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TECHNICAL REPORT

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Excessive Sleepiness in Adolescents and Young Adults: Causes, Consequences, and Treatment Strategies

ABSTRACT. Adolescents and young adults are often excessively sleepy. This excessive sleepiness can have a profound negative effect on school performance, cognitive function, and mood and has been associated with other serious consequences such as increased incidence of automobile crashes. In this article we review available scientific knowledge about normal sleep changes in adolescents (13–22 years of age), the factors associated with chronic insufficient sleep, the effect of insufficient sleep on a variety of systems and functions, and the primary sleep disorders or organic dysfunctions that, if untreated, can cause excessive daytime sleepiness in this population. *Pediatrics* **2005;115:1774–1786;** ***sleep, sleepiness, adolescents, pediatric sleep problems, daytime sleepiness, young adults, circadian rhythm, melatonin, motor vehicle accidents, depression, attention-deficit/hyperactivity disorder, school start times, school performance, obstructive sleep apnea, narcolepsy, idiopathic hypersomnia, delayed phase syndrome, insufficient sleep.***

ABBREVIATIONS. REM, rapid eye movement; MSLT, Multiple Sleep Latency Test; GPA, grade point average; ADHD, attention-deficit/hyperactivity disorder; PSG, polysomnography; SDB, sleep-disordered breathing; RLS, restless-legs syndrome; DSPS, delayed sleep-phase syndrome.

INTRODUCTION

During adolescence (13–22 years of age), many changes occur in sleep patterns, and there are many influences on sleep quality and quantity. Excessive daytime sleepiness in this population is a widespread problem and can have major negative effects on the individuals' performance, health, and safety. Pediatricians and other health care professionals have an important opportunity to evaluate their adolescent patients for evidence of excessive daytime sleepiness and underlying sleep deprivation and/or sleep disorders.

Development of Normal Sleep and Waking

A variety of sleep-pattern changes occur from childhood through adolescence. Laboratory evaluations, as well as field studies and surveys, have shown that across the second decade of life, there are numerous alterations in sleep physiology associated with consistent developmental patterns of sleep. Notable findings include decreased sleep duration with increasing age, a delay in bedtime and rise time (except on school mornings), and an increasingly large discrepancy between school-night and weekend sleep patterns. Children at 9 to 10 years of age who sleep approximately 10 hours on school nights usually will not sleep more than that (and sometimes will sleep less) on weekends. In contrast, adolescents typically will extend sleep on weekends, and this tendency increases as they age and as their school-night sleep decreases, causing them to accumulate a significant sleep debt. Sleep-research data indicate that adolescents still require 9 to 10 hours of sleep per night.[1,2]

A number of groups have examined sleep across adolescent development in laboratory-based studies. In these studies, laboratory constraints may affect outcomes, and this needs to be kept in mind when interpreting findings. For instance, although some studies have used participants' usual schedules to set bedtimes and rise times,[3–7] other studies have used a fixed period of time (10:00 PM to 8:00 AM) for sleep.[2,8,9] In addition, a number of studies have used a longitudinal approach,[2,6,8,9] whereas others have used a cross-sectional approach.[7,10,11] In laboratory studies in which the sleep schedule was varied, total sleep time has consistently been decreased in older adolescents, as expected, because of changed (school-night) sleep habits. Despite the varied methodologies used, consistent changes in sleep/wake architecture have been reported in adolescents. These changes include a decrease in slow-wave sleep time (decreased by nearly 40% from prepubertal to late pubertal adolescents with a 10-hour sleep opportunity[8]), an increase in the amount of stage 2 sleep, and a decrease in the latency to the first episode of rapid eye movement (REM) sleep.

