residual daytime sedation, orthostatic hypotension, and weight gain.

- Trazodone is a 5-HT antagonist commonly used in adults and children, particularly in mental health settings. It is one of the most sedating antidepressants because it both inhibits binding of serotonin and blocks histamine receptors. Despite patient reports of improved sleep quality, the empirical evidence supporting the efficacy of trazodone is modest at best. A recent small clinical trial in adults with primary insomnia found fewer nighttime awakenings and subjective report of difficulty sleeping, but that it produced small but significant impairments in short-term memory, verbal learning, and equilibrium. Trazodone has suppressant effects on REM and may increase SWS. It is associated with a number of other significant side effects, including “morning hangover” and reports of priapism in adults in the 50- to 150-mg dose range. In addition, a study of treatment resistant depression in adolescents found that those who had received trazodone plus an SSRI were 6 times less likely to respond to an alternative antidepressant trial and 3 times more likely to express self-harm statements than those on no medication. No similar differences were observed with concurrent administration of other sleep medications.

Serotonin Antagonists and SSRIs

These agents are felt to promote sleep primarily by inhibiting uptake of serotonin. They vary widely in their propensity to cause sedation (e.g., fluvoxamine, paroxetine, citalopram) versus activation with sleep-onset delay and sleep disruption (e.g., fluoxetine, sertraline), although a recent systematic review did not find significant differences among SSRIs in regards to treatment efficacy for insomnia in adults. The most activating SSRIs are often associated with reports of sleep disruption in adults. Newer generation SSRI medications such as citalopram (Celexa) appear to have fewer sleep-disrupting effects and may be useful in the management of insomnia associated with depression. SSRIs suppress REM sleep and often prolong REM onset, while increasing the number of REMs; a characteristic polysomnographic finding in patients on SSRIs is so-called “Prozac eyes” due to this increased REM density. SSRIs also tend to suppress SWS. Most increase SOL and sleep efficiency (time asleep/time in bed). SSRIs frequently are associated with motor restlessness, and may exacerbate preexisting RLS and periodic limb movements.

Additional Medications

Other classes of medications that reportedly have been used in clinical practice for pediatric insomnia include mood stabilizers and anticonvulsants (carbamazepine, valproic acid, topiramate, gabapentin), atypical antipsychotics (risperidone, olanzapine, quetiapine), and chloral hydrate. In most instances, these medications are prescribed for other indications (e.g., bipolar disorder, aggression) but may simultaneously be used for their sedating (i.e., sleep-promoting) properties. It should be emphasized that all of these medications should be used with extreme caution, if at all, for insomnia in children, as there are no or limited data on safety and tolerability for this indication in either adults or children. Furthermore, the sedating effects may interfere with daytime functioning and learning, and tolerance to the effect of decreasing SOL frequently develops, necessitating dosage adjustments. In addition, there may be effects on other sleep parameters; for example, many of the newer atypical antipsychotics
have weight gain as a significant side effect, and thus can worsen sleep-disordered breathing. They also tend to suppress REM sleep and increase motor restlessness during sleep. There have been a handful of case reports/case series reporting effective treatment of insomnia with risperidone in special populations that include children. However, an NIH State-of-the-Science Panel on insomnia treatment in 2005 concluded that antipsychotic “… use in the treatment of chronic insomnia cannot be recommended” due to the high risk-benefit ratio.

Chloral hydrate and barbiturates are not indicated for use in children because of significant side effects. In 1993, the American Academy of Pediatricians recommended that chloral hydrate be used in children for short-term sedation only, because of the risk of hepatotoxicity.

MEDICATION EFFECTS ON SLEEP

Many OTC and prescription drugs have profound effects on sleep in adults. Although these effects are much less well studied in children and adolescents, certain basic pharmacologic principles may be applied to both adults and children. Drugs may exert their effects on sleep in several ways:

- Direct pharmacologic effect on sleep architecture, sleep fragmentation.
- Disturbance in sleep patterns (e.g., nightmares).
- Exacerbation of a primary sleep disorder (e.g., OSA, RLS).
- Drug withdrawal effects.
- Daytime sleepiness.

Understanding a medication’s effects on sleep architecture and sleep-wake regulation (either direct or as a result of medication withdrawal) can help predict the likely clinical effect on sleep.

- Decreased slow wave sleep (SWS) may lead to subjective feelings of “nonrestorative sleep” or the sense of “not being well rested.”
- Increased SWS may increase partial arousal parasomnias, such as sleepwalking, in susceptible individuals.
- Decreased REM sleep may result in impairment of memory consolidation.
- Increased REM sleep may result in increased nightmares and may potentially worsen sleep apnea (as obstructive events are typically increased in REM).

Drug effects on various neurotransmitters of sleep and wakefulness may also be responsible for their clinical effects.

Examples of Drug Effects of Neurotransmitters and Sleep

Wakefulness
- Antihistamines promote sedation, drowsiness

NREM Sleep
- Adenosine receptor blockers (e.g., theophylline, caffeine) disrupt sleep, promote wakefulness
- GABA-receptor agonists (benzodiazepines, zaleplon) promote sleep

REM Sleep
- Nicotinic drugs (e.g., nicotine) promote REM sleep
- Anticholinergic drugs (e.g., TCAs) depress REM sleep

Sleep-Wake Effects of Selected Drugs Used in Pediatrics

Anticonvulsants
Anticonvulsants typically cause dose-dependent sedation, although tolerance may develop.

- Carbamazepine (Tegretol) is associated with increased daytime somnolence and may decrease SOL.
- Phenobarbital tends to be very sedating in a dose-dependent fashion, with some development of tolerance over time. It also decreases SOL.
- Phenytoin (Dilantin) may cause relatively less daytime sleepiness than other anticonvulsants.
- Valproic acid (Depakote) is associated with increased daytime somnolence. There are no reported major direct effects on sleep.
- Other anticonvulsants have varying degrees of daytime sedation. For example, sedation rates with topiramate (Topamax) are 15% to 25%, and with gabapentin (Neurontin) are 5% to 15%; sedation tends to improve over time. Direct effects on sleep are largely unknown. It should be noted that gabapentin, which has effects on dopamine, serotonin, and norepinephrine, appears to increase SWS and has been shown to decrease RLS symptoms. Both carbamazepine and valproic acid may also be used to treat symptoms of RLS.

Allergy and Asthma Medications

- β-Adrenergic may reduce nocturnal arousals.
- Corticosteroid use has been associated with insomnia in asthma and cancer patients as well as subjective increases in wakefulness. Objective documentation of sleep-disrupting effects is less consistent. Corticosteroids decrease REM sleep, although inhaled steroids do not appear to have significant sleep effects.
- Decongestants (such as pseudoephedrine and phenylpropanolamine) may cause insomnia as well as increase plasma caffeine levels, potentiating the stimulant effect of caffeine.
- Theophylline use is associated with increased sleep complaints (difficulty falling asleep, disrupted sleep) in patients with asthma in comparison with other medications. However, alleviation of nocturnal asthma symptoms may improve sleep quality.

Analgesics
Nonsteroidal anti-inflammatory agents have a suppressant effect on nocturnal melatonin secretion and have been shown to decrease sleep efficiency and increase nightwaking. However, improved sleep quality resulting from pain reduction may offset these sleep-disrupting effects. Opioids often result in daytime sedation, which may be persistent. They also disrupt sleep continuity and suppress REM sleep and SWS. Because opioids are respiratory depressants, they may worsen OSA. Abrupt
discontinuation may lead to insomnia and nightmares. Suppression of muscle activity, however, may result in improvement in RLS symptoms.

Cardiovascular Drugs

Antihypertensive agents that are β-antagonists, such as propranolol, may be associated with insomnia and increased nightmares. Other antihypertensives appear to have negligible effects on sleep. Anti-arrhythmic drugs may cause daytime fatigue. For example, digoxin has been associated with both fatigue and insomnia.

Psychotropics

Antidepressants

- **Bupropion (Wellbutrin)** is a norepinephrine and dopamine reuptake inhibitor that increases REM sleep and reduces REM onset latency. Insomnia has been reported in 5% to 19% of adults with the use of bupropion; thus, bupropion should be avoided in patients with preexisting insomnia.

- **Venlafaxine (Effexor)** is a serotonin and norepinephrine reuptake inhibitor that has been associated at medium-level doses with difficulty initiating and maintaining sleep and at high doses with severe insomnia in adults, as well as increased daytime somnolence.

Antipsychotics in general interfere with neurotransmitters regulating sleep and wakefulness, including dopamine, norepinephrine, serotonin, acetylcholine, and histamine. “Traditional” antipsychotics (e.g., thioridazine [Mellaril] and to a lesser extent haloperidol [Haldol]) are often associated with significant daytime somnolence. Newer atypical agents overall tend to be less sedating, but there is some variability among individual agents (clozapine [Clozaril] > olanzapine [Zyprexa] = quetiapine [Seroquel] > risperidone [Risperdal]). Most antipsychotics decrease SOL, increase sleep continuity, and suppress REM sleep (in higher doses). They may also promote sleep by attenuating psychiatric symptoms that interfere with sleep.

- **Lithium** may improve nocturnal sleep, but also increase daytime sleepiness. It tends to increase SWS, with variable suppression of REM sleep.

Psychostimulants

Both amphetamine and methylphenidate may have a direct dose-dependent medication effect that interferes with sleep. Studies have demonstrated an increase in subjective parental perception of severe sleep difficulties, including sleep-onset delay and nightwakings in children on psychostimulants for ADHD. Results of studies assessing objective measures of direct psychostimulant effects on sleep, although less consistent, have found increased sleep onset latency, decreased total sleep time, REM sleep suppression, and variable decreases in SWS. A “rebound” effect late in the day may also result in an increase in evening ADHD symptoms above baseline and increased SOL. Discontinuation of stimulants after prolonged therapy may cause rebound increases in REM sleep, SWS, and subjective sleepiness. The wake-promoting properties of the psychostimulants make them useful agents in the treatment of primary disorders of excessive daytime sleepiness such as narcolepsy. Atomoxetine (Strattera), a nonstimulant medication indicated for ADHD, typically has less effect on sleep initiation than do the stimulants, although an increase in sleep maintenance insomnia has been noted in adults; evening coverage of ADHD symptoms with atomoxetine may reduce bedtime resistance. Other newer nonstimulant medications for ADHD, including extended release preparations of clonidine (Kapvay) and guanfacine (Intuniv), may be associated with daytime somnolence and fatigue compared to placebo.

Other Substances

- **Alcohol** is frequently used by adults with sleep initiation insomnia to hasten sleep onset. However, despite the fact that alcohol decreases SOL by facilitating GABA and inhibiting glutamate, it may also significantly impair sleep continuity and cause sleep disruption. Alcohol has a “biphasic” effect on sleep architecture, in that it increases SWS and suppresses REM sleep in the first part of the night but, as alcohol levels decline, it results in REM rebound, decreased SWS, and sleep fragmentation. The increase in SWS may exacerbate partial arousal parasomnias. REM rebound can increase nightmares, as well as OSA symptoms. Acute withdrawal is associated with increased wakefulness, increased REM sleep, and decreased SWS. Alcohol may also exacerbate comorbid sleep disorders such as OSA and RLS.

- **Caffeine** temporarily increases alertness by blocking the sedative effects of the neurotransmitter adenosine and increasing excitatory neurotransmitter release. Increased consumption is associated with decreased daytime sleepiness and increased alertness. The degree of impact on SOL, sleep disruption, and nonrestorative sleep is both time and dose dependent, although sensitivity to these effects may vary across individuals. Caffeine reduces TST, SWS, and REM sleep, as well as increases SOL and sleep fragmentation. Caffeine may exacerbate RLS symptoms. Caffeine is a psychoactive drug and thus is associated with such phenomenon as dependence, tolerance, and withdrawal. Many products contain caffeine (see Table 5.1), including a number of OTC cold remedies, pain relievers, and weight loss preparations. There are a number of herbal stimulants (see above) that also contain caffeine as the active ingredient.

- **Nicotine (cigarette smoking)** is associated with increased SOL and disrupted and nonrestorative sleep. Nicotine may also increase symptoms of RLS and sleep-disordered breathing. Nicotine withdrawal may result in disrupted sleep and daytime somnolence. In terms of treatment for nicotine dependence, nicotine gum reduces SWS, and the patch reduces sleep efficiency (time asleep and time in bed) and prolongs SOL.
21
Sleep and Neurodevelopmental Disorders

GENERAL CONSIDERATIONS
Sleep disturbances in children with neurodevelopmental disabilities, such as autism and a variety of congenital and inherited conditions, are extremely common and often a source of considerable stress for the families of these children. The types of sleep disorders that occur in children with neurodevelopmental disabilities are varied and generally not unique to these populations. However, the prevalence of sleep issues is considerably higher, and the sleep problems tend to be more severe, chronic, treatment-resistant, and more likely to recur or relapse compared to those in typically developing children. Multiple sleep disorders are also likely to occur concurrently. Furthermore, the impact of disrupted and/or insufficient sleep on cognitive, emotional, and social development and behavior in these already at-risk children is potentially profound.

This chapter will focus on the etiology, clinical evaluation, and management of sleep problems in children across a spectrum of neurodevelopmental disorders. Sleep problems in children with primarily mental health conditions, including attention-deficit/hyperactivity disorder (ADHD) and depression, are addressed in Chapter 23.

Sleep Screening in Children with Neurodevelopmental Disorders
The high prevalence of sleep disturbances in developmentally delayed children underscores the importance of ongoing screening for sleep problems in these populations. In addition, practitioners need to consider multiple factors in evaluating an individual child with a sleep disturbance. The heterogeneity of possible etiologies for and contributing factors to sleep disorders in this population is considerable, and successful treatment is contingent on identifying and addressing these multiple issues.

ETIOLOGY AND RISK FACTORS
Much of the sleep research in children with neurodevelopmental disabilities has been conducted with heterogeneous diagnostic groups, rather than on homogeneous groups of children with specific diagnoses. Overall, nonspecific sleep problems, such as shortened sleep duration, irregular sleeping patterns, delayed sleep onset, frequent nightwakings, and early morning waking, are common across the spectrum of neurodevelopmental disorders. Some sleep disorders are more commonly associated with specific neurodevelopmental disorders or syndromes, such as obstructive sleep apnea (OSA) in Down syndrome (DS) and circadian rhythm disturbances in blindness. Although there is obviously considerable heterogeneity among the various disorders that may be included under the umbrella of neurodevelopmental disorders, many children with developmental delays and other special needs share common risk factors that may predispose them to sleep disorders. These include the following:

- **Medical issues:**
  - Central nervous system malformations or injuries that affect structures involved in control of sleep-wake rhythms (thalamus, basal forebrain) or respiration (brainstem) increase the likelihood of sleep disturbances and sleep-disordered breathing (SDB).
  - Craniofacial abnormalities that result in obstruction at various levels of the upper airway, such as choanal stenosis, midface hypoplasia, maxillary and mandibular hypoplasia, or hypertrophy of soft tissue structures, are frequently found in a number of syndromes (e.g., Pierre Robin
sequence, achondroplasia, Treacher-Collins, Goldenhar, Crouzon, and Apert syndromes and mucopolysaccharidoses). These craniofacial anomalies may predispose a child to SDB (see Chapter 22).

- **Neuromuscular disease** with associated weakness of respiratory muscles, such as congenital myopathies, muscular dystrophies, and other forms of hypotonia, often results in hypoventilation. The ability to maintain a patent airway during sleep, especially in the presence of upper airway obstruction, is also decreased in these children (sleep issues related to neuromuscular disease are discussed in more detail in Chapter 22).

- **Medication use** (including a variety of psychotropics) may result in altered sleep patterns and sleep disturbance, as well as may also cause respiratory depression.

- **Obesity** associated with Prader-Willi syndrome (PWS) and other congenital syndromes significantly increases the risk of OSA.

- **Seizure disorders** (especially of the frontal and temporal lobes) may disrupt nocturnal sleep and result in daytime sleepiness and fatigue. For example, Landau-Kleffner syndrome is associated with continuous epileptiform activity during sleep, and children with Lennox-Gastaut syndrome frequently have tonic seizures during sleep. Sleep deprivation, in turn, increases seizure propensity; comorbid SDB may also increase seizure frequency in susceptible children. In addition, although suppression of nocturnal seizures by anticonvulsants generally improves sleep quality and continuity, medications used to treat seizures may also contribute to insomnia and daytime somnolence (sleep and seizures are presented in more detail in Chapter 22).

- **Sensory deficits:** Sensory deficits can significantly impact sleep. For example, visual impairment involving complete lack of light perception is associated with profound sleep disturbances resulting from disruption of the normal circadian sleep-wake pattern (e.g., delayed or advanced sleep-wake phase, irregular sleep-wake rhythm, and non-24-hour or “free-running” sleep-wake rhythm disorders), with associated difficulty falling asleep, early morning waking, and daytime sleepiness (see also Chapter 18). This is presumably due to a lack of photic (light) input to the circadian pacemaker, resulting in altered or lack of entrainment to the 24-hour day. Lack of a normal response to social and environmental cues in children with autism and other developmental disorders may contribute to irregular sleep-wake patterns. Alternatively, heightened or altered sensitivity to tactile, auditory, or other environmental sensory stimuli (e.g., sounds, ambient light, tags and seams on pajamas, texture, and weight of bedclothes) may lead to difficulties in settling and problematic nightwakings.

- **Daytime behavioral problems:** Children with daytime behavior problems, such as aggression, noncompliance, and poor impulse control, may engage in similar behaviors at bedtime and throughout the night, resulting in bedtime resistance, delayed sleep onset, and prolonged nightwakings. In addition, self-injurious behavior has been reported both to result from and to be exacerbated by sleep problems. Children with repetitive, stereotypic, or ritualistic behaviors may increase these behaviors in the evening, prolonging bedtime routines and interfering with settling at bedtime and sleep onset.

- **Cognitive impairment:** In general, higher degrees of cognitive impairment tend to be associated with more frequent and severe sleep problems; prevalence of sleep problems in children with severe mental retardation has been estimated to be on the order of 30% to 80%, compared to 50% in children with less severe cognitive impairment. Children who have difficulty communicating their needs or understanding and responding to environmental demands are more likely to have problematic sleep. In addition, children with social interaction impairments may be more prone to behavioral sleep disorders.

- **Comorbid psychiatric disorders:** Psychiatric disorders, such as depression and anxiety, are common in children and adolescents with developmental delays and autistic spectrum disorders, and may contribute to
sleep problems. Severe hyperactivity and ADHD and/or mood instability are common to many chromosomal and genetic disorders, including velocardiofacial syndrome, Angelman syndrome (AS), Williams syndrome (WS), Fragile X syndrome, and Klinefelter syndrome, and may contribute to the genesis of sleep onset and maintenance difficulties in these children (see Chapter 23 for more detailed information).

- **Parenting-related variables:** Parenting issues, such as difficulty with limit setting and high levels of family stress, may contribute to and be exacerbated by sleep difficulties in the developmentally delayed child. Parents of developmentally delayed children may have inappropriate expectations regarding sleep patterns and behaviors that are based on the child's chronologic rather than on the developmental age. Alternatively, caregivers may see sleep problems as “inevitable” and, therefore, make only brief or limited attempts at managing them; for similar reasons, parents may be less likely to seek professional advice for treating sleep problems.

**SPECIFIC NEURODEVELOPMENTAL DISORDERS**

The association between autism spectrum disorder (ASD) and sleep problems is one that poses significant clinical challenges and thus is presented in some detail below. Other specific neurodevelopmental syndromes that have also been reported in association with sleep disturbances are also discussed below, in alphabetical order.

- **Autism spectrum disorder (ASD):** Studies examining sleep problems in children with ASD suggest that these children are at high risk for a variety of sleep problems, even when compared with nonautistic children with similarly low levels of intellectual functioning. The prevalence of caregiver-reported sleep problems in ASD ranges from 34% to 89%, while for children with intellectual disabilities it ranges from 44% to 86% (highest in younger children and those with more severe intellectual compromise). In autism, the prevalence of past or concurrent sleep problems, depending on the definitions used, has been estimated from 44% to 89%. Rates of sleep problems appear to be largely independent of IQ, although there appears to be some positive correlation between sleep disturbances and the severity of daytime behavior problems and communication impairments.

The types of subjective sleep problems most commonly described include highly irregular sleep-wake cycles, difficulty settling and delayed sleep onset, frequent, prolonged, and disruptive nightwakings, short sleep duration, and early morning waketimes. In contrast to typically developing children, sleep issues do not appear to diminish significantly with age. Moreover, there appears to be a strong association between sleep problems and internalizing and externalizing behaviors in these children. Sleep routines may be both unusual and problematic in these children because of stereotypic and repetitive behaviors, and they frequently have difficulty adapting to any alterations in routines. Some studies have reported an increase in parasomnias, such as sleepwalking and bruxism; also reported is a relative increase in REM behavior disorder (RBD), an otherwise extremely rare condition in childhood, which is characterized by absence of the normal muscle atonia associated with REM sleep and results in “acting out” of dream content. In general, sleep problems in these children are distinguished less by their unique characteristics than by their severity, chronicity, and high relapse rate, as well as the magnitude of impact on caregivers. In contrast to subjective sleep complaints, there have been few reliable and consistent clinically significant objective (polysomnography [PSG] and actigraphy) differences in sleep architecture demonstrated in autistic children, although some studies have reported increased sleep-onset latency, wake after sleep onset, and sleep fragmentation, as well as decreased sleep efficiency. Another possible exception is the observation that these children appear to have alterations in REM sleep variables (e.g., reduced REM percent, increased muscle tone in REM), which has
raised the intriguing possibility that administration of REM enhancing drugs (e.g., donepezil) could potentially improve cognitive function in these children.

There are a number of biological hypotheses that have been put forth to explain the high prevalence of sleep disturbances in ASD, although it should be pointed out that these are largely speculative at this time. First, because of the irregular sleep-wake patterns commonly displayed by autistic children, circadian rhythm abnormalities have been postulated to account for a significant percentage of sleep issues. An underlying disturbance in melatonin synthesis in autism potentially related to genetic variation in melatonin pathway enzymes has been proposed. In particular, there appear to be altered levels of melatonin precursors (e.g., tryptophan, serotonin) in these children, and other studies have suggested they may also have alterations in melatonin receptors. Furthermore, children with ASD demonstrate abnormal patterns of melatonin secretion, including decreased amplitude, and paradoxical daytime elevations accompanied by nocturnal decreases in melatonin. Other hypotheses regarding circadian dysregulation include possible anomalies in circadian clock genes, which in turn impact both motor planning and sleep; greater sensitivity to changes in photoperiod, resulting in the frequently observed seasonal variations in sleep problems in these children; and a decreased awareness of and sensitivity to social and environmental factors, resulting in lower levels of “entrainment” or synchronization of the circadian system by environmental cues. Other proposed biologic mechanisms include abnormalities in secretin, an endogenous gastrointestinal polypeptide which has been hypothesized to have a role in language development, behavior, and regulation, of sleep; increased activation of the inflammatory response system in autism, resulting in higher levels of inflammatory markers (e.g., cytokines) with effects on sleep regulation; a primary arousal dysfunction characterized by altered sleep homeostasis or “sleep drive” (disruption of -aminobutyric acid [GABA]-ergic neurons has been described); and an underlying dysfunction in monoaminergic pathways (dopamine, serotonin).

A variety of medical issues may also contribute to sleep problems in children with ASD. These include seizures, gastrointestinal disturbances, and concomitant use of medications such as psychotropics and anticonvulsants that can impact sleep. In addition, children with ASD may be at higher risk for primary sleep disorders (e.g., OSA syndrome, restless legs syndrome/periodic limb movement disorder (PLMD), bruxism) for a variety of reasons. For example, atypical eating behaviors resulting in extremely limited food choices may lead to iron deficiency and exacerbate restless leg syndrome symptoms. Thus, it is important to recognize the possible contribution of these disorders to sleep complaints in children with ASD and screen for them accordingly.

Furthermore, some of the core behaviors associated with ASD may contribute to the genesis of sleep problems. These include repetitive behaviors and perseveration, emotional dysregulation, self-stimulatory and self-injurious behaviors, poor communication skills, unresponsiveness to social cues, and sensory integration deficits.

Finally, a number of psychosocial and environmental factors are likely to contribute to the increase in severe sleep problems in these children. These include learned maladaptive sleep patterns, inadequate parent limit setting, and increased parental awareness of sleep issues. Children and adolescents with ASD are particularly prone to anxiety. Thus, sleep problems related to nighttime fears, separation anxiety, and obsessive compulsive behaviors are commonly observed. Finally, it is important to recognize that many patients will have a multifactorial etiology for their sleep problems.

Note that the Autism Treatment Network of Autism Speaks has developed a number of excellent resources for screening, diagnosis, and management of sleep problems in children with ASD, which may be found on their website www.autismspeaks.org.
Sleep in ASD

Sleep problems in children with ASD are extremely common. Parents of these children may view the sleep disturbance as “intrinsic” or “inevitable” to the disorder and, thus, may not spontaneously seek medical advice until the problem becomes severe. Sleep problems in these children are often chronic, but can be successfully treated with a combination of behavioral and pharmacologic strategies.

- **Angelman syndrome (AS):** The main clinical features of AS include intellectual disability (cognitive performance at the severe functional impairment level), lack of speech, seizures (including myoclonic, generalized tonic-clonic, atypical absence, and atonic seizures), and a characteristic behavioral profile consisting of a “happy” demeanor, easily provoked laughter, short attention span, hypermotoric behavior, and mouthing of objects. The prevalence of subjective caregiver reports of sleep problems in children with AS ranges from 20% to 80%; increased rates are found in younger patients (toddlers and preschoolers), with a general trend toward improvement with increasing age. Shortened sleep duration, with a seemingly decreased need for sleep (e.g., 5-6 hours), frequent and/or prolonged nightwakings, early morning awakening, and irregular and non-24-hour sleep-wake cycles are reported. Other sleep features that have been described by caregivers include a high level of reliance on sleep facilitators, being easily awakened by loud noises, and disorientation upon arousal. Daytime somnolence, however, is rare. PSG studies have demonstrated an increase in sleep-onset latency and nighttime, decreased REM and increased slow-wave sleep (SWS) percent, and increased sleep fragmentation, especially associated with seizures. Some studies have reported an increase in PLMs in these children, although not all studies have concurred with this finding. The etiology of sleep disturbances in children with AS is likely to be multifactorial, potentially involving dysregulation of GABA-mediated inhibitory influences in thalamocortical interactions. One study reported low evening levels of melatonin in children with AS, supporting clinical observations that supplementation with exogenous melatonin may be helpful in these children.

- **Down syndrome (DS):** Children with DS are clearly at increased risk for SDB (estimated OSA prevalence range across studies 30%-80%). SDB severity also tends to be worse in these children, and they are more likely to have residual disease following adenotonsillectomy, compared to typically developing children. The etiology of SDB is related to a number of interrelated factors, including generalized hypotonia, obesity with central distribution of adipose tissue, midfacial hypoplasia and glossoptosis (large, posteriorly placed tongue), hypothyroidism, and adenotonsillar hypertrophy; upper airway imaging may be helpful in identifying the precise sites of obstruction. OSA prevalence does not appear to be related to age or the presence of congenital heart disease. The AAP (2011) recommends initiating a discussion of the symptoms of OSA with caregivers of children with DS during the first 6 months of life. Central sleep apnea and Cheyne-Stokes respiratory pattern have also been reported to be more common in DS. PSG studies have demonstrated increased arousals and sleep fragmentation not solely attributable to OSA and an increase in movement arousals and PLMs. Children with DS also exhibit lower sleep efficiency, reduced REM sleep, and increased SWS compared to controls. In addition, subjective sleep complaints, including difficulties with initiating and maintaining sleep and daytime sleepiness, occur in up to 70% of these children. The presence of both behavioral sleep problems and SDB is associated with impairments in cognition, quality of life and functional domains.

- **Prader-Willi syndrome (PWS):** PWS is a complex neurogenetic disorder characterized by severe hypotonia with poor sucking and feeding difficulties in early infancy, followed by excessive eating and gradual development of morbid obesity in later infancy or early childhood, hypogonadism, behavioral problems, and various endocrinopathies (growth hormone [GH] and thyroid deficiencies). Reported rates of SDB are highly
variable (0%-100%) in children with PWS, but most studies have supported a significant increase in OSA related to the associated obesity. The severity of the OSA appears to correlate with body mass index and adenotonsillar hypertrophy. These children have high rates of post-adenotonsillectomy complications. In addition, several studies have suggested that there is an increased risk for sudden death in children with PWS associated with GH replacement therapy for short stature, presumably due to increased adenotonsillar growth. While a rare occurrence, the risk appears to be increased both by the presence of comorbid OSA and concurrent acute upper respiratory illnesses and may be highest soon after initiation of therapy. On the other hand, studies have suggested that GH replacement improves oxygenation and cardiovascular function during sleep. Recommendations are to obtain a screening PSG prior to initiation of GH therapy and at regular intervals (at least annually) during treatment; untreated severe OSA is considered to be a contraindication to GH treatment. Additional ventilatory abnormalities found in these children include chronic hypoventilation and central sleep apnea, which are associated with oxygen desaturation, especially in infants; the latter may respond to supplemental oxygen therapy.

REM sleep abnormalities, including decreased REM onset latency and an increased prevalence of REM intrusion phenomena, such as sleep attacks, cataplexy, and sleep paralysis, have also been identified in children with PWS. The percentage and depth of SWS also appear to be increased in these patients. Finally, both subjective complaints and objective evidence (mean sleep-onset latency less than 5 minutes and sleep-onset REM periods on multiple sleep latency tests) of significant daytime sleepiness are common in PWS, especially in adolescence and adulthood, and some of these children meet diagnostic criteria for narcolepsy. Although hypersomnolence may be associated with OSA and may improve with treatments such as continuous positive airway pressure, it may also occur independently of obesity and SDB, and significantly compromise daytime functioning; some of these children may benefit from treatment with drugs for hypersomnia such as modafinil. It has been hypothesized that these patients may have a primary hypersomnia that is secondary to hypothalamic dysfunction; alternative explanations have included non-REM-REM (ultradian) cycling dysregulation and abnormalities in melatonin secretion patterns.

Rett syndrome (RS): RS is a rare neurodevelopmental disorder usually affecting females, and is caused by mutations in the X-linked MECP2 gene encoding methyl-CpG-binding protein. Clinical features include intellectual disabilities, loss of motor and social functions, development of stereotypic hand movements, breathing dysfunction (hyperventilation, irregular respiration and apnea), seizures, spasticity, peripheral vasomotor disturbance, scoliosis, and growth retardation. Recent studies suggest that ventilatory abnormalities may also be present on nocturnal PSG, including obstructive apneas and hypopneas. PLM and continuous nocturnal spike-wave activity may be observed as well. Sleep problems are estimated to occur in 80% or more of children with RS, with the prevalence decreasing with age. These children have more difficulties falling asleep, fragmented sleep, nightwakings, early morning awakening, and shortened sleep duration. Nocturnal events, including laughing and inconsolable screaming, are also common. Dysregulation of melatonin secretion responsive to exogenous melatonin administration has been demonstrated in some girls with RS. They may also have increased sleep fragmentation related to nocturnal seizure activity.