Developmental changes in the amount of REM sleep obtained in a number of these studies typically parallel the findings for total sleep time. With increasing age, the time in bed for sleep and, hence, the total sleep time is decreased, with a concurrent de-

The guidance in this report does not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

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crease in the amount of time spent in REM sleep. Conversely, the duration of REM sleep is maintained at a constant level in adolescent subjects when time in bed is fixed.[8,9] Differences in the time of child versus adolescent spontaneous morning awakenings have also been reported (ie, differences in Tanner stages 1 and 2 vs 3–5). In a longitudinal study with a fixed time in bed for sleep, younger children were more likely than adolescents to spontaneously awake before 8:00 AM.[2] Although a small number of studies have reported gender differences in sleep patterns of older children and adolescents,[12] these findings are not consistent.[13]

Sleep/Wake Regulatory Processes

The circadian and sleep homeostatic systems act to coordinate most physiologic and behavioral systems of the body and brain. These 2 systems, working together or in opposition, influence the activities of the endocrine, thermoregulatory, neurobehavioral, renal, cardiovascular, digestive, and sleep/wake systems. With regard to sleep/wake, the circadian system may be viewed as wake promoting, and the homeostatic sleep system reflects sleep need or sleep debt and provides a drive for sleep.

In humans, the central circadian pacemaker, or biological clock, is located in the suprachiasmatic nuclei of the anterior hypothalamus.[14,15] These tiny paired nuclei are responsible for the generation of the daily (circadian) rhythms of physiologic, neurobiologic, and behavioral systems.[16] The circadian pacemaker is synchronized to the 24-hour day through external time cues from the environment (zeitgebers). The strongest of these zeitgebers is exposure to the light/dark cycle. Phototransduction from the retina to the suprachiasmatic nuclei occurs primarily via the retinohypothalamic tract. Entrainment of the circadian system to Earth's 24-hour day provides temporal balance between endogenous activities and the external environment such that sleeping/waking behavior, hormonal activity, temperature fluctuations, and neurobehavioral functioning occur in proper synchrony with the environmental day.

The circadian system plays an integral role not only in regulating the timing of sleeping and waking periods but also in influencing waking neurobehavioral functioning capabilities, alertness or fatigue levels,[17] sleep/wake duration, and sleep structure (REM sleep).[18–20] Opposing the wake drive provided by the circadian system, the sleep homeostatic system affects sleep propensity, sleep duration, and sleep structure.[21]

During waking periods, and especially during periods of extended wakefulness, the homeostatic drive for sleep gradually increases, with a consequent increase in the likelihood that sleep onset will occur. The circadian variation in wake drive occurs concurrently, producing peaks and troughs in sleepiness and alertness levels across the 24-hour day. The magnitude of sleepiness or alertness experienced at a given time is a product of the opposing influences of these 2 systems.[22] As a result, across a normal waking day, clock-dependent (circadian) alertness is usually lowest in the early morning and increases into the late afternoon or evening, thus opposing the growing sleepiness from having been awake all day. Consequently, alertness may be greater in the evening even if wakefulness has been sustained or a sleep debt has accumulated.

In 1993, a theory emerged attributing certain changes in adolescent sleep to an alteration in the circadian timing system. Carskadon and colleagues[23] demonstrated that circadian phase preference was delayed in association with more mature self-reported pubertal ratings in sixth-grade girls. In a laboratory-based study,[23] it was reported that the offset time of melatonin secretion in the morning was significantly correlated with Tanner stage. These findings indicate that a change in the biological system regulating circadian timing may accompany adolescent development. Such a change may promote the later timing of sleep that occurs during adolescence.

Surveys addressing sleep habits of older children and adolescents have been performed in many countries and across many decades. These surveys demonstrate remarkable consistency with regard to developmental changes in sleep habits, although specifics of bedtimes, rise times, and sleep length tend to vary among ethnic groups. Several review articles have summarized these findings[24,25] and concluded that, irrespective of location, developmental trends are similar. Older children and young adolescents tend to sleep about the same length of time on school days as non–school days. With increasing adolescent age, however, bedtime gets later on school and non–school days, with the magnitude of the delay greater on non–school days. Additionally, rising time on non–school days also gets later as adolescence progresses. We should note that the tendency for a phase delay of sleeping pattern is not a novel finding; Terman and Hocking[26] in 1913, for example, noted a shift from "vesperal" to "matinal" sleeping during adolescence, attributing the change to increasing homework.