Smith-Magenis syndrome (SMS): SMS is a syndrome characterized by intellectual disabilities, dysmorphic features, obesity, and congenital anomalies related to an interstitial deletion of chromosome 17p11.2. The prevalence of subjective significant sleep difficulties is high (60%-100%) in these children. Caregivers frequently report difficulty falling asleep, short sleep duration, frequent and prolonged nightwakings, early morning awakening, daytime sleepiness, coughing/snoring, and nocturnal enuresis. Circadian rhythm disturbances are especially common. Clinical and polysomnographic features of RBD, which is typically very
rare in children, have also been described. PSG studies indicate decreased total sleep time and REM percentage, with a tendency for early sleep onset and offset. Inversion of the normal circadian rhythm of melatonin secretion (i.e., low evening and high daytime levels) and other circadian clock abnormalities have been reported in those with SMS; evening administration of melatonin has been successfully used in some patients.

**Williams syndrome (WS):** WS is a rare genetic disorder characterized by a complex physical, cognitive, and behavioral phenotype. Subjective sleep complaints, including bedtime resistance, difficulties initiating and maintaining sleep, and restless sleep, are very common in children with WS and may be linked to greater deficits in language. PSG findings include decreased sleep efficiency and total sleep time, increased wake after sleep-onset and respiratory-related arousals, increased SWS, and decreased REM. There is mixed evidence that WS is associated with an increase in PLMs and associated arousals and sleep fragmentation. Recent studies suggest that increased cortisol at bedtime and a less pronounced rise in melatonin levels before sleep may play a role in the occurrence of sleep disturbances, especially prolonged sleep onset.

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**Sleep and Breathing in Neurodevelopmental Disorders**

SDB is particularly common in many populations of children with neurodevelopmental disabilities, especially in children who have conditions characterized by craniofacial abnormalities, such as midface hypoplasia and micrognathia, hypotonia, and/or obesity. The resulting sleep fragmentation may contribute significantly to learning and behavior problems already present as part of their underlying disorder. While challenging, successful treatment with positive airway pressure can be accomplished if appropriate behavioral support is available.

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**EVALUATION**

Evaluation of sleep problems in children with neurodevelopmental disabilities is similar to the screening and assessment of sleep problems in other pediatric populations, although given the high prevalence, a high index of suspicion should be maintained and regular screening is key. A functional assessment of sleep problems, in which parents are asked to track the antecedent and consequence of each disruptive nighttime behavior, can also be performed. Nighttime behaviors of children with developmental disabilities are often perpetuated by social reinforcement, such as parent attention in the form of comfort, play, or even verbal warnings or any type of interaction at bedtime or during the night.

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**TREATMENT**

**Sleep Practices**

- **Principles of healthy sleep practices**, including environmental (e.g., temperature, noise level, ambient light), scheduling (e.g., regular sleep-wake patterns), sleep practice (e.g., bedtime routine), and physiologic (e.g., exercise, timing of meals, caffeine use) factors, are frequently overlooked in preventing and treating sleep problems in developmentally delayed children. Healthy sleep practices have a positive impact, especially in this population, because they help to entrain intrinsic circadian rhythms to the external environment and 24-hour-day/night cycle. In addition, behavioral conditioning results in the association of certain activities and environments with sleep.

- **Sleep environment** that is conducive to sleep can be challenging to implement and often need to be modified and individually tailored. Achieving an appropriate level of comfort and safety in the sleeping
environment, particularly for children with sensory and motor disabilities, is important. Positioning in bed of children with limited mobility and choosing an appropriate sleep surface (e.g., waterbeds, adjustable mattresses) that is tailored for physical comfort may need to be considered. Some children may require a “fortified” crib or bed that is surrounded by a mesh material to confine them to bed while allowing visibility both from within and without.

**Sensory Issues**

Specific occupational therapy techniques and devices that have been used to address sensory integration issues (e.g., brushing, weighted vests and blankets for children with tactile sensory issues, “white noise” generators that produce a mixture of all frequencies and can mask environmental sounds, body pillows) may be useful in alleviating sleep problems in children with sensory issues. Evaluation and consultation with a pediatric occupational therapist may be helpful.

- **Alarm systems** that alert caregivers when a child has left the bedroom may be necessary, as some children may wander around the house during the night. A two-way monitor or Webcam in the bedroom may reassure parents and reduce the need for parental intervention during the night.

- **Bedroom lighting** may need to be adapted. Although a dark sleeping environment is preferable, some anxious children might benefit from a dim night-light. However, children with cortical blindness may stare into a light source for self-stimulation (“light gazing”), keeping them in an alert state.

- **Bedtime routines** are especially important and should include specific and predictable steps. Children with neurodevelopmental disorders may be easily overstimulated; so bedtime activities must be carefully planned and calming, such as the use of gentle, rhythmic, repetitive, low-frequency movements, quiet sounds, light massaging, brushing, vibrating pillows and beds, and soft music. The use of pictorial representations of the sequential components of the bedtime routine may assist in “cueing” the child to participate. However, parental presence at time of falling asleep should be discouraged if at all possible to avoid the development of problematic sleep-onset associations. Expectations around nighttime behavior must be clearly communicated and consistently enforced. Encouraging the use of appropriate transitional objects may be particularly important in these children.

- **Caregivers** need to be considered in the development of the treatment plan. That is, sleep recommendations are most effective when it provides for the sleep needs of both the child and the parents, and it is important for healthcare providers to directly address and support caregivers’ need for sufficient sleep. Thus, assisting parents to make operational changes, such as arranging for respite or night nursing care, can have a significant positive impact.

- **Additional sleep practices** include ensuring that the development of conditioned associations between the child’s bed and bedroom and behaviors incompatible with sleep (e.g., watching television) is avoided. Expectations around nighttime behavior must also be clearly communicated and consistently enforced. It is also particularly important to maintain a regular daytime routine for mealtime, playtime, and other activities with these children to help synchronize sleep-wake rhythms. Unless developmentally appropriate, daytime sleep periods and naps should be restricted to avoid compromising consolidation of nighttime sleep.

**Behavioral Management of Sleep Problems**

Sleep disturbances in children with developmental delays are frequently amenable to management with a variety of behavioral strategies. These should be tailored to the developmental level of the child.
and the resources of the family. Choosing reasonable, attainable, and mutually acceptable target goals in terms of bedtime behaviors, nightwakings, and sleep duration is particularly important in assisting families.

**BEHAVIORAL MANAGEMENT**

Children with neurodevelopmental disorders have many of the same sleep problems as typically developing children, such as sleep-onset association and limit-setting subtypes of behavioral insomnia of childhood (see Chapters 7 and 8), which warrant similar intervention strategies, sleep scheduling, graduated extinction procedures, and fading of adult intervention at bedtime and during nightwakings. These strategies can be as effective with children with neurodevelopmental disorders; however, positive changes may simply take longer. Of course, all of these strategies involve commitment and consistency from parents; as with many behavioral treatments, the targeted problem often becomes worse before it gets better. Families with disabled children often face numerous challenges on a daily basis, and caregivers may be hesitant to disrupt established patterns of home behavior for the sake of intervening with their children's sleep problems. Educating parents about the link between decreased daytime functioning and disrupted sleep and providing ongoing support are necessary for caregivers to successfully approach what is likely to be a difficult and possibly lengthy process.

Some special considerations should be given to the application of behavioral management strategies in these special populations. Ensuring the safety of these children, especially if nightwaking is a problem or if there is a history of self-injurious behavior, needs to be a key consideration. In general, parents tend to be more comfortable with more gradual treatment approaches. Furthermore, the severity and chronicity of sleep problems in many of these children often necessitate long-term treatment and utilization of a variety of treatment strategies. Finally, collaboration with a behavioral therapist in designing and implementing treatment plans may be prudent if there are complex, chronic, or multiple sleep problems or if initial behavioral strategies have failed.

**Pharmacologic Treatment**

The use of pharmacologic intervention may be required in some children with severe sleep problems that are not or only partially responsive to behavioral techniques, particularly if daytime functioning is compromised and/or caregiver mental or physical health is adversely affected. A wide variety of medications have been used in children with neurodevelopmental disorders. General principles of drug selection and medication management are detailed in Chapter 20.

As in all children, pharmacologic treatment should be combined with behavioral interventions. Short-term use of a hypnotic medication can rapidly decrease sleep-onset delay, eliminate prolonged bedtime struggles, and reduce problematic nightwakings, enabling parents to be more successful in implementing bedtime behavioral strategies, which ultimately are likely to have the more enduring impact. However, there are situations in which the severity and chronicity of the sleep problem necessitate long-term use of sedating medication; in these cases, the importance of choosing an agent with minimal long-term side effects is paramount.

Practitioners should be aware of the potential pitfalls in using hypnotic medications, especially unpredictable side effects in this population. In general, using medications with combined effects when appropriate (e.g., sedating anticonvulsants for children with seizures, sedating atypical antipsychotics in patients with aggression), thus potentially reducing drug exposure and minimizing adverse effects, is
desirable. Finally, specific medications have been demonstrated to be helpful in select conditions; for example, the PLMs associated with WS often respond to clonazepam, and sleep disruption in children with ASD has been shown to improve with clonazepam as well.

**Melatonin**

Melatonin is the best-studied pharmacologic intervention for developmentally delayed children with sleep problems, particularly in children with persistent alterations in circadian rhythm or sleep-wake schedule. Studies indicate that melatonin can result in significant improvements in sleep-onset delay, nightwakings, early morning waking, and total hours of sleep in up to 80% of children with a variety of neurodevelopmental disorders (cortical blindness, RS, autism, tuberous sclerosis, Asperger syndrome).

Studies have typically used doses in the range of 2 to 5 mg of melatonin, administered 30 to 60 minutes before bedtime (see Chapter 20 for more detailed information). Some studies have used doses up to the 10-mg range for refractory cases without reported adverse effects, although the rationale for such “supraphysiologic” doses remains unproven. It should be emphasized that melatonin's positive effect on sleep may be due either to its “chronobiotic” (circadian sleep-wake pattern resetting) or “hypnotic” (sedating) effects; studies in adults with delayed sleep phase have suggested that a small (i.e., 0.1-0.5 mg) physiologic dose of melatonin 5 to 7 hours before the habitual late bedtime may actually be more effective in advancing sleep-onset time in the case of a true circadian-based sleep-onset delay, while larger doses just before bedtime have primarily a hypnotic effect. Melatonin is available in liquid and sublingual as well as tablet formulations, and as a slow-release preparation.

Although generally considered safe, there are some caveats regarding the use of melatonin in children with neurodevelopmental delays. Due to its suppressive effects on the hypothalamic-gonadal axis, there is a theoretical possibility of initiation of precocious puberty if melatonin is withdrawn abruptly after chronic administration. In addition, little is known about long-term side effects with chronic use. Given the over-the-counter availability of melatonin, the purity of preparation and inconsistent dosing are also concerns. Overall, melatonin, as with all pharmacologic interventions, is likely to work best in combination with behavioral interventions.

**Light Therapy and Sleep-Wake Scheduling**

Primary circadian rhythm disturbances may also be treated with bright-light therapy and sleep-wake scheduling or chronotherapy. Morning phototherapy with a light box delivering 2,000 to 8,000 lux has been used to successfully treat refractory sleep-phase delay in children with severe brain injury. Duration and timing of light exposure are typically 1 hour right after morning waketime, and are often combined with a gradual advance to an earlier sleep onset and offset time over a period of days to weeks. However, inappropriate timing of the phototherapy may actually worsen the circadian disturbance; so this treatment is best done under the supervision of a sleep professional. Chronotherapy, or the successive delay of the sleep-wake cycle over a period of days until the desired bedtime is reached, has also been used effectively to treat severe sleep-onset delay in autistic and blind children. It should also be noted that children who are prone to circadian rhythm disturbances may be relatively sensitive to and disrupted by circadian desynchronization (e.g., jet lag, daylight savings shift). Therefore, parents may need to carefully anticipate these situations.
GENERAL CONSIDERATIONS

Children with both acute and chronic medical conditions may suffer from sleep disturbances, including both primary sleep disorders and sleep problems that are secondary to the underlying disease processes. Sleep disturbances, including difficulty initiating sleep, bedtime resistance, nightwakings, decreased sleep efficiency (time asleep/time in bed), and daytime sleepiness are common in children with both acute and chronic medical conditions. It is likely that a host of factors contribute to the development and exacerbation of sleep problems in medically compromised children, including physical issues, such as pain, immobility, and effects of treatment including surgical procedures, chemotherapy, and medications; psychological factors, such as comorbid mood disorders, anxiety, and family stress; and environmental variables such as hospitalizations and disrupted daytime schedules. In turn, the effects of sleep fragmentation and sleep loss in these children are also likely to be particularly significant, given what is known about the psychological and emotional impact of insufficient sleep and the physiologic effects of deficient sleep on the immune system and the body's ability to respond to stress.

Sleepiness versus Fatigue

A distinction between sleepiness and fatigue may be relevant in assessing the role of sleep in some medical conditions, although this is sometimes more of a semantic difference that may be difficult to elicit in young children. By definition, "sleepiness" is the propensity to doze off or fall asleep. Although sleepiness may be affected by a host of intervening variables, including time of day, motivation, physiologic states such as hunger, and level of stimulation, true daytime sleepiness generally implies the presence of insufficient and/or disrupted sleep. Fatigue, on the other hand, is more likely to refer to a subjective state of low energy and low motivation, which may or may not primarily be a result of poor sleep.

ETIOLOGY AND RISK FACTORS

Pain

Both acute and chronic pain have major physical and psychological effects on sleep. Complex multidirectional relationships exist among sleep, pain parameters (including etiology, chronicity, frequency, severity, location, and pain perception), physiology, biologic and circadian factors, mood, age/developmental stage, gender, race and ethnicity, socio-cultural factors, and functional outcomes in a variety of pediatric pain populations. For example, most studies indicate that even after controlling for other factors, pain intensity, frequency, and duration are all predictive of disrupted sleep patterns. Poor quality sleep, in turn, is associated with increased next-day pain intensity and frequency. In addition, the sleepiness and fatigue associated with sleep problems may exacerbate pain. Moreover, studies have linked sleep problems in pediatric pain populations to a range of functional outcomes, including reduced executive function, slowed reaction time, and impaired quality of life.

Pain may cause sleep-onset delay, nightwakings, fragmented and restless sleep, and frequent arousals, as well as daytime sleepiness and fatigue. Even adaptive behaviors, such as protection of the injured area by positioning and increased attention to movements during sleep, may further impact on sleep quality. Overall, behavioral assessments and polysomnography (PSG) studies have consistently identified more sleep disturbances in children and adolescents with persistent pain compared
to healthy children (actigraphy results are less consistent). For example, one study reported that approximately half of a group of children with newly diagnosed cancer suffered pain-related sleep disturbances. There is also evidence that certain subpopulations of children with chronic pain conditions are at higher risk for primary sleep disorders, such as obstructive sleep apnea (OSA) and periodic limb movement disorder (PLMD).

The interaction between pain and sleep is also impacted by a number of psychological variables. Coping skills needed to deal with pain may be compromised by mood changes, irritability, low frustration tolerance, and poor regulation of emotions related to sleep loss. Learned associations with both sleep and pain tend to be very powerful, and children may become conditioned to associate bedtime and sleep with negative and painful experiences. Finally, implementation of behavioral management strategies may be impacted by decreased motivation and goal-directed behavior and by decrements in attention and in behavioral and impulse control in a child experiencing chronic sleep loss.

Additional factors that may be involved in the complex relationship between sleep and pain include stress, hyper-arousal, and anxiety. These may be particularly problematic at sleep onset, as falling asleep requires the relaxation of vigilance and attention to the surrounding environment. On the other hand, sleep may also represent a temporary psychological escape from pain and some studies have suggested that sleep itself may be a coping strategy. Thus, the inability to initiate or maintain sleep may further heighten anxiety and decrease pain tolerance. Some children with chronic medical conditions associated with pain may have comorbid psychiatric disorders, such as anxiety and depression, which may lead to further deterioration of sleep patterns and quality (see Chapter 23). Negative mood may also mediate the influence of poor sleep quality on pain perception. Children with pain conditions related to trauma may also have posttraumatic stress disorder (PTSD) symptoms that may significantly disrupt sleep.

Medications used for pain management also may directly impact sleep by affecting sleep patterns and architecture. For example, opiates decrease rapid eye movement (REM) sleep and slow-wave sleep (SWS) and increase sleep fragmentation. Side effects of analgesics, such as gastrointestinal discomfort, and withdrawal effects of pain medications may result in sleep disturbances. For example, withdrawal of medications with REM-suppressant effects, may result in REM sleep rebound and increased nightmares. Finally, inadequate pharmacologic control of nocturnal pain may lead to more disrupted sleep, increased daytime fatigue, and a further deterioration in pain management.

Finally, somewhat surprisingly, given the clear association between sleep and pain in children with a variety of pain syndromes (e.g., headache, juvenile rheumatoid arthritis (JRA), sickle cell disease), there have been only a few small uncontrolled intervention studies aimed at managing pain and sleep in the pediatric population. While preliminary are promising in that pain, sleep, fatigue, and psychological and functional ability have been shown to improve with targeted treatment, more studies are clearly needed.

**Family Issues**

Families of children who have had life-threatening illnesses or who have chronic medical conditions may develop a “vulnerable child syndrome,” in which parents are unwilling or unable to set developmentally appropriate limits because of concerns about compromising the child's physical condition or causing emotional distress. Increased bedtime resistance, prolonged nightwakings requiring caregiver intervention, inappropriate daytime napping, and reactive co-sleeping with parents may result. In addition, parents of children with medical problems may be unwilling to set and enforce healthy sleep habits, such as a consistent bedtime and avoidance of evening electronic media exposure, further compounding poor sleep.

**Hospitalization**
Hospitalization is a stressful event for children and their families, and behavioral regression to a previous developmental level (e.g., dependence on parental presence in order to fall asleep) is a common response, particularly in younger children. This situation may result in an acute adjustment sleep disorder with difficulties initiating and maintaining sleep, or exacerbate preexisting sleep problems, or both. Studies of sleep disturbances in hospitalized children have reported significantly later bedtimes and waketimes, prolonged sleep latencies, shortened sleep duration, increased nightwakings, and curtailment of developmentally appropriate daytime napping opportunities. Children with more prolonged and/or life-threatening illnesses requiring frequent hospitalizations, such as cancer, may be even more at risk for chronic sleep disturbances.

In addition to issues related to the underlying medical condition and stress and anxiety about hospitalization, factors in the hospital environment may contribute significantly to the development of sleep problems in children in the inpatient setting. These factors include noise (including abrupt and unexpected increases in noise, as well as persistently elevated levels of background sound), frequent nocturnal interruptions for medication administration and vital sign assessments, and bright and/or inappropriately timed lighting, which may impact evening melatonin onset and thus disrupt normal circadian sleep-wake patterns. Sleep loss and sleep fragmentation are particularly common in intensive care settings for multiple reasons, including the underlying severity of the medical condition, the need for complex medication regimens, and the level of monitoring required. These sleep disturbances may also persist after discharge from the hospital, often as a result of learned associations and inadvertent parental reinforcement of maladaptive sleep patterns.

A number of potentially modifiable factors related to the hospital environment and hospital procedures may have a significant detrimental impact on the quality and quantity of sleep in hospitalized patients. Accordingly, relatively minor adjustments in the patient's surroundings could potentially have major positive consequences. These include the following:

- Increasing daytime activity levels and limiting daytime naps to promote nocturnal sleep
- Maintaining home patterns of bedtimes and waketimes
- Encouraging daytime and limiting nighttime light exposure as well as providing a regular schedule of daily activities to assist in maintaining normal circadian rhythms
- Decreasing late-evening stimulation (noise level, social interactions)
- Limiting nursing and medical assessments and procedures during the night
- Encouraging parents to provide familiar bedtime comfort objects from home and to maintain home bedtime routines as much as possible in the hospital
- Making the hospital room a “safe haven” by conducting potentially painful and anxiety-provoking procedures as much as possible in alternative locations
- Implementing proactive sleep-promoting measures, including the use of relaxation exercises and tapes, massage, and “dream-catchers,” which can also facilitate restorative sleep and prevent the development of sleep problems

While studies have suggested that between 50% and 75% of hospitalized adult patients are prescribed a sedative or hypnotic to improve sleep, hypnotic use in pediatric inpatients appears to be much less common. A large multisite study of sleep medication use in almost 10,000 pediatric inpatients reported that approximately 3% to 6% of hospitalized children had been treated pharmacologically with a variety of sleep medications (most of these were drugs that had already been prescribed in the outpatient setting). However, children with a psychiatric diagnosis were almost 3 times more likely to receive a sleep medication. Given that there may be
situations in which behavioral interventions alone are not practical for hospitalized children because of the length of time and staff resources needed to implement these treatments, use of hypnotic medications may be indicated (see Chapter 20). Finally, there is limited evidence that excessive daytime sleepiness (EDS) and fatigue, particularly in cancer patients, may be responsive to psychostimulant medication, such as methylphenidate or modafinil, as well as bright light therapy.

**Medications**

Many of the medications used to treat children with a variety of medical problems are known to have potential adverse effects on sleep and/or to be associated with daytime sedation (see Chapter 20 for a more detailed discussion of pharmacologic effects on sleep). These include analgesics for pain management, antihistamines and antipruritics for allergies, bronchodilators and corticosteroids for asthma, various chemotherapeutic agents, and anticonvulsants for seizures.

**Obesity**

Congenital or acquired conditions and syndromes characterized by or commonly accompanied by obesity (e.g., Prader-Willi syndrome) have an increased risk of sleep problems, particularly sleep-disordered breathing (SDB) and daytime sleepiness; these are discussed in more detail in Chapter 21. Alternatively, shortened sleep duration in children is both concurrently associated with and predictive of the development of being overweight and obesity. A separate section below (Sleep and Obesity) provides an overview of the complex relationship between sleep and obesity.

**Sleep and Quality of Life**

Virtually any medical condition, acute or chronic, may have adverse effects on a child's sleep quality and quantity. Multiple possible contributing factors should be considered, including concomitant medications, psychosocial factors, and pain. Because of the potential impact of sleep disturbances on both the underlying disease process and the child's and family's quality of life, the practitioner should make every effort to assess for and address sleep problems in the setting of medical illness.

**SPECIFIC MEDICAL CONDITIONS**

Specific medical conditions that have been reported in association with poor-quality and/or insufficient sleep are discussed below. However, it should be noted that virtually every medical condition in children and adolescents, because of related issues such as stress, pain, and medication use, might be accompanied and exacerbated by disrupted sleep. For example, a primary cardiac or respiratory disease may result in sleep disturbances, and the underlying disease may be exacerbated by sleep disruption.

(Note: Medical conditions are presented in alphabetical order, with obesity and seizures covered as separate sections at the end of the chapter.)

**Allergies**

Chronic allergy-mediated rhinitis with nasal congestion, postnasal drip, and nocturnal cough, as well as pruritus associated with dermatologic manifestations of atopy (e.g., eczema; see below) are associated with difficulties initiating sleep and particularly with frequent arousals and disrupted sleep. Environmental allergies also contribute to upper airway inflammation and are considered to be risk factors for OSA (see Chapter 15). Use of sedating antihistamines to control allergic symptoms during the day may also disrupt regular sleep-wake patterns, and decongestants such as pseudoephedrine have been associated with insomnia. Adequate
Asthma

Asthma in children is associated with sleep problems including decreased sleep duration, reduced SWS, reduced subjective sleep quality and sleep efficiency, more frequent nighttime arousals, and increased daytime sleepiness and SDB. Asthma symptoms often worsen at night as a result of physiologic changes (e.g., increased airway inflammation and resistance; increased coughing, wheezing, shortness of breath, and work of breathing; reduced mucociliary clearance; increased nocturnal gastroesophageal reflux and decreased lung volumes) and as a result of circadian variations in respiratory function and peak flows (e.g., lung function is at its lowest at about 4 a.m.), resulting in an up to 50% reduction in lung function during sleep compared to during the day. Other factors such as increased allergen exposure in the bedroom (e.g., dust mites, animal dander) and use of short-acting bronchodilators, which wear off during sleep, may also play important roles in nocturnal asthma exacerbations. In a number of studies, sleep symptoms have been correlated with subjective (symptoms) and objective (such as pulmonary function tests) measures of disease severity.

Pediatric studies indicate that one-third of asthmatic children report at least one awakening per night, and the overall prevalence of sleep problems in children with asthma has been reported to be 60%. Asthma severity has also been shown to impact both objective and subjective reports of sleep disturbance, and sleep issues have been shown to predict more severe asthma symptoms the following day. Nocturnal asthma symptoms have been associated with impaired daytime cognitive functioning and attention, poor school performance and decreased attendance, and fatigue. Furthermore, asthmatic children tend to rate themselves as significantly more tired in the morning compared to healthy controls. Daytime function appears to improve with medication adjustment and optimal symptom control; however, some studies suggest that even children with well-controlled asthma may have more disrupted sleep than normal controls.

Asthma also seems to be an independent risk factor for OSA, further disrupting sleep and exacerbating daytime impairment. Finally, medications commonly used to treat asthma may have adverse effects on sleep. For example, oral corticosteroids may cause significant sleep disruption, and theophylline is known to have a stimulatory effect on the central nervous system (CNS) with resultant increased wakefulness. However, most of the newer asthma medications have more selective β-adrenergic and fewer CNS-alerting effects. Insomnia has been reported as an occasional side effect of leukotriene esterase inhibitors, such as montelukast. In considering treatment options, it is important to weigh the benefits of optimal pharmacologic control of asthma symptoms against the potential negative effects of bronchodilators and other medications on sleep.

Atopic Dermatitis and Eczema

Atopic dermatitis may cause difficulty falling asleep, sleep disruption and fragmentation, night awakenings, decreased sleep duration, and resulting daytime sleepiness as a result of pruritus, resulting in frequent scratching and discomfort. Parents of children with chronic allergies and/or atopic dermatitis may also be more aware of and pay more attention to nightwakings, further perpetuating the cycle of disrupted sleep. Aggressive treatment and prevention measures for the underlying skin condition may include use of topical steroid and nonsteroid creams and ointments and oral sedating antihistamines at bedtime, physical barriers (e.g., socks on
hands to prevent scratching during sleep), bedroom environmental allergen control (e.g., cotton pajamas), and dietary changes if appropriate.

**Burns**

Children with severe and/or extensive burns may be at particularly high risk for sleep problems, which, in turn, may have significant adverse effects on their recovery. Difficulty falling asleep, increased arousals, nightmares, and increased daytime sleepiness are common in the acute phase and may persist for up to 1 year after the traumatic event. Changes in sleep architecture have been reported, including decreased SWS, which may contribute to the growth hormone insufficiency observed in burned children. It has been postulated that increases in particular types of peripheral cytokines in response to thermal injury (interleukin-1 and tumor necrosis factor) may contribute to sleep disruption through their specific role in sleep regulation. Burned children obviously also have multiple sources of pain, as well as severe pruritus, which may further disrupt sleep.

Additional factors are those common to all medically compromised and traumatized children, such as depression, anxiety and stress, PTSD, and hospitalization or intensive care unit (ICU)-related factors. Narcotic analgesics, sedatives, and other medications used to treat anxiety (benzodiazepines) and depression in burned children may also alter sleep architecture.

**Cancer**

Sleep problems, including difficulty initiating and maintaining sleep, have been reported to be common in children with cancer, particularly leukemia and brain tumors, which account for about 50% of childhood cancers. Studies report poor sleep quality, difficulty falling asleep, frequent night awakenings, and difficulty reinitiating sleep both during hospitalizations and at home. There are a myriad factors that are likely to increase the risk for sleep problems in these children, including stress and anxiety related to the diagnosis, pain, nausea and other effects of chemotherapy, effects of other medications (e.g., insomnia and sedation), frequent hospitalization, caregiver exhaustion and reluctance to enforce appropriate limits, and disruption of normal developmental processes with increased dependence on and decreased tolerance of separation from parents. Chemotherapeutic agents may be primarily sleep disrupting (e.g., corticosteroids) or sedating (e.g., l-asparaginase, -interferon) or both (e.g., vincristine, cytarabine); for example, adolescents on chemotherapy report significantly worse sleep quality. Children with CNS malignancies, particularly involving the brain stem and hypothalamus such as craniopharyngiomas (tumors located in the basal forebrain near sleep-regulating structures), have been linked to a host of childhood sleep disturbances, including nighttime awakenings, inability to maintain sleep, and secondary narcolepsy. Tumors located near the pineal gland can result in disruption in normal melatonin secretion, contributing to circadian-based sleep-wake disturbances. Patients who receive cranial radiation treatment are also at high risk for disruption of normal sleep-wake regulation and circadian rhythm processes as well as nocturnal seizures; injury to central respiratory control centers (e.g., medulla) may result in central sleep apnea. Long-term survivors of childhood cancer may continue to experience sleep problems in up to 50% of cases; sleep problems associated with PTSD may also occur in these patients.

In particular, cancer-related fatigue, which has been reported in 70% of cancer patients overall, can have a devastating effect on quality of life for these children. Fatigue may be related to physical factors such as anemia, poor nutrition, and underlying disease processes as well as to depression and other emotional issues. Complaints of fatigue may persist for months to years, even after treatment has been concluded, and continue to impact significantly on these patients’ ability to resume a normal life. Alterations in daytime activity levels and sleep-wake schedules in response to fatigue and EDS may further disrupt nocturnal sleep regulation and circadian rhythms. Fatigue-related symptoms may be difficult to distinguish from EDS (e.g., difficulty getting up in the morning, increased sleep duration, frequent napping, dozing off during activities), and both are commonly
present, especially in children with CNS neoplasms; these children are also at increased risk for secondary narcolepsy. Neoplasms in the hypothalamus, thalamus, and brain stem are particularly prone to causing EDS. Hypersomnia associated with cranial radiation therapy occurs in some 60% of children, potentially through injury to the optic nerve or retinohypothalmic tract, and includes a constellation of symptoms, including nausea, headaches, and low-grade fever that occurs 3 to 12 weeks post radiation and lasts for up to 2 weeks. Radiation-related sleepiness appears to be dose dependent, and evidence of increased sleep needs have been demonstrated as long as 15 years after treatment. Treatment with stimulants including methylphenidate, modafinil, armodafinil may alleviate both chronic fatigue/EDS and cancer-related cognitive dysfunction, but there is very limited experience in children. Initial studies in adults with breast cancer indicate that bright light exposure (phototherapy) in the morning can also ameliorate daytime fatigue.

Chronic Fatigue Syndrome

Chronic fatigue syndrome in adults and children has been reported to be associated with chronic sleep disturbances, including sleep-onset and maintenance problems, dysregulated sleep-wake patterns, and daytime sleepiness despite what is often an increase in total sleep time. Daytime napping may actually increase daytime sleepiness and contribute to worsening cognitive function. However, the precise relationship between sleep and physical and mental functioning in chronic fatigue syndrome has yet to be fully elucidated. PSG findings in adults suggest an increase in SWS and microarousals. In addition, sleep disruption may be associated with increased levels of circulating cytokines.

Cystic Fibrosis

Sleep disturbances are frequently observed in cystic fibrosis (CF). Prolonged sleep onset, increased sleep fragmentation, short sleep duration, and decreased sleep efficiency have been reported in children and adolescents with CF. Nocturnal awakenings and reduced sleep efficiency are frequently associated with disease severity. Prolonged REM latency and reduced amounts of REM sleep also appear to be related to severity. Complaints of EDS and fatigue are common, further contributing to associated neurocognitive sequelae. Gas-exchange abnormalities such as nocturnal hypoxemia are more common with progressive disease. There also appears to be an increased prevalence of sleep related breathing disorders (SRBDs) in patients with CF, especially in the presence of nasal polyps and chronic sinusitis. These respiratory disorders are postulated to compound the cardiovascular and metabolic abnormalities. Therapy with supplemental oxygen and bi-level positive airway pressure ventilation is likely effective in the short-term, but the longterm impact on morbidity and mortality is still uncertain.