As a consequence of these changes, the total sleep time obtained by older adolescents is shorter than sleep time for children and younger adolescents, and the discrepancies between school-day and weekend sleep and wake times increase with age. Maternal questionnaires from the Zurich longitudinal studies[27] have provided impressive data regarding developmental changes in sleep patterns of adolescents. Although the findings are not as extreme as those reported for US teenagers, similar trends are apparent. In contrast, differences have been found in total sleep obtained by adolescents in the United States and those in Zurich, Switzerland. A recent sample of eighth-grade students in the United States demonstrates an average school-night bedtime of 10:44 PM, wake time of 6:35 AM, and total sleep period of 7.9 hours.[28] The Zurich data, on the other hand, show an average bedtime of 10:02 PM, wake time of 6:30 AM, and total sleep period of 8.5 hours. Thus, although overall trends are similar for data from developed nations, significant differences in total sleep time may point to important concerns for young people who obtain minimal sleep.

Daytime Sleepiness

The Multiple Sleep Latency Test (MSLT) is an objective test that measures speed of falling asleep to determine the tendency to fall asleep during the day. A faster sleep onset indicates a greater level of sleepiness, as does an increased number of sleep onsets during multiple tests. During an MSLT, subjects are asked to fall asleep while lying in bed in a dark and quiet room during 4 or 5 20-minute periods spaced at 2-hour intervals.[29] If sleep occurs during this time period, it is allowed for only 1.5 minutes (experimental test) or 15 minutes (clinical test); if no sleep occurs, lights are turned on after the 20-minute test and the subject has to get out of bed and stay awake until the next testing period. In a longitudinal study using the MSLT, Carskadon and colleagues[2] assessed developmental changes in daytime sleepiness and demonstrated a change in the pattern of daytime alertness occurring at midpuberty. The prepubertal and early pubertal adolescents did not fall asleep on most of the tests (average latency across all naps of approximately 19.5 minutes of a maximum of 20 minutes), whereas midpubertal and late pubertal adolescents were more likely to fall asleep during the midafternoon tests, and the average sleep latency across all the naps decreased to approximately 15 minutes. This increase in sleep propensity occurred even though the more mature adolescents were sleeping as much as the less mature adolescents. This finding indicates that either adolescents may need more sleep than children or the pattern of sleepiness is reorganized during adolescent development.[24]

External Determinants of Sleep Patterns

Parental Influence

With the transition from childhood to adolescence, parents seem to change the manner in which they exert influence on children's sleep patterns, particularly on school days.[30] In Carskadon's study,[30] children 10 and 11 years of age were significantly more likely than children 12 and 13 years of age to report that parents set their school-night bedtimes. In later studies of high school students in 9th through 12th grades, only 5% of these older adolescents had a school-night bedtime set by their parents, and more than 75% went to bed when homework, television viewing, or socializing was done for the day or whenever they felt sleepy.[31]

Thirteen-year-old children reported more frequently than younger children that they required either alarm clocks or their parents to wake them up on school mornings. This difficulty in waking in the morning continues into the older years. More than 85% of high school students in 1 study used an alarm or their parents to awaken them in the morning on school days.[32] Thus, the influence of parents shifts from setting bedtimes during childhood to assisting with rising time during adolescence.

School Start Times

Historically, schools in the United States have started early in the morning. In addition, many US school districts use a 3- or 4-tiered schedule in which high schools open first, followed by middle or junior high schools, and then elementary schools.[33] In a preliminary survey of 40 schedules posted on the Internet from high schools throughout the United States for the 1996–1997 academic year, 48% started at 7:30 AM or earlier, whereas only 12% started between 8:15 AM and 8:55 AM.[34] Most recently, in the 2001–2002 academic year, 35% of 50 high schools surveyed started earlier than 7:30 AM, nearly 50% started between 7:31 AM and 8:14 AM, and only 16% started between 8:15 AM and 8:55 AM.

Early high school start time is a significant, externally imposed constraint on teenagers' sleep/wake schedules; for most adolescents, waking up to go to school is neither spontaneous nor negotiable. Szymczak and colleagues[35] followed Polish students between 10 and 14 years of age for more than 1 year and found that all of them slept longer on weekends and during vacations by extending their sleep and waking up later. These investigators concluded that the school schedule was the predominant determinant of wake times for these students. Similarly, several surveys of high school students have found that students who start school at 7:30 AM or earlier obtain less total sleep on school nights because of earlier rise times.[31,36–38]