Diabetes

Children and adolescents with type 1 diabetes have more frequent nightwakings, insufficient sleep, and daytime sleepiness, which in turn appear to be related to reduced quality of life and impairments in mood and academic functioning. The impact of type 1 diabetes on sleep may explain much of the neurocognitive and behavioral deficits reported in these children, and maintenance of adequate nocturnal glycemic control may play an important role in the relationship between diabetes and daytime functioning. Sleep architecture appears to be altered in these children, with more time spent in lighter stages of sleep and less in SWS; these changes are associated with higher HbA1C levels. In addition, the need for caregivers to monitor nocturnal blood glucose levels in their children may disrupt parental sleep and contribute to family stress and anxiety. Mounting evidence suggests that sleep duration, alterations in sleep architecture, chronotype, and circadian misalignment increase the risk of developing diabetes and have a negative impact on the metabolic control of both type 1 and type 2 diabetes. Patients with type 2 diabetes are more likely to be overweight and obese, increasing the risk for SRBD and further exacerbating impaired glycemic control.
Fibromyalgia
Juvenile-onset fibromyalgia is characterized by widespread chronic musculoskeletal pain and other associated symptoms, including fatigue, nonrestorative sleep, headaches, irritable bowel symptoms, dysautonomia, and mood disorders. Sleep complaints are almost universal in these patients and may include prolonged sleep onset, frequent arousals/awakenings, decreased sleep efficiency, and "nonrestorative" sleep (waking unrefreshed), as well as periodic limb movements (PLMs) disrupting sleep. PSG in patients with juvenile-onset fibromyalgia shows reduced sleep efficiency, increased arousals/awakenings, and more alpha-delta EEG wave intrusion (light sleep) into deep (delta) sleep. These sleep disturbances can potentially contribute to the daytime sleepiness or fatigue, depressed mood, and cognitive difficulties frequently associated with this condition.

Gastrointestinal Disorders
Gastrointestinal disorders, especially those of a more chronic nature such as irritable bowel syndrome and functional abdominal pain, have been associated with poor sleep quality in both adults and children; an underlying dysregulation mechanism resulting in both altered gastrointestinal function and sleep-wake regulation has been postulated. Sleep problems have been reported in as many as 50% of these children; most often difficulties with sleep onset and maintenance, as well as EDS. Sleep issues also seem to be related to functional disability and symptom severity. Drug therapy, including chronic steroid use in inflammatory bowel disease, may result in additional sleep disruption.

Gastroesophageal Reflux Disease
During sleep, the esophageal mucosa has prolonged contact with acid. Furthermore, acid clearance is reduced. As a result, chronic acid reflux is often associated with frequent arousals during sleep. In addition, gastroesophageal reflux disease (GERD) is associated with increased risk of aspiration, particularly in children with neurodevelopmental problems such as cerebral palsy. GERD may disrupt sleep because of secondary chronic cough and other respiratory symptoms as well as heartburn. GERD is also considered to be a risk factor for OSA because of the associated mucosal edema. Children with reflux demonstrate an increased number of apneas and hypopneas, particularly during REM sleep. Antireflux medications that have serotonergic effects, such as cisapride, may also result in sleep disruption.

Headaches
Migraine headaches have been associated with sleep-onset and maintenance problems, as well as reduced sleep duration. On PSG, adults with migraines show an increase in both SWS and REM sleep. Children with chronic and severe migraine headaches also have disrupted sleep architecture, including reduced total sleep time, prolonged sleep-onset latency, increased arousals, and reduced SWS and REM sleep. These changes in sleep architecture appear to be associated with both headache severity and chronicity. Some studies have reported an increased prevalence of SRBDs and PLMDs in pediatric patients with migraine. Parasomnias such as sleepwalking are also reported to be more common. Furthermore, sleep loss may trigger migraines as well as other types of headaches in susceptible individuals, and migraines may awaken the child from sleep or early in the morning. The underlying pathophysiology of this relationship between migraines and sleep disruption has been postulated to involve abnormalities in the serotonergic system. The prodromal symptoms of migraine, such as drowsiness, mood dysregulation, and behavioral hyperactivity, also suggest a direct role for the dopaminergic system. Alterations in melatonin have also been proposed, and melatonin has been used clinically as prophylaxis for migraines in both adults and children.

More sleep problems, including insomnia and EDS, have been reported to occur with other types of headaches in children. With tension headaches, this may be related to increased stress and anxiety. Children with tension
headaches also appear to have reduced total sleep time and frequent awakenings on PSG. In addition, the risk of bruxism may be increased two-fold with both tension and migraine headaches two-fold. Rebound headache related to frequent use of analgesic medications and secondary depression and anxiety may be contributing factors in some children. Finally, not only does SRBDs seem to be more common in children with headaches in general, but children with OSA may complain of morning headaches on waking, presumably related to elevated CO₂ levels and/or poor-quality sleep.

**Juvenile Rheumatoid Arthritis (JRA)**

As noted above, any chronic medical condition that is characterized by recurrent pain may be associated with sleep disruption. Children with JRA report more sleep disturbances compared to healthy controls, including frequent nightwakings and sleep fragmentation, which may be associated with daytime sleepiness. However, while pain severity and frequency appear associated with some aspects of sleep disturbances, neither pain nor functional disability has been found to be consistently related to sleep, suggesting that these relationships are complex and likely bidirectional. PSG data have shown that children with JRA have shorter SWS and REM sleep stages, as well as more arousals, PLMs, and alpha intrusion.

**Meningomyelocele and Chiari Malformations**

Meningomyelocele (especially thoracic and thoracolumbar) and Chiari malformation type II can be associated with blunted ventilatory and arousal responses, central hypoventilation syndrome, and increased central and obstructive apneas. It is estimated that about one-third of the children with meningomyelocele have moderate to severe ventilatory abnormalities; patients with thoracic or lumbar lesions, restrictive lung disease, and wheelchair-dependent patients with scoliosis are at particularly high risk for SDB. Most individuals with type I Chiari malformation are asymptomatic, but SRBD (both obstructive and central apneas) can be a rare complication. Surgical decompression of the posterior fossa in Chiari malformation or tethered cord release in children with meningomyelocele may be necessary. Adenotonsillectomy can also improve obstructive disease, but positive pressure mechanical ventilation with continuous positive airway pressure or bilevel positive airway pressure is often needed, especially in children whose respiratory function is further compromised by kyphoscoliosis or obesity.

**Neuromuscular Disorders**

SRBDs are more common in children with a variety of neuromuscular conditions, including Duchenne muscular dystrophy, Charcot-Marie-Tooth disease, myotonic dystrophy, and congenital myopathies characterized by hypotonia. In some of these conditions, the SRBD is a result of respiratory muscle weakness, and in others, it is related to reduced central ventilatory drive. The risk of respiratory desaturations or hypoxemia is greatest during REM sleep. Many of these children also have reduced vital capacity associated with scoliosis, which may further compromise respiratory function and exacerbate SRBD. Many of these patients do not report OSA-associated symptoms (e.g., snoring, daytime sleepiness, morning headaches) unless specifically asked, so that frequent screening for SRBD symptoms is mandatory.

Increased risk for respiratory failure is also associated with generalized or proximal muscle weakness in these children, particularly those with diaphragmatic involvement and those who have had an earlier age of onset of symptoms. Significant SRBD can occur in the setting of even mild respiratory muscle weakness. The hypoxemia and chronic respiratory failure in these patients is usually preceded by periods of increased end-tidal CO₂ during sleep. Daytime blood gases may be normal. Pulmonary function tests are often noncontributory but may show restrictive lung disease, and there may be minimal symptoms of SDB. Furthermore, pulmonary function testing to assess maximal inspiratory and expiratory pressures can be helpful to assess the severity of muscle weakness.
Sleep and Neurologic Disorders

The primary care practitioner should maintain a high level of suspicion for SDB, particularly in children with neuromuscular disorders and spina bifida, as this may be overlooked in the context of multiple other medical needs. Close collaboration with a multidisciplinary neurorehabilitation team for follow-up is strongly recommended.

Phenylketonuria

Phenylketonuria may be associated with difficulties in sleep onset and maintenance as well as nightmares related to an increase in REM sleep. These sleep disturbances may be a result of alterations in the phenylalanine/tyrosine/tryptophan ratio. A balanced phenylalanine-restricted diet may be helpful in reducing sleep disruption.

Renal Failure

Sleep disruption in children with chronic renal failure may be related to a number of factors, including medications, dialysis, or decreased urine output. There is an increased prevalence of restless legs syndrome and PLMD (see Chapter 16) in chronic renal failure, which may lead to delayed sleep onset, fragmented sleep, and EDS. Increased daytime somnolence may also be a result of increasing renal failure.

Sickle Cell Disease

Parents of children with sickle cell disease report more nightwakings, restless sleep, and parasomnias compared to healthy children. A number of factors potentially contribute to sleep disruption in these children, including painful nocturnal (vaso-occlusive) crises, infections (especially pneumococcal), cerebrovascular accidents, anemic episodes (aplastic or sequestration crises), nocturnal enuresis, and direct and indirect effects of analgesics, including opiates. These children may also be prone to SRBD as a result of adenotonsillar hypertrophy related to compensatory lymphoid hyperplasia with functional asplenia, repeated bouts of tonsillitis infections, and airway narrowing related to the anatomic effects of bone marrow hyperplasia. Nocturnal oxygen desaturations are common, especially in children with the HbSS genotype. Hypoxemia related to OSA may lead to increased nocturnal sickling and vaso-occlusive crises, with resultant exacerbation of the disease. Several studies have reported associations among nocturnal hypoxemia, SRBD, and sleep disruption secondary to pain. Hypoxemia may significantly improve with adenotonsillectomy, although clearly these children are at higher surgical risk for perioperative complications as a result of their disease. A recent PSG study reported alterations in sleep architecture, including prolonged sleep-onset latency, decreased total sleep time, increased wake after sleep onset, and reduced sleep efficiency. An increase in obstructive events and PLMs has also been described.

Traumatic Brain Injury

Sleep disturbance is a common complaint following head injury, especially in patients with milder (compared to more severe) trauma. This apparent discrepancy may be due to less attention paid to sleep problems in more severely affected patients and/or a more pressure to rapidly return to preinjury status in mild traumatic brain injury (TBI), resulting in increased stress and anxiety. Common sleep issues include difficulties initiating and maintaining sleep (occurring in up to half of patients), decreased sleep quality, reduced sleep efficiency, and early morning waking, with or without associated daytime sleepiness. Sleep disturbances are also significant predictors of poorer functional outcomes in children with moderate or severe TBI, and may delay rehabilitation and recovery due to its negative impact on mood and pain perception. Causation can be due to diverse clinical and other factors, such as pain, injury to sleep-wake regulatory pathways in the brain, and PTSD symptoms.
Post-TBI insomnia may also be a manifestation of a circadian rhythm sleep-wake disorder, such as delayed sleep-wake phase or irregular sleep-wake type. In some cases, there may be an iatrogenic contribution resulting from recommendations to sleep “ad lib” and avoid physical activity, resulting in disruption of both the homeostatic and circadian sleep-regulating systems.

Changes occurring in sleep architecture during the acute phase following head injury electroencephalography include increased SWS. These sleep abnormalities may persist for several years. Furthermore, significant hypersomnia with or without involuntary sleep attacks, so-called “secondary narcolepsy” or “posttraumatic hypersomnia,” may develop and persist for a number of years after the trauma, often with accompanying impairments in concentration, memory, and cognition (executive functioning). In a handful of cases, TBI has also been reported to precipitate Kleine-Levin syndrome (a rare disorder consisting of recurrent hypersomnia and cognitive or behavioral disturbances, hypersexuality, and compulsive eating). An increase in parasomnias, including sleepwalking, sleep terrors, and REM behavior disorder, has also been described in association with TBI. Nonpharmacologic treatments for TBI-related insomnia include cognitive behavioral therapy for insomnia, as well as sleep scheduling and phototherapy for circadian sleep-wake rhythm disturbances. Patients with narcolepsy or hypersomnia may require stimulant medications such as modafinil, methylphenidate, or amphetamines.

**SLEEP AND OBESITY**

Childhood obesity is a national epidemic in the United States, with over one-third of US children and adolescents 2 to 19 years old considered overweight or obese. The high prevalence and myriad of associated health consequences have led to an intensive search for possible contributing factors, which could impact both treatment and prevention. A number of both cross-sectional and longitudinal studies published in the past 10-15 years have demonstrated an association between short sleep duration and both increased weight and anthropometric measures of body fat in both adults and children. This relationship appears to be more robust in children versus adults and to persist despite controlling for a number of potential confounding factors, such as diet, exercise, parental obesity, and television viewing. Most of the evidence comes from pediatric cross-sectional studies, which have demonstrated a dose-dependent relationship between decreased sleep amounts and increased weight. Prospective cohort studies in a number of pediatric populations around the world have also shown that short sleep duration at Time A, particularly before the age of 3 years, predicts weight status at Time B (e.g., in middle childhood), independent of weight status at Time A. Furthermore, the demographic groups at increased risk for obesity are also the same populations (blacks and Hispanics and those with low socioeconomic status) at increased risk for late bedtimes and insufficient sleep duration. However, the relationship between short sleep duration and increased risk of obesity in infants, children, and adolescents is a highly complex and hardly straightforward one. For example, a recently published national survey of US households, in which caregivers of over 102,000 US children were surveyed, failed to find an association between parent-reported insufficient sleep and obesity after adjustment for sociodemographic variables. Thus, while a general outline of the identified contributing factors in the relationship between sleep and obesity is presented below, for a more detailed discussion, the reader is referred to additional references and reviews.

In addition to sleep duration, timing of sleep may play a role in childhood obesity risk. For example, late bedtimes (but not rise times) appear to be associated with overweight/obesity. With regard to weekend “catch up” sleep, results of a recent study suggested that compared to children who get more than 10 hours per night, children who persistently sleep 8 hours or less on school days and nonschool days are at the greatest risk for obesity.
Clinical Practice Recommendations: Sleep and Obesity

Screen

- Pediatric providers should screen all overweight and obese adolescents for insufficient sleep as a risk factor for weight gain and failure to achieve weight loss.

Educate

- Parents and adolescents should be educated about the relationship between insufficient sleep and weight gain and obesity, and metabolic dysfunction and impaired glucose metabolism.

The mechanisms underlying this association between sleep and weight are still unclear but likely to be multifactorial involving neurohormonal and metabolic alterations, including increased leptin and decreased ghrelin, the combination of which is associated with increased hunger, appetite, motivation to eat, and food intake. Furthermore, recent experimental studies with adults have demonstrated increases in caloric intake when sleep is restricted, suggesting that less sleep may increase the risk of obesity via neuroendocrine changes. In addition, sleep loss appears to increase insulin resistance by increasing circulating cortisol, activating the sympathetic nervous system, and upregulating proinflammatory markers such as C-reactive protein. Fatigue associated with insufficient sleep may lead to an increase in sedentary activities and a decrease in physical activity and calorie expenditure. Behavioral, mood-related, and cognitive mechanisms linked to insufficient sleep may also play a role, especially in children. For example, parents may use food to pacify an irritable, sleep-deprived child, and children themselves may use food to self-soothe; alternatively, caregivers may have difficulty setting limits on both eating and sleep behavior. In older children and adolescents, sleepiness-related impairments in impulse control, judgment, and motivation (i.e., executive functions) may contribute to overeating and the development of obesity.