In a laboratory and field study, Carskadon and colleagues[39] evaluated the effect of a 65-minute advance in school start time on approximately 40 9th-graders in their transition to 10th grade. Specifically, junior high school started at 8:25 AM and high school started at 7:20 AM in a large urban school district. Sixty-two percent of the students in 9th grade and less than half the students in 10th grade got an average of as much as 7 hours of sleep on school nights. Students awoke earlier on school days in 10th grade than in 9th grade and had shorter sleep latencies on the MSLT in 10th than in 9th grade, particularly on the 8:30 AM assessment. In addition, 16% of participants experienced 2 REM episodes on the MSLT in 10th grade (48% of subjects experienced 1 REM episode). The occurrence of REM sleep episodes on the MSLT was associated with a delayed timing of melatonin secretory pattern in these adolescents. In a study of nearly 600 young adolescents (10–12 years of age), Epstein and colleagues[40] compared a 7:10 AM with an 8:00 AM school start time. In their survey, children with early start times reported significantly shorter mean sleep times (ie, approximately 25 minutes less) than did children who started school after 8:00 AM.

Employment and Extracurricular Activities

Another major influence on sleep patterns of high school students in the United States is the number of hours they spend working for pay. Students who work 20 or more hours per week report going to bed later at night, sleeping fewer hours per night, oversleeping more in the morning, and falling asleep more in class than those who do not work or who work fewer than 20 hours per week. In a survey of more than 3000 high school students (grades 9–12) from several Rhode Island school districts, nearly 60% of the students reported that they held part-time

jobs, and almost 30% indicated that they worked 20 hours or more per week.[31,34] The 11th- and 12th-grade students who worked more than 20 hours per week reported significantly different sleep/wake behaviors from those of their peers who worked less than 20 hours per week or not at all. The high-work group reported more symptoms of daytime sleepiness such as struggling to stay awake while driving, in classes, and while reading, studying, or doing homework. This group also reported greater use of caffeine, alcohol/drugs, and tobacco.

Clinical Consequences of Inadequate Sleep

Excessive Daytime Sleepiness

Alertness is defined as the inherent ability of the brain to sustain attentive wakefulness with little or no external stimulation. When someone is excessively sleepy, alertness and vigilance become unstable and unreliable. Cognitive capabilities slow down, and over time there is an increased risk of making errors[41] and an increased risk of accidents (for example, automobile crashes). When excessively sleepy, individuals may begin tasks well, but as time on tasks continues, performance will decrease. Sleepy individuals may increasingly neglect activities judged to be nonessential. High levels of sleepiness impair complex performance, leading to lapses in attention, slowing of motor and cognitive reactions, mental mistakes, working-memory errors, time-on-task decrements, and potentially uncontrolled sleep attacks.[42]

Despite the laboratory findings on the effects of sleep loss on neurocognitive functioning, it is commonly assumed that sleep loss has little or no effect on waking brain function, that the effects of sleep loss are primarily motivational, and that the amount of sleep required to maintain stable waking performance is less than that obtained.[43] In reality, the opposite is true. There have been several studies assessing the effect of sleep deprivation on tendency to fall asleep.[30,44,45] In these studies, successive days of restricting sleep duration led to a significant tendency to doze off in quiet settings. This might manifest as falling asleep in class, or there may be uncontrollable "microsleeps" leading to poor task performance. More recent studies have supported these earlier findings. For example, Fallone and colleagues[25] studied young people between 8 and 15 years of age and restricted them to 1 night of only 4 hours of sleep. Their daytime sleepiness was increased both subjectively and objectively. The effect of sleepiness on neurobehavioral functioning in these studies is less clear and may depend on whether the sleep loss is across a single night or multiple nights. There also seems to be an emotional overlay. Maayan and colleagues[46] studied 10 adolescents the day after a night of total sleep deprivation. They found that the adolescents exposed to emotion-producing pictures during the test demonstrated decreased performance on a working-memory task.