It should also be emphasized that the relationship between short sleep and obesity is further complicated by the presence of SDB, the risk for which is increased not only by obesity, but by which is more relevant in the same populations at risk for inadequate sleep (e.g., minority and poor children). Comorbid SRBD may further intensify the metabolic consequences and longterm cardiovascular morbidity associated with both obesity and inadequate sleep (e.g., insulin resistance and glycemic control, systemic inflammation), and insufficient sleep may add to the neurocognitive sequelae of SRBD (e.g., impairments in attention and memory, academic failure) in a kind of “perfect storm” scenario (see Chapter 15).

Sleep and Obesity

There appears to be an important bidirectional relationship between sleep and obesity. A number of studies have convincingly supported a robust association between short sleep duration and an increased risk of being overweight and obesity in the pediatric population. The underlying mechanisms are likely to involve alterations in neurohormonal and metabolic pathways as well as behavioral components. Alternatively, obesity increases the risk for SRBDs and other sleep disturbances. Thus, systematic screening for both inadequate sleep duration and symptoms of SDB in overweight and obese children, especially in high-risk groups, is an important role for primary care practitioners.

SLEEP AND SEIZURE DISORDERS

Although a detailed description of the complex interaction between epilepsy and sleep is beyond the scope of
this book, a number of important points relevant to the primary care physician must be mentioned. It is estimated that 20% to 40% of seizures in childhood occur during sleep, and sleep related epilepsy accounts for about 30% of seizure disorders in children. The most commonly encountered types of sleep related epilepsies (i.e., occurring during sleep or following arousal) include frontal and temporal lobe partial epilepsies in adults, benign epilepsy of childhood with centrotemporal spikes, benign rolandic epilepsy (BRE), and juvenile myoclonic epilepsy in children and adolescents. In one study, about half of first-time unprovoked seizures occurred during sleep. Furthermore, the likelihood of recurrence is significantly increased (42% versus 18%) in those children who presented with nocturnal seizures.

Childhood seizures are especially prominent in lighter stages of sleep. While interictal epileptiform discharges are differentially activated during SWS (N3), ictal seizure events occur more frequently during NREM stages N1 and N2. A significant percentage of seizures also occur at the sleep-wake transition (e.g., infantile spasms, some tonic-clonic seizures). In general, seizures are uncommon during other sleep stages, such as SWS, although partial complex seizures may occur solely in REM sleep. Some seizures, such as juvenile myoclonic epilepsy or generalized tonic-clonic seizures upon awakening, appear to be activated by arousal and often occur within the first hour after awakening. Finally, circadian factors may also play a role in the timing of seizure activity.

**Seizures and Sleep**

In contrast to prevalence in adults, nocturnal seizures are quite common in children, and some types of seizures (e.g., BRE, juvenile myoclonic epilepsy) occur predominantly during sleep. Nocturnal seizures may cause significant sleep disruption and daytime sleepiness. Conversely, sleep loss is a well-established trigger factor for seizures.

BRE is a common, autosomal dominant form of epilepsy with features that include simple partial seizures with hypersalivation and drooling, hemifacial focal motor clonic, and secondary generalized tonic-clonic seizure activity, as well as subjective sensations of tingling, twitching, and/or numbness that may occasionally spread to generalized tonic-clonic activity. Seizures are confined to sleep in approximately three-quarters of patients. Mean age of onset is approximately age 7 (range 3-13 years), with recovery by mid-adolescence. A family history is common. Development is normal, and treatment with anticonvulsants is only occasionally necessary.

Juvenile myoclonic epilepsy is an autosomal dominant idiopathic primary generalized epilepsy syndrome characterized by myoclonic jerking of the extremities, absence and generalized tonic-clonic seizures, and usually occurs shortly after arousal, although may also occur during sleep or during the day. In contrast to BRE, mean age of onset is in mid-adolescence and most patients require lifelong medication management. Finally, generalized tonic-clonic seizures upon awakening have a similar pattern of seizure occurrence to juvenile myoclonic epilepsy, but does not have a myoclonic component.

Seizures (including rolandic epilepsy, tonic-clonic) may cause sleep disruption, and there is a higher incidence of sleep disturbances in patients with epilepsy in general. Furthermore, comorbid sleep disorders are frequent in patients with epilepsy and may increase seizure burden. Seizure activity does not necessarily need to be overt to disrupt sleep but rather may consist of brief stereotypic arousals and “microarousals” that result in significant daytime sleepiness. Successful anticonvulsant therapy may alleviate daytime somnolence even in the absence of frank seizure activity, although anticonvulsants may clearly cause sedation as well. Neurodevelopmental disorders (e.g., severe developmental delay, blindness) that are commonly associated with seizures as part of their clinical presentation may also predispose the patient to sleep disorders. Sleep disorders and sleep loss may also exacerbate seizures in patients with known epilepsy. For example, hypoxia and sleep fragmentation.
associated with OSA may trigger seizures in a predisposed child; on the other hand, OSA treatment may reduce seizure frequency.

Sleep Studies and Seizures

Because not all sleep laboratories perform a full EEG seizure montage as part of routine PSG, an overnight sleep study alone may not be sufficient to rule out nocturnal seizures. Additional diagnostic procedures, such as 24-hour ambulatory EEG monitoring, may be necessary in cases in which there is a high index of suspicion.

Distinguishing nocturnal seizures from other episodic nocturnal events such as NREM parasomnias (confusional arousals, sleep walking, and night terrors) and REM parasomnias, including REM sleep behavior disorder, can be challenging (see Chapter 11 for complete coverage of differentiating seizures from parasomnias). Therefore, a careful history is imperative. Parental video recording of events in the home setting can be a helpful screening tool; full night in-lab video-EEG-PSG is necessary in selected cases. Important potential differentiating factors between seizure and other nocturnal phenomena include timing of the events and arousal threshold (indicative of sleep stage); level of autonomic arousal; presence of stereotypic or unusual behaviors such as drooling, incontinence, tongue biting; recall for the event; and daytime sleepiness. The differential diagnosis also includes other primary sleep disorders: bruxism, rhythmic movement disorders, nocturnal myoclonus and sleep starts, PLMs, OSA, REM behavior disorder (extremely rare in childhood and typically reported only in the setting of CNS pathology or narcolepsy), enuresis, hypnagogic hallucinations and sleep paralysis associated with narcolepsy, nocturnal physiologic arousals related to medical conditions (e.g., GERD, asthma), psychiatric disorders (nocturnal panic attacks, PTSD), and psychogenic seizures.

Finally, although effective treatment of nocturnal seizures may substantially improve sleep quality, anticonvulsants may also compromise sleep by causing daytime sedation and impacting sleep architecture. Phenobarbital acts as a REM sleep suppressant, while phenytoin and carbamazepine increase SWS; lamotrigine increases REM percentage. Withdrawal from anticonvulsants following discontinuation can also result in sleep difficulties. For example, anticonvulsants associated with decreased REM sleep may lead to REM rebound symptoms (e.g., increased nightmares) on withdrawal, and discontinuation of GABA-ergic medications like benzodiazepines may result in increased SWS. Oftentimes, however, appropriate treatment with anticonvulsants to eliminate or reduce nocturnal seizure activity actually improves sleep by decreasing sleep-onset time and nighttime arousals.

TREATMENT

Management of sleep problems in children with acute and chronic medical conditions first requires recognition on the part of the practitioner of the existence of possible risk factors for sleep disturbances and the high likelihood of their occurrence, particularly in some disease states and in certain settings, as outlined above. Symptoms such as fatigue, mood changes, and, particularly, daytime sleepiness in chronically ill children should not be automatically attributed to the underlying disease process, and every effort should be made to screen for and address primary and secondary sleep problems when they occur. Medication choices should take into consideration any possible negative effects on sleep. In addition, those pain medications likely to disrupt sleep or significantly alter sleep architecture, such as opiates and benzodiazepines, should be avoided or used for short time intervals. In contrast, nonsteroidal anti-inflammatory and nonopiate analgesics, especially when used in the short-term, appear to have little overall
Approaches to pain management in children that may have particularly beneficial effects on sleep as well include the following:

- Use of relaxation techniques, including hypnosis and biofeedback.
- Use of other nonpharmacologic pain measures such as warm baths, massage, and application of warm or cold compresses.
- Incorporation of cognitive behavioral therapy aimed at management of chronic pain and development of age-appropriate and adaptive coping skills.
- Support of parent education and modeling of good sleep habits.
- Maintenance of regular daytime and particularly bedtime routines.

**Consultation for Sleep Issues**

Children with chronic medical conditions and sleep issues, and hospitalized children in particular, often benefit from consultation with a child life specialist and/or behavioral therapist regarding specific management strategies. Additional input from a consultation-liaison child psychiatry team may be beneficial if pharmacologic intervention is being considered.

Any child with a medical condition that may pose an increased risk for SRBD (e.g., neuromuscular disorders, chronic allergies) should be monitored regularly for the development of possible signs and symptoms, such as snoring, restless sleep, and daytime sleepiness. A high index of suspicion and a low threshold for obtaining an overnight sleep study evaluation is particularly important in these children. Ongoing consultation with a pediatric sleep specialist, child neurologist, and/or pulmonologist is recommended.
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Sleep and Psychiatric Disorders

GENERAL CONSIDERATIONS
The relationship between disturbed sleep and psychiatric disorders in children and adolescents is one that is complex, but highly clinically relevant for both mental health professionals and primary care practitioners. Studies indicate that sleep disturbances are very common in children with mental health concerns. These sleep problems often have a significant impact upon both the clinical presentation and symptom severity of psychiatric disorders and pose significant management challenges in clinical practice. Virtually all psychiatric disorders in children and adolescents may be associated with sleep disruption. The spectrum of sleep disturbances ranges from difficulties with initiating and maintaining sleep and complaints of poor quality sleep, to daytime fatigue and sleepiness, and increased sleeping (hypersomnia), to circadian rhythm dysregulation. Treatment of psychiatric disorders with psychotropic medications that potentially have significant negative effects on sleep often adds an additional layer of complexity. In addition, growing evidence suggests that insomnia is a risk factor for the later development of psychiatric conditions, particularly depression and anxiety disorders. Finally, there is considerable overlap between symptoms of mental health disorders, such as mood lability, inattention, and disruptive behaviors, and those associated with a wide range of sleep disorders, including obstructive sleep apnea (OSA; see Chapter 15) and restless legs syndrome (RLS) and periodic limb movement disorder (PLMD; see Chapter 16).

Given the prevalence of sleep problems associated with psychiatric conditions, it is important that both healthcare practitioners and mental health providers develop a systematic approach to screening, diagnosing, and managing the spectrum of sleep disturbances in all children presenting with psychiatric, behavioral, and academic concerns. Although a detailed discussion of the management of sleep concerns in specific psychiatric disorders is beyond the scope of this chapter, and many of these patients will require referral for more intensive behavioral and pharmacologic management, a general approach to understanding and evaluating sleep problems in the most common psychiatric disorders in childhood is highlighted below.

Sleep and Neurobehavioral Symptoms: A Bidirectional Relationship
There is clearly a bidirectional relationship between sleep and neurobehavioral symptoms, including mood, cognitions, and behavioral regulation in childhood. Insufficient sleep often results in neurobehavioral symptoms, including mood dysregulation, cognitive dysfunction, impairments in executive functions, including inattentiveness, poor judgment, disorganization, increased impulsivity, and risk taking behavior, and behavior problems. Conversely, children with mood and behavior disorders often experience sleep disturbances.

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER
General Description
While many of the core diagnostic criteria for attention-deficit/hyperactivity disorder (ADHD) in Diagnostic and Statistical Manual, 5th ed. (DSM-V) remain the same, there are some important differences compared to DSM-IV-TR Symptom presentation now needs to occur by age 12 years (rather than by age 6); a number of symptoms now need to be present in more than one setting; and for adults and adolescents age 17 or older, only five symptoms are needed instead of the six needed

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for younger children. Sleep problems, particularly difficulty initiating and maintaining sleep, are commonly
reported in children and adolescents with ADHD (including in predominantly hyperactive-impulsive,
predominantly inattentive, and combined subtypes). Parents of children with ADHD frequently complain of
bedtime struggles and delayed sleep onset as well as increased nightwakings, restless sleep, and shortened
sleep duration; however, it should be noted that there is often a discrepancy between these subjective
complaints and more “objective” measures of sleep such as polysomnography (PSG) and actigraphy. In addition,
the relationship between sleep problems and ADHD is not straightforward, as there is considerable overlap
between many of the most common behavioral consequences of insufficient or disrupted sleep in children and
the symptoms of ADHD (i.e., problems with attention and focusing, hyperactivity, poor impulse control, irritability
and disturbed mood, and increased acting-out behaviors such as oppositionality and aggression). In fact, a
number of studies have now suggested that a percentage of children labeled as “ADHD” actually have a primary
sleep disorder that accounts for their symptoms. Although the number of children misdiagnosed in this way is not
known, one study reported that up to 25% of children with ADHD symptoms also had symptoms of sleep-
disordered breathing, and in another study, more than half of children evaluated for ADHD had evidence of RLS
and/or PLMD. Furthermore, sleep disorders can add to the severity of ADHD symptoms when they coexist.

Sleep Disorders and ADHD
The relationship between sleep disturbances and ADHD is a complex and multifactorial one. Sleep
problems in these children may be related to a variety of issues, including coexisting primary sleep
disorders, comorbid psychiatric conditions, or concomitant psychotropic medications; in some children, a
more “intrinsic” ADHD-related difficulty with settling may be present. A thorough and systematic search for
possible etiologic factors is necessary in order to make an accurate diagnostic formulation and develop the
most appropriate treatment plan.

Epidemiology
Parent-reported mild to severe sleep problems, particularly difficulties in initiating and maintaining sleep, are
reported in as many as 70% of children with ADHD. Prevalence rates appear to differ somewhat as a function of
ADHD subtype (highest prevalence in the combined subtype, although sleepiness may be more common in the
inattentive subtype). These prevalence rates are generally at least 2 to 3 times that of children without ADHD.
Differences in the rates of sleep problems are also found within ADHD samples as a function of psychiatric
comorbidities and medication use.

A recent meta-analysis of sleep problems in children with ADHD found that children and/or their parents reported
a wide range of issues. Bedtime resistance, prolonged sleep-onset latency (SOL), nighttime awakenings,
difficulties with early morning awakening, sleep-disordered breathing, and daytime sleepiness were reported
more frequently compared to typically developing controls. The most commonly reported problem was “difficulty
falling asleep.”

Etiology
From a theoretical perspective, there is substantial empirical evidence supporting an overlap in those central
nervous system (CNS) centers as well as neurotransmitter pathways that regulate sleep and those that regulate
attention and arousal, suggesting disruptions in one system might well have an impact on the other. Six primary
areas of interest have been identified: (1) brain centers regulating sleep, arousal, and attention; (2)
neurotransmitter systems involved in both sleep and attention regulation (e.g., catecholamines norepinephrine α²

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and dopamine D\(^1\); (3) alterations of neural systems regulating sleep in ADHD; (4) phenotypic similarities between behavioral, mood, and cognitive manifestations of insufficient/disrupted sleep and ADHD; (5) hypoarousal and sleepiness in ADHD; and (6) external sleep-wake signals that affect sleep regulation in ADHD. The framework for the common underlying CNS pathology linking sleep disruption or deprivation and ADHD primarily involves the prefrontal cortex, which houses complex cognitive or “executive” functions (e.g., time management, decision-making, organization, selective attention, judgment, motivation, monitoring and modifying behavior, predicting outcomes), and related brain structures (e.g., dorsolateral and ventrolateral prefrontal and dorsal anterior cingulate cortices). Both acute and chronic sleep loss under both experimental and “real world” conditions also selectively negatively impact other brain functions critical to ADHD, such as the amygdala, which regulates emotional responses and regulation, and the striatum, which controls reward-related behaviors. For example, insufficient sleep is linked to changes in reward-related decision-making, resulting in a tendency to take greater risks accompanied by a lower level of concern about the potential negative consequences of that risky behavior, a common feature in children with ADHD.