School Performance Problems

In a recent critical review, academic performance and sleep were analyzed extensively by Wolfson and Carskadon.[47] Studies clearly suggest that shortened total sleep and irregular sleep schedules are highly associated with poor school performance for adolescents. After a year-long study of 17 school districts in Minnesota, Minneapolis Public Schools changed their high school start time from 7:15 AM to 8:40 AM, beginning with the 1997–1998 school year. The Center for Applied Research and Educational Improvement at the University of Minnesota has examined the effect of the later start time.[48] The study examined student grades and attendance through district records and administered the School Sleep Habits Survey to 50 962 students in 7 high schools (grades 9–12). Analyses found that daily attendance rates were higher in the 1999–2000 academic year than they were in 1995–1996; the percentage of high school students who were continuously enrolled in the district or in the same school increased in 1999–2000, relative to the percentage in 1995–1996; and with the later start time, the dropout rate decreased. The School Sleep Habits Survey showed no change in average school-night bedtimes or weekend bedtimes and rise times. However, because of later rise times on weekdays, Minneapolis students reported that they obtained, on average, 60 more minutes of sleep on school nights than did their peers in high schools with start times 1 hour earlier. In addition, Epstein and colleagues[40] compared young adolescents who started school at 7:15 AM or earlier at least 2 times a week with those who started at 8:00 AM. Early risers complained more of daytime fatigue and sleepiness throughout the school day, greater tendency to doze off in class, and attention/concentration difficulties in school.

Wolfson and Carskadon[31] administered the School Sleep Habits Survey to 3120 high school students from 4 high schools representing 3 school districts in southern New England. Adolescents with self-reported higher grades reported significantly longer and more regular sleep/wake schedules. Specifically, they said that they got more total sleep and had earlier bedtimes on school nights than did students with lower grades. In fact, these differences distinguished students reporting mostly Bs or better from those reporting Cs and worse. Students' weekend sleep habits also differed according to self-reported grades. Specifically, A and B students reported earlier bedtimes and earlier rise times than did students with poorer grades. Students with worse grades reported greater weekend delays of sleep schedule than did those with better grades.

Meijer and colleagues[49] focused more on the relationship between sleep/wake patterns and young adolescents' perceptions of their school functioning than on academic grades or examination scores. They assessed young adolescents in 7th and 8th grades. Adolescents who reported having difficulty getting up were less motivated to do their best at school, whereas children with higher-quality sleep and reports of feeling more rested were more receptive to teacher influence, had a more positive image of themselves as students, and reported higher motivation to do their best in school.

A few studies have examined the relationship be-

tween sleep/wake patterns and academic performance in college students. Trockel and colleagues[50] interviewed or surveyed 185 randomly selected first-year college students regarding sleep/wake habits, exercise, eating, mood, perceived stress, social support, religious habits, and semester grade point averages (GPAs). Sleep habits, particularly rise times, accounted for the largest amount of variance in GPAs. In particular, later weekday and weekend wake times and increased number of work hours (paid/volunteer) were associated with lower GPAs. Eating habits, mood, stress, time management, and social support were not associated with these first-year college students' grades. Kelly and colleagues[51] also studied college students and found that short-sleepers reported significantly lower overall GPAs than did long-sleepers. Although there were no age or gender differences, long-sleepers (≥9 hours per night) reported significantly higher GPAs than did short-sleepers (≤6 hours per night; mean GPA: 3.24 vs 2.74, respectively). Average-sleepers (7–8 hours per night) were not significantly different from long- or short-sleepers.

Sleep and Attention-Deficit/Hyperactivity Disorder in Adolescents and Young Adults

As noted above, lack of sleep may cause problems with attention and concentration in the laboratory setting. The well-recognized clinical syndrome is attention-deficit/hyperactivity disorder (ADHD), which is estimated to affect 5% to 10% of the school-aged population. This disorder persists into adolescence and young adulthood in 10% to 60% of affected individuals.[52] The relationship between sleep problems and ADHD in children has been examined in multiple studies, using a number of approaches,[53–66] but similar data in adolescents and adults are largely unavailable. Reviews of clinical complications of ADHD in adolescents include some anecdotal references to sleep disturbances,[67–69] but studies of childhood ADHD and sleep are rare and have included small numbers of subjects between 12 and 18 years of age.[64]

Studies of children with ADHD have used either parental- or self-report surveys or all-night sleep testing (polysomnography [PSG]) to examine the relationship between sleep architecture/disturbances and ADHD. Methodologic limitations of these studies include small sample sizes and selection bias, variability in diagnostic criteria for ADHD, failure to document pubertal status, and variability in control groups. In addition, although parental assessment of their children's sleep behavior and disturbances is clearly more subjective, relatively objective methods such as PSG may not reflect "real-world" conditions accurately. Finally, many studies have failed to consider the effects of medication and the presence of comorbid psychiatric conditions. Despite these limitations, most of the "objective" studies have failed to find consistent differences in sleep architecture and patterns between children with ADHD and controls,[70–79] yet most parental-report studies have reported increased sleep problems in children with ADHD, including difficulty falling asleep, night wakings, and restless sleep. However, more recent studies have suggested that many of these sleep disturbances are attributable to either medication-related effects from psychostimulants or common psychiatric comorbid conditions rather than to ADHD per se.[80]