In regards to other CNS mechanisms potentially linking sleep and ADHD, a role for a primary circadian disruption in ADHD has also been proposed, which appear to involve alterations in the circadian clock's timing or responsiveness to environmental cues such as light and may reflect specific genetic alterations, such as those of CLOCKQ1 genes. For example, a number of studies have documented the presence of a circadian-based phase delay in children with ADHD, as evidenced by a delayed onset of nocturnal melatonin secretion compared to controls. Finally, a few studies that have examined the issue of arousal level and daytime sleepiness in children with ADHD have reported that children with ADHD demonstrate an increase in objectively measured sleep propensity compared to control children, suggesting that hyperactivity, at least in some children, may actually be an adaptive behavior that counteracts the effects of hypoarousal and underlying daytime sleepiness.

Despite this, studies do not in general demonstrate reliable objective differences in either sleep architecture and/or sleep patterns in children with ADHD compared to typically developing controls, with the possible exception of increased activity levels and movement during sleep and more night-to-night variability in sleep patterns. However, in direct contrast to studies that have utilized more objective measures, parental report surveys have almost universally reported a higher frequency of significant sleep problems in children with ADHD compared with various control groups, which is reflected in the clinical experience of most healthcare practitioners who work with these children. It has been suggested that this discrepancy between objective measures and subjective reports may be related to the fact that parents of behaviorally disordered children may be more likely to perceive and report high levels of both daytime and sleep related disruptive behaviors; in addition, both caregivers and clinicians may be relatively more vigilant about observing and eliciting evidence of sleep problems in these children. Caregivers may be more likely to remember and report extremes in sleep behaviors, such as a 2-hour sleep onset latency (SOL), even if these occur only occasionally. Finally, what parents attribute to “sleep” problems may actually be more accurately described as problematic “evening behaviors” (e.g., increased activity level, difficulty settling, “oppositionality”) that may either be due to untreated ADHD symptoms, a recurrence of ADHD symptoms once medication has worn off at the end of the day, or an actual increase in ADHD behaviors related to decreased drug levels (so-called “rebound”).

Because the etiology of sleep problems in children with ADHD is so varied and often multifactorial, a systematic approach is necessary to tease out the relevant contributory factors. The discussion below and Figure 23.1 outlines the major issues that should be considered; it should be noted that many of these etiologic factors may also play an important role in sleep problems in children with other psychiatric disorders, such as depression and anxiety, and thus the evaluation algorithm outlined in Figure 23.1 serves as a useful template for examining sleep problems in other mental health conditions as well.
A primary sleep disorder may result in ADHD-like symptoms: A patient with any sleep disorder that results in insufficient sleep duration or fragmented or disrupted sleep may present primarily with daytime sleepiness and neurobehavioral symptoms, many of which overlap with the core symptoms of ADHD. For example, a large number of pediatric studies have now been published that provide compelling evidence of a wide range of neurobehavioral and neurocognitive deficits in children with both clinical symptoms of and polysomnographically confirmed OSA, including inattention, impaired memory, and executive functions, mood disturbance, externalizing and internalizing behavior problems, and academic difficulties (see Chapter 15). Furthermore, studies that have looked at changes in behavior and neuropsychological functioning in children following treatment for OSA (e.g., adenotonsillectomy) have also largely reported significant improvements in daytime sleepiness, neuropsychological measures of impairment, behavior, and academic performance. Significant neurobehavioral consequences may also occur related to RLS and PLMD, and may present as symptoms of ADHD. Delayed sleep-phase disorder and narcolepsy are other sleep disorders that have been reported to result in “ADHD-like” symptoms.

Coexisting sleep disturbances may exacerbate the symptoms of ADHD: Sleep disorders such as OSA may also occur in conjunction with ADHD, and the cognitive, mood, and behavioral disturbances associated with ADHD may be worsened by their presence. Insufficient sleep related to environmental or lifestyle factors (e.g., erratic schedules), particularly if chronic, may also exacerbate existing ADHD symptoms. Some children with ADHD, especially older children, may have a delayed sleep-wake phase, which may contribute to difficulties settling at bedtime, prolonged sleep onset, and difficulty waking in the morning for school. Treatment of comorbid sleep disorders and interventions targeted at ensuring adequate sleep may
Coexisting psychiatric disorders may be the underlying cause of sleep disturbance in a child with ADHD: The majority of children with ADHD (about 70%) have comorbid psychiatric disorders in addition to ADHD that may further contribute to sleep problems. The most common disorders co-occurring with ADHD in younger children are oppositional defiant disorder and anxiety disorders. Among older children and adolescents, depression or underlying mood disorders begin to become more common. For example, limit-setting problems associated with disruptive, impulse-control, and conduct disorders (DSM-5), such as oppositional defiant disorder (ODD; 40%-60% of children with ADHD), may result in bedtime resistance. An estimated 30% of children with ADHD have a comorbid anxiety disorder and 10% to 30% a comorbid mood disorder; sleep-onset problems, particularly relating to bedtime fears and need for parental presence at bedtime, are common presenting complaints in children with anxiety disorders, and sleep initiation complaints are common in depression. Problems with sensory integration and associated difficulties in self-soothing may also predispose children with ADHD to settling and sleep-onset problems.

Pharmacologic agents used to treat ADHD and/or comorbid psychiatric conditions may cause or exacerbate sleep problems: In particular, psychostimulant medication may be associated with sleep-onset and maintenance problems as well as restless sleep. Sleep problems may be a direct effect of the medication itself. In other cases, stimulant medication may wear off just before bedtime, resulting in “rebound” hyperactivity and, indirectly, sleep-onset difficulties. Finally, other psychiatric medications used in children with ADHD, including different classes of antidepressants (see Chapter 20), may also potentially either alleviate or exacerbate sleep problems.

CNS dysregulation of arousal and activity associated with ADHD may result in sleep disturbances: Although, as discussed above, the presence of an “intrinsic” dysfunction linking sleep disturbances and ADHD is still largely speculative at this point, difficulties in “settling” or “turning off” at bedtime are commonly reported in children with ADHD. Significant problems with delayed sleep onset appear to occur in some children in the absence of other identifiable causes, such as use of medication or comorbid psychiatric disorders. It has been postulated that dysregulation of the homeostatic sleep drive and resulting alterations in the normal build-up of sleep propensity with continuous wakefulness may be involved. Alternatively, some of these children may have a primary circadian disruption (e.g., delayed sleep-wake phase).

Unhealthy sleep practices may contribute to sleep problems in ADHD. Sleep-inhibiting practices such as late and/or inconsistent (or nonexistent) bedtimes, evening “screen” exposure (computers, televisions), caffeine consumption, and lack of bedtime routines have been shown to negatively impact sleep in typically developing children (see Chapter 5). These practices may be even more common in families of children with ADHD. On the other hand, studies suggest that addressing these maladaptive sleep habits may have a positive effect on sleep and may even reduce the need for pharmacologic interventions such as melatonin. Another common concern encountered in clinical practice is middle-of-the-night eating. These are typically children on high doses of appetite-suppressing psychostimulants who “raid the refrigerator” after medication effects have worn off, adding further to disrupted sleep.

Evaluation

Screening for sleep disorders should be part of the evaluation for every child with suspected ADHD: Screening questions regarding sleep onset, nightwakings, restless sleep, snoring, sleep regularity and duration, and daytime sleepiness should be part of every ADHD evaluation (see Chapter 3). Sleep diaries (and in some cases actigraphy) can be particularly helpful in assessing night-to-night variability and providing
a more accurate assessment of sleep patterns. Clinical suspicion of a sleep disorder following screening warrants a more detailed clinical assessment of the severity (e.g., time to fall asleep), frequency (e.g., nights per week), and duration (e.g., number of months or years symptom has been present) of the presenting complaint. Further diagnostic testing may be indicated.

Sleep Diagnostic Tests in ADHD
Polysomnography is considered the “gold standard” in children for the diagnosis of OSA syndrome and PLMD, and should be strongly considered if there is clinical suspicion of symptoms (e.g., snoring, restless sleep) and/or risk factors (e.g., adenotonsillar hypertrophy, positive family history) of either. A multiple sleep latency test should be reserved for situations in which there is documented pathologic sleepiness (e.g., narcolepsy).

Because of the clinical association between ADHD and RLS and PLMD, and recent studies which suggest that iron deficiency is a risk factor for both RLS/PLMD and ADHD, a serum ferritin level should be considered, particularly in populations at increased risk for iron deficiency (e.g., young children, adolescent girls). Although there is no formal consensus of the cut-off indicating iron deficiency in children, a value of 50 mg/L has been proposed as indicative of conveying an increased risk of RLS.

Periodic screening for sleep disorders should be part of the ongoing management of every child with diagnosed ADHD: Comorbid psychiatric conditions and associated sleep problems may evolve in children with ADHD. Changes in medication type, dose, and dosing schedule over the course of treatment may result in sleep disturbances. Comorbid sleep problems, such as OSA, may also develop over time, especially in high-risk groups, such as children who are overweight or obese. Thus, the importance of regular clinical assessment for emergent sleep problems is paramount.

Overall evaluation strategy: Figure 23.1 illustrates a comprehensive evaluation strategy for the child who presents with the most common sleep complaint reported in ADHD, that of bedtime refusal or resistance and/or delayed sleep onset (i.e., time to fall asleep once in bed). In younger children, these two symptoms are often indistinguishable; that is, children who have difficulty falling asleep for a variety of reasons often exhibit protest behavior (crying, getting out of bed, calling out to parents) at bedtime. In contrast, older children and adolescents may lie in bed quietly after “lights out,” and parents may not even be aware of the sleep-onset difficulties. Thus, it is important to question the child directly as well as the caretaker about symptoms. The following specific issues are important to assess:

Sleep practices: Many families of children with ADHD have either never developed or have abandoned basic good sleep habits. These include environmental (e.g., low noise and ambient light levels, cool temperature), scheduling (e.g., regular bedtime, sleep-wake schedule), sleep practice (e.g., bedtime routine), and physiologic (e.g., exercise, timing of meals, caffeine use) factors which promote optimal sleep.

ADHD symptoms in the evening and at bedtime: Children on ADHD medications dosed in the morning may experience a return to baseline levels of ADHD symptoms as the medication effects diminish, or have a temporary increase in symptoms above baseline (i.e., “rebound”). The associated hyperactivity and fidgetiness or restlessness may result in problematic evening behaviors and difficulty settling at bedtime. Psychostimulants may also have a direct effect on sleep, resulting in prolonged sleep onset. Conversely, nighttime ADHD symptoms (motor restlessness), as well as symptoms associated with sensory integration deficits, may actually reflect the presence of a primary sleep disorder (e.g., RLS). Finally, for some children, difficulty in settling occurs independent of medication status, suggesting a more intrinsic ADHD-related
mechanism or one related to circadian phase delay.

- **Timing of sleep onset relative to bedtime:** Since some parents will attempt to put a child to bed earlier as a strategy to reduce sleep-onset delay, it is first important to establish that the current bedtime is developmentally appropriate. Older children, particularly adolescents, who fall asleep easily at a later bedtime (and adopt this later bedtime, as well as a later waketime on weekends and during school vacations), may have a circadian-based phase delay (see above).

- **Degree and persistence of bedtime resistance:** In families in which limit setting is particularly problematic, bedtime resistance may be a corollary of the caregivers’ inability to set limits in general. In this situation, parents may need assistance in developing and implementing appropriate bedtime schedules, rules, and routines. Children with ADHD and behavior disorders, such as ODD or obsessive compulsive disorder, may be particularly prone to develop bedtime conflicts.

**Treatment**

- **Diagnostically driven intervention:** It is paramount that treatment for sleep disturbances in ADHD be diagnostically driven and should correspond to the underlying etiology in each individual case. Thus, if an underlying primary sleep disorder, such as OSA or PLMD, is felt to play an etiologic or exacerbating role regarding ADHD symptoms, a prudent approach would then be to treat any documented sleep disorder (e.g., adenotonsillectomy for OSA) before confirming (or rejecting) the diagnosis of ADHD and initiating treatment. The treatment of sleep problems related to comorbid psychiatric conditions should focus on the specific symptoms; for example, treatment of anxiety might involve progressive muscle relaxation, anti-anxiety agents, and/or psychotherapy.

- **Behavioral interventions and sleep hygiene:** Standard behavioral techniques used to treat sleep problems in typically developing children (see Chapters 6 and 7), such as graduated extinction and bedtime fading, have also been found to be highly effective in children with ADHD. Problems with limit setting, for example, generally respond to some combination of decreased parental attention for bedtime delaying behavior, establishment of a consistent bedtime routine that does not include stimulating activities such as television viewing or playing video games, and positive reinforcement (e.g., sticker charts) for appropriate behavior at bedtime. Studies have suggested that adoption of healthy sleep practices alone may be adequate to successfully treat sleep initiation problems in some children with ADHD; thus, it should be a component of every treatment package.

- **ADHD medication adjustments:** Adjustments in the types (e.g., methylphenidate versus dextroamphetamine, stimulants versus nonstimulants, short-acting versus longer duration of action), dose, and dosing schedules of ADHD medications may be warranted if these are suspected of contributing to the sleep problems. In some children, delayed sleep onset may be responsive to a decrease in drug dosage, as sleep related effects of stimulants may be dose dependent. If inadequate evening ADHD coverage appears to be a contributing issue or there appears to be a more intrinsic settling problem, one approach would be to treat with an ADHD medication with 24-hour coverage, such as atomoxetine. An alternative strategy frequently employed in clinical practice is the use of medication to reduce adrenergic tone at bedtime (i.e., -agonists). Evening return of, or rebound in, ADHD symptoms may respond to late-day supplementation with a short-acting psychostimulant. Finally, in patients with comorbid Tourette syndrome, pharmacologic treatment of the tics (e.g., clonidine, clonazepam) may improve sleep quality in these patients.

- **Pharmacologic interventions for sleep:** As an adjunct to behavioral intervention and institution of appropriate sleep hygiene, in cases of severe insomnia, or in that subgroup of ADHD children for whom
sleep problems appear to be “intrinsic” and not apparently attributable to other modifiable factors, medication may be an appropriate intervention. The focus in making medication decisions should be choosing the most appropriate drug for the individual patient's presentation and comorbid conditions (e.g., melatonin or melatonin receptor agonists for delayed sleep-wake phase; iron supplementation or dopamine agonists for RLS; sedating antidepressants for children with comorbid mood disorders; and -agonists or melatonin for sleep initiation insomnia), safety profile, duration of action (short-acting medications for sleep-onset problems; longer-acting medications for sleep maintenance), possible interaction with current psychotropic medications, and potential exacerbation of sleep disorders by psychotropic medication (e.g., selective serotonin reuptake inhibitors [SSRIs] and tricyclic antidepressants may exacerbate RLS and PLMD).

Although there are currently no medications approved for use in children for ADHD-related sleep problems, a variety of medications are prescribed in clinical practice (for detailed information regarding sedatives and hypnotics, see Chapter 20). These include the -agonist clonidine, originally developed as an antihypertensive medication, and sometimes used to treat daytime symptoms of hyperactivity and impulsivity in ADHD, as well as melatonin. A number of newer nonstimulant ADHD drugs such as extended-release clonidine and guanfacine may have clinical utility in selected clinical situations. It should be noted that results of recent clinical trials of the nonbenzodiazepine-receptor agonists zolpidem and eszopiclone specifically for insomnia in children with ADHD largely failed to demonstrate efficacy, and, in the case of zolpidem, were associated with significant adverse events (e.g., hallucinations).

MOOD AND ANXIETY DISORDERS

General Description

As with ADHD, the interaction between sleep disturbances and symptoms of mood dysregulation and anxiety may pose a diagnostic dilemma because of the overlap in symptoms. Studies of mixed clinical psychiatric populations suggest that 40% to 50% of these children have parent-reported nightwakings, nightmares, and difficulty waking in the morning. In addition, there may be therapeutic challenges in setting treatment priorities regarding interventions for mood disorders. For example, some pharmacologic agents for mood disorders may cause sleep disruption and result in worsening of the mood problems. At the very least, the consequences of poor sleep can exacerbate mood disorders, which may lead to additional sleep disturbances in a negative downward spiral.