The etiology of sleep disturbances associated with ADHD in childhood is likely to be multifactorial and vary across patients. In addition to medication-related effects on sleep and the influence on sleep behavior of such common comorbid conditions as oppositional defiant disorder, depression, and anxiety disorders, primary sleep disorders may present itself with ADHD-like symptoms or may exacerbate underlying ADHD.[81–83] For example, there is mounting evidence not only that sleep-disordered breathing (SDB) is strongly associated with inattention, hyperactivity, impaired "executive" cognitive functioning, and disruptive behaviors in children but also that treatment of SDB results in significant improvement or even complete amelioration of ADHD-related symptoms.[84,85] Likewise, other primary sleep disorders such as restless-legs syndrome (RLS), periodic limb-movement disorder,[86–89] delayed sleep-phase syndrome (DSPS),[90] and narcolepsy[91] may present with ADHD symptomatology.

Primary abnormalities in central nervous system regulation of arousal, behavioral inhibition and self-regulation, and/or vigilance associated with ADHD also have been postulated to result in sleep disturbances. These disturbances suggest a more "intrinsic" sleep/wake dysregulation in at least some individuals with ADHD.[92,93] There is considerable empirical evidence to suggest that brain systems regulating sleep and attention/arousal are linked and that abnormalities in similar neurotransmitters such as the noradrenergic and dopaminergic systems may be found in ADHD and sleep disturbances.[94] Although intriguing, at the current time these hypotheses are still largely speculative. Nevertheless, it is clear from clinical observations and on theoretic grounds that an association between ADHD and sleep disturbances exists, that symptoms of ADHD and sleep disorders frequently overlap, and furthermore that the presence of a comorbid sleep disorder may significantly increase the level of impairment in an individual with ADHD.

Mood Disturbances

The relationship between sleepiness and depressed mood in adolescents must be considered in both directions. That is, there is extensive evidence that adolescents with clinical mood disorders (particularly major depressive disorder) report high rates of sleep disturbances and complaints.[95,96] There are also data indicating that adolescents with sleep problems report increased negative mood and/or difficulties with mood regulation.[97–99] Part of the relationship may be accounted for by the effects of stress and emotional arousal interfering with sleep in adolescents with emotional problems,[100] whereas there is also evidence that sleep disruption can cause irritability and negative mood in adolescents.[92]

Studies of sleep and major depressive disorder in

adolescents provide evidence for subjective sleep complaints (especially difficulty falling asleep) and are extremely common, affecting most adolescents interviewed during an episode of depression.[96] Objective electroencephalogram studies of clinical samples have revealed evidence of sleep disturbances in some depressed adolescents but at lower rates than sleep disturbances seen in adult depression.[101–105] However, subjects who showed objective evidence of sleep-onset abnormalities in adolescence were more likely to develop depressive episodes in the future.[106,107] More recently, studies focusing on the microarchitecture of sleep in depressed adolescents provide some evidence that more subtle disturbances in sleep also may be predictive of a worse clinical course among adolescents with mood problems.[108]

Given the evidence of negative effects in both directions, one of the major concerns regarding the co-occurrence of sleep and mood problems in adolescents is that it can contribute to a "negative spiral" in school and social functioning. For example, late-night and erratic schedules and early school start times can lead to sleep deprivation, which in turn can erode mood and motivation. Difficulties with mood, motivation, and school performance create greater stress and affective problems. The negative affective experiences further interfere with sleep and arousal regulation and circadian effects and lead to difficulty falling asleep, more erratic schedules, and additional deterioration across these systems. Research is needed to examine the effects of early intervention aimed at sleep while examining mood symptoms to address these important concerns.