Specific Disorders

Depression

In DSM-V, the term persistent depressive disorder now encompasses both chronic major depressive disorder and dysthymic disorder. Significant difficulty in initiating and maintaining sleep is commonly associated with childhood depression; it is estimated that some three-quarters of depressed prepubertal children and 90% of depressed adolescents report significant sleep disturbance. Subjective poor sleep quality, sleep-wake cycle irregularities, and disturbing dreams are also common. Up to half of prepubertal children with depression are reported to have terminal insomnia (“early morning awakening”). Hypersomnia, or excessive sleep, is also associated with childhood depression, occurring in as many as 25% of depressed adolescents.

Not only do sleep disturbances persist once the mood symptoms are resolved in approximately 10% of children, the persistence of sleep problems following successful treatment of mood symptoms may in fact predict later recurrence of depression. For example, reduced sleep efficiency and prolonged sleep onset latency appear to be
uniquely powerful predictors of short-term (i.e., within 12 months) depression relapse in adolescents. Depressed children with insomnia also appear to have more severe and chronic mood symptoms, and are more likely to have comorbid anxiety. However, similar to what has been observed in children with ADHD, the lack of objective documentation of prolonged sleep onset latencies and increased sleep fragmentation in depressed children contrasts with the high frequency of subjective sleep complaints.

It is estimated that, at least in young adults, a history of insomnia increases the risk of developing future major depression up to four-fold. Similar results have been found in preadolescents and adolescents. A recent study found that parent-reported severe sleep problems at age 5 years were associated with an almost two-fold increased risk of depression in young adulthood. Chronic sleep loss from any cause increases the risk of depression in both adolescents and young adults. On the other hand, healthy sleep has been identified as a modifiable protective factor for adolescent depression with a sound evidence base.

A number of recent studies have focused on the possible relationship between sleep and risk behaviors, including suicidal ideation and attempts, in adolescents. Sleeping less than 8 hours at night seems to be associated with an almost three-fold increased risk of suicide attempts. Increased risk of the most severe forms of suicidality (attempts requiring treatment) is correlated with significantly shorter sleep duration (total sleep time ≤4 hours). In addition, the risk of suicidal behavior may be similarly increased in adolescents whose parents also have insufficient sleep, raising some interesting questions about multigenerational environmental and/or genetic factors. Furthermore, adolescents with parental-set bedtimes of midnight or later are significantly more likely to suffer from depression and to have suicidal ideation compared with adolescents with parental-set bedtimes of 10:00 p.m. or earlier. Finally, adolescents with comorbid depression and sleep problems are also more likely to abuse substances, such as caffeine, nicotine, and alcohol, potentially further contributing to sleep disturbances.

There is increasing evidence for a biologic basis for the relationship between mood disorders and sleep disturbance; in particular, monoaminergic neurotransmitter systems (e.g., norepinephrine, dopamine, serotonin) play a role in the regulation of both sleep and mood. Electroencephalographic sleep changes consistently found in adults with depression, particularly decreased latency to rapid eye movement (REM) sleep onset and an increased percentage of REM sleep, are considered to be a reflection of this underlying imbalance in monoaminergic and cholinergic activity. Similarly, most antidepressant medications, which primarily affect the norepinephrine and serotonin systems, have powerful REM sleep suppressant effects. Depressed adults also demonstrate decreased slow-wave sleep (SWS) and increased sleep discontinuity on PSG. These sleep architecture abnormalities have not been consistently found in depressed children or adolescents. However, there may be other sleep electroencephalographic markers that have more relevance for the adolescent population. Circadian factors may also play a role in mood regulation; extreme self-reported “eveningness,” a marker of circadian phase delay, has been associated with depression and emotional dysregulation.

Clinical Practice Recommendations: Sleep and Depression

Screen

- Pediatric providers should screen all adolescents presenting with symptoms of depression and mood dysregulation (irritability, dysphoria) for insufficient sleep and sleep problems. Adolescents with depression and sleep loss may be at increased risk for suicidality.
- Pediatric providers should routinely inquire about significant mood changes in their adolescent patients getting less than the recommended 9 hours of sleep per night.

Educate

- Parents and adolescents should be educated about the relationship between insufficient sleep and
**Bipolar/Disruptive Mood Dysregulation Disorder**

Disruptive mood dysregulation disorder (DMDD) is a new condition introduced in the DSM-V to address symptoms that had been previously labeled as “childhood bipolar disorder (BD).” This new disorder can be diagnosed in children up to age 18 years who exhibit persistent irritability and frequent episodes of extreme, out-of-control behavior. While the diagnostic criteria for BD have not changed dramatically in DSM-V, the primary criteria for manic and hypomanic episodes now include an emphasis on changes in activity and energy rather than just mood.

Children with BD very commonly (in up to 95% of young children with early onset BD/DMDD) have significant parent-reported sleep problems, including highly erratic sleep patterns, restless sleep, and intense nightmares. Adolescents with BD appear to have distinct patterns of prolonged sleep onset latency, frequent nighttime awakenings, and increased total time awake. Sleep impairment has also been reported to be associated with mania and depression severity scores, as well as psychosocial impairment ratings. An apparent “decreased need for sleep” without accompanying daytime fatigue or sleepiness, especially during hypomanic periods, appears to be an important characteristic distinguishing BD from other psychiatric disorders. This seemingly decreased sleep requirement and/or insomnia have been reported to occur in up to 84% of children with BD during mood episodes. Furthermore, a period of increasingly shorter sleep intervals may herald the onset of a manic episode, with insomnia more common during the depressive phase.

One small pediatric bipolar study utilizing PSG found decreased sleep efficiency, increased number of awakenings, increased SWS, and decreased REM sleep percentage compared to controls. In contrast to studies of children with ADHD and depression, in this study parent-reported sleep disturbance correlated with PSG findings. Interestingly, an increased prevalence of OSA has been identified in adults with BD. Findings from a small pediatric study suggest that extreme obesity (body mass index >40 kg/m), oxygen desaturation during sleep, and frequent nocturnal awakenings are associated with OSA in children with BD. Finally, medications used to treat BD may result in sleep disruption or daytime sedation (e.g., valproic acid, risperidone) or lead to weight gain, further increasing the risk for sleep disordered breathing.

**Seasonal Affective Disorder**

Seasonal affective disorder (SAD), also known as winter depression, winter blues, or seasonal depression, is characterized by periods of depression that occur only in the autumn and winter months (in the Western hemisphere). In DSM IV-TR and V, its status was changed from a unique mood disorder to a recurrent major depressive disorder with a specifier of a seasonal pattern of symptoms that fully remits otherwise. In contrast to adults, children and adolescents with SAD do not appear to sleep more during the winter months, although low energy levels, fatigue, and decreased daytime activity levels have been described. It is postulated that there is an attenuation of the normal circadian rhythm amplitude in children with SAD. Bright light phototherapy in the morning and early evening has been reported to be beneficial.

**Anxiety Disorders**

In DSM-V, the broad category of anxiety disorders is now separated into three separate sequential sections: Anxiety Disorders, Obsessive-Compulsive and Related Disorders, and Trauma- and Stressor-Related Disorders. Furthermore, separation anxiety disorder and selective mutism are now included under Anxiety Disorders. Symptoms typically include physiologic hyperarousal (e.g., tachycardia, somatic complaints), cognitive hypervigilance, and behavioral symptoms, such as avoidance of anxiety-inducing situations and increased need...
for caregiver proximity. Given these characteristic features, it is not surprising that anxious children are particularly prone to developing sleep disturbances. Not only are anxiety disorders associated with a significantly increased risk of subsequently developing insomnia, persistent early sleep problems have been shown to be a reliable predictor of the later development of anxiety disorders; difficulties initiating and/or maintaining sleep are often the initial presenting complaint in anxious children.

The biologic basis for this relationship between anxiety and sleep is postulated to involve an overlap between the systems regulating sleep and affect and arousal. In particular, dysregulation of the hypothalamic-pituitary axis and alterations in cortisol secretion patterns (e.g., increased cortisol levels prior to sleep onset and decreased levels in the early morning) have been implicated in increasing the risk for both anxiety disorders and sleep disturbances in children. However, while laboratory-based PSG studies have demonstrated significantly prolonged sleep onset latency, reduced latency to REM sleep, increased awakenings, and less SWS in children with anxiety disorders compared to healthy controls, a home-based PSG monitoring study failed to provide evidence of disrupted sleep patterns in children with generalized anxiety disorder (GAD).

- **Nighttime fears**: Nighttime fears, which are a reflection of normal developmental processes, are extremely common, especially in young children, and may lead to significant bedtime resistance and problematic nightwakings (see Chapter 9). It is important to distinguish between developmentally appropriate nighttime anxiety-related behaviors and sleep-disrupting fears that are accompanied by daytime anxiety symptoms. The former is usually circumscribed and self-limited, and seldom requires intervention, whereas the latter may be indicative of a more global anxiety disorder. If an anxiety disorder is present, the cognitive-behavioral techniques often used successfully to treat isolated nighttime fears may be ineffective, and more intensive intervention strategies (e.g., psychotherapy, anxiolytics) may be warranted.

- **Adjustment disorders**: Sleep problems are commonly associated with adjustment issues, which involve temporary but maladaptive emotional and behavioral responses to both major and minor stressful life events, such as brief separation from a parent, birth of a sibling, or school transitions. These sleep problems, which commonly include bedtime resistance and nightwakings, may immediately follow the stressful event or may have a more gradual onset. Delayed sleep onset is often characterized by clinginess and refusal to be separated from the caregiver in younger children, and rumination and “free-floating” anxiety in the older child. Although adjustment reactions are self-limited by definition, parents may inadvertently prolong sleep problems by providing ongoing reinforcement (attention) for bedtime resistance, even after the original anxiety symptoms have been alleviated, and by failing to provide children with developmentally appropriate coping strategies.

- **Separation anxiety and GAD**: Children with separation anxiety and GAD frequently experience an exacerbation of symptoms at night. The degree, duration, and pervasiveness of the anxiety symptoms as well as a family history of anxiety and mood disorders may help to differentiate the child with more transient and situational nighttime anxiety from one with more chronic symptoms. Experiencing anxiety during the day typically indicates a more global anxiety issue rather than a more specific sleep problem.

- **Posttraumatic stress disorder (PTSD)**: Reports of sleep complaints, especially bedtime resistance, refusal to sleep alone, increased nighttime fears, and recurrent nightmares, are common in children with PTSD who have experienced severely traumatic events (including physical and sexual abuse). Children may also experience nocturnal panic attacks characterized by extreme hyperarousal and increased autonomic activity, which may be misinterpreted as behaviorally based nightwakings, sleep terrors, or even nocturnal seizures.
Sleep difficulties commonly occur in children and adolescents with acute and/or chronic anxiety. Similar to mood disorders, this relationship is a bidirectional one in which the presence of disturbed sleep and/or insufficient sleep, as well as daytime sleepiness, are associated with anxiety symptoms. In older children and adolescents in particular, it is important to differentiate between more transient, situational, and circumscribed nighttime anxiety and sleep symptoms associated with more global daytime anxiety. The approach to treatment and the need for additional counseling and psychopharmacology in these two situations may be quite different.

Epidemiology
Sleep disturbances, especially insomnia (usually defined as difficulty initiating and/or maintaining sleep), are reported in up to 90% of children and adolescents with depression and anxiety disorders. For example, it has been reported that up to 75% of studies of children with major depressive disorder meet the diagnostic criteria for insomnia, and 30% for severe insomnia; although as noted above, objective data (e.g., PSG) do not always support these subjective complaints. It is reported that up to 85% of children with an anxiety disorder also have significant sleep complaints.

Evaluation
It is often difficult from a clinical standpoint to clearly distinguish between primary and comorbid sleep disorders in children with depression and anxiety. However, it is incumbent on healthcare providers to evaluate and appropriately treat both, because improvement in one is likely to have a positive impact on the other. As discussed above, screening for sleep disorders should be part of the psychiatric evaluation for every child. Furthermore, periodic screening for sleep disorders should be part of the ongoing management of every child with diagnosed psychiatric disorders, especially depression and anxiety.

Treatment
Integrated Treatment Approaches
Because of the frequent coexistence and bidirectional effects of sleep disturbances and mood and anxiety disorders, the most effective treatment approach generally involves addressing both concerns simultaneously. At the very least, care should be taken to avoid treatment strategies that may exacerbate comorbid sleep problems (e.g., antidepressants that have disruptive effects on sleep).

Although a substantial number of treatment studies have demonstrated the efficacy of cognitive-behavioral (e.g., controlled exposure, relaxation-mental imagery), psychosocial, and pharmacologic (e.g., SSRIs) interventions for relieving mood and anxiety symptoms in children, there is a marked paucity of studies evaluating treatment interventions of sleep problems in pediatric depression and anxiety disorders. However, clinical experience indicates that treatment should include the following:

- **Development of strategies to optimize regular and sufficient sleep** that include basic principles of healthy sleep practices, such as a consistent bedtime and bedtime routine (see Chapter 5).
- **Specific behavioral strategies to reduce nighttime anxiety** may be helpful (see also Chapter 9). Note that bedtime strategies may be negotiated initially with the child, but once established, should be consistently and firmly enforced to avoid inadvertent reinforcement of stalling behaviors. However, in
cases of severe anxiety, the primary goal initially is to avoid exacerbating the anxiety symptoms at bedtime, and any of these approaches may need to be modified. An excellent self-help workbook resource for school-aged children with sleep related anxiety is “What to Do When You Dread Your Bed.”

- **Bedtime fading:** Setting a temporary later bedtime that coincides more closely with the actual sleep-onset time may relieve some of the anxiety associated with trying to fall asleep.

- **Reducing reliance on parental presence:** A variety of developmentally appropriate graduated extinction procedures targeted at reducing the child's reliance on parental presence at bedtime are available (see Chapter 7). Two-way “baby monitors,” which allow the child to have verbal contact with a parent without having to leave the bed or bedroom, may be a useful interim step for younger children.

- **Positive reinforcement:** The use of reward systems (e.g., sticker or star charts) can increase appropriate bedtime behaviors.

- **Bedtime pass:** The “bedtime pass,” a technique in which the child is given a single nightly opportunity (e.g., pass) to get up out of bed after “lights out,” is particularly well suited to anxious children, who may feel reassured simply by having this option available.

- **Institution of environmental changes** such as the use of night-lights, installation of a fish tank, or allowing a pet to sleep in the child's bedroom may help relieve anxiety symptoms that occur at bedtime and throughout the night.

- **Teaching of relaxation techniques** such as deep breathing, progressive muscle relaxation, and guided visual imagery may help children to develop a sense of control over anxiety symptoms.

- **Identification and elimination of additional factors** (e.g., alcohol or nicotine use) that may be impacting both the psychiatric disorder and the sleep disturbance.

- **Treatment of the primary psychiatric disorder** with psychotherapy and pharmacologic agents that do not exacerbate sleep disturbances. Because of the sleep-disrupting effects of some antidepressant medications (including some SSRIs, venlafaxine, and bupropion), treatment of the underlying depression may actually exacerbate the sleep disturbance.

- **Pharmacologic treatment of insomnia** may be appropriate when combined with behavioral interventions (e.g., stimulus control, cognitive-behavioral therapy) and sleep hygiene measures. In general, although sedatives and hypnotics may provide a more rapid therapeutic response, behavioral interventions are more effective long-term solutions. Use of single-agent medications that combine mood and anxiety effects with sedating properties may be a reasonable choice that limits exposure to multiple drugs and thus minimizes side effects.

- **Referral for mental health counseling** in cases of severe anxiety or if mood symptoms are persistent and affect daytime functioning is recommended.