Drowsy Driving

Motor vehicle crashes are a leading cause of death in adolescents and young adults, and sleepiness can be deadly for adolescents behind the wheel. Studies analyzing motor vehicle crash data by age group have found that young people between 16 and 29 years of age were the most likely to be involved in crashes caused by the driver falling asleep.[109–112] Pack and colleagues[109] reviewed the 5104 crash reports from North Carolina from 1990 to 1992 in which the driver was judged to have fallen asleep. In 771 of the crashes, the driver was also thought to be intoxicated. Of the remaining 4333 crashes, the majority occurred with younger individuals. Fifty-five percent occurred with a driver who was 25 years or younger. Unlike crashes with adults older than 65 years, which typically occur during midafternoon, crashes with this younger age range generally take place at night and involve young males driving alone and going off the road.

Lack of sleep has been implicated as a cause of falling asleep at the wheel.[113] Compared with sleeping 8 or more hours each night, sleeping 6 to 7 hours was associated with a 1.8 times higher risk for involvement in a sleep-related crash versus a non–sleep-related crash, and sleeping fewer than 5 hours per night invoked a 4.5 times higher risk. Forty percent of drivers had been awake for 15 or more hours before falling asleep at the wheel and crashing, and nearly 20% had been awake for 20 or more hours.

Additional Causes of Daytime Sleepiness

As described in the previous sections, there is an inherent tendency for adolescents and young adults to get insufficient sleep, and as a result, excessive daytime sleepiness may be frequently seen in this age group. Nevertheless, it is also essential to acknowledge the importance of untreated sleep disorders and other organic causes of excessive daytime sleepiness. It is very difficult to differentiate insufficient sleep from other causes of daytime sleepiness without taking a formal sleep history. The following sections will review clinical sleep disorders and propose a working algorithm to help clinicians sort through the differential diagnoses.

Insomnia and Circadian-Rhythm Disorders

Insomnia is a broad term used to describe a wide range of complaints relating to disorders of sleep. For many people, insomnia is a subjective complaint of dissatisfaction with sleep, including decreased sleep quality, decreased sleep quantity, trouble getting to sleep, and trouble maintaining sleep. In some cases, insomnia is a symptom of another underlying medical or psychological disorder, and in other cases there is no apparent physiologic cause (this type of insomnia generally is called "psychophysiological insomnia"). The waking effects of insomnia include daytime fatigue or sleepiness, neurocognitive deficits, and altered mood.

The major insomnia complaint in adolescents is difficulty initiating sleep. In this age group, a major cause of this insomnia is DSPS. DSPS is a circadian-based disorder in which an individual's internal circadian pacemaker is not in synchrony with external or environmental time. Affected adolescents typically experience difficulty in initiating and terminating sleep at a "normal" time[114] and prefer later sleep times (between 2:00 AM and 6:00 AM) and wake times (between 10:00 AM and 1:00 PM). The sleep structure of DSPS patients is otherwise normal. DSPS is characterized by a delay in the timing of activities demonstrating circadian rhythmicity, such as melatonin secretion,[115] changes in core body temperature, and the sleep/wake cycle.

The incidence of DSPS in the general population is unclear. Some reports suggest that only 0.7% of middle-aged adults have DSPS and 7% or more of adolescents suffer from this disorder.[116–118] In adolescents, there may be an overestimation of the incidence of DSPS because of developmental and/or environmental influences on the circadian system that produce a DSPS-like profile in this age group. As mentioned previously, Carskadon and colleagues[30,39] reported a phase delay in the timing of daytime sleepiness in midpubertal children by using the MSLT as an index of sleepiness. Adolescents typically do not go to sleep until late because of school, work, social, and family commitments but still must awaken early for school. When allowed to sleep without time constraints on weekends, adolescents choose to go to bed and rise later than on weekdays

and sleep for longer periods (in essence, "catching up" on sleep).[119]

The diagnosis of DSPS is based on clinical history. The classic patient with DSPS is more frequently tardy or absent from school because of an inability to get out of bed in the morning despite parental intervention. If the adolescent with DSPS does make it to class, he or she may fall asleep, frequently has poor grades, and frequently is labeled as having a behavioral problem. Although not a practical treatment option, these symptoms would abate if the adolescent were allowed to sleep late in the morning and arrive later at school.

Treatment regimens for DSPS need to be designed to resynchronize the circadian system. There is a high incidence of relapse, however, after discontinuation of treatment, especially if patients are unable to maintain a rigid sleep/wake schedule. Czeisler and colleagues[120] delayed the sleep times of adult patients by 3 hours each day over a period of 5 to 6 days until the desired sleep time was reached. An alternative approach is to have the patient get up and stare into a bright light box for 30 minutes first thing in the morning to "reset the clock." However, studies using light exposure to re–entrain subjects have reported mixed findings.[117,121]

Another proposed treatment for DSPS is the administration of melatonin. Melatonin is an endogenous hormone that plays a role in the control of the circadian system[122] and potentially in the initiation of sleep.[123,124] Exogenously administered melatonin has been reported to have both chronobiotic (phase-shifting)[125] and soporific (sleep-inducing) properties.[126] As a treatment for DSPS, melatonin exerts its effects primarily by shifting the timing of the circadian system and has been demonstrated to be successful in re-entraining sleep/wake rhythms in people with jet lag and people who perform shift work.[127]

The efficacy of melatonin treatment for DSPS has been examined in a number of studies. Phase advance of the sleep/wake cycle was reported after daily administration of melatonin for up to 6 weeks.[76,128–130] After termination of the treatment, however, the melatonin-induced phase advances were reversed and subjects returned to being phase delayed. Although melatonin administration may be an effective treatment for DSPS, it should be used with caution. As yet, there is no established dose of melatonin to be administered for effective results, and the timing of administration is also important to achieve phase shifts in the desired direction.[131] In addition, the safety of melatonin administration, particularly in adolescents and young adults, is not established. Melatonin levels are high during puberty,[132] and melatonin has been implicated in reproductive development and seasonal breeding in several species. Additionally, studies on the safety of long-term melatonin administration have not been performed. Therefore, at present, melatonin should be thought of as a research tool and not a clinical solution.

Sleep-Disordered Breathing

SDB, or obstructive sleep apnea, is a condition in which the pharynx intermittently and repetitively collapses during sleep. Arterial oxygen levels decrease and carbon dioxide levels increase until a subconscious arousal from sleep occurs. The pharyngeal dilator muscles then contract, the pharynx opens, air rushes in under pressure creating a loud snoring sound, ventilation resumes, and blood gas abnormalities are corrected. SDB in adolescents and young adults leads to the same daytime sequelae related to excessive daytime sleepiness as seen with insufficient sleep (sleep restriction).

The most common cause of sleep apnea in this age range is enlarged tonsils and adenoids. The epidemic of childhood weight problems, however, has resulted in obesity becoming a major cause of SDB in children as well as adults. Other contributing factors include retrognathia, nasal obstruction,[133] evening alcohol ingestion, family history of sleep apnea,[134] black race,[134] and history of wheezing and cough.[134]

The profile of an adolescent or young adult with sleep apnea is of someone who snores loudly and frequently, has been observed to have episodes of apnea during sleep, has awakened choking, and is excessively sleepy. Clues on physical examination that would help the physician suspect SDB include enlarged tonsils, retrognathia, mouth breathing, and upper-body obesity.

The true prevalence of SDB in this age range is unknown because of a lack of studies and lack of consensus about minimum criteria for diagnosis. Part of the problem is that some studies have included younger children as well as adolescents.[135] Although an obstructive-apnea index (the number of episodes of obstructive apnea per hour of sleep) greater than or equal to 1 per hour may be diagnostic of SDB in younger children,[136] the threshold may be higher in adolescents and young adults. Acebo and colleagues[137] reported that an obstructive apnea index of 1 per hour was normal in 13-year-old boys and girls and in 22-year-olds.[137] In fact, in this study of normal subjects, an apnea index up to 3.6 in young boys and 4.5 in young men was considered to be normal.

Hui and colleagues[138] evaluated 1910 freshman at the Chinese University of Hong Kong and found that 26% snored, 11% reported impaired performance ability, and 42% reported daytime sleepiness. A limited number of these college students underwent all-night sleep studies, leading to a calculation of 0.1% prevalence of SDB in this population. In contrast, Sanchez-Armengol and colleagues[139] studied 101 adolescents 12 to 16 years of age. Of this population, 29% snored, 14% were excessively sleepy, and 3% had SDB.

The diagnosis of sleep apnea requires overnight assessment by PSG, typically in a sleep laboratory. Once sleep apnea is diagnosed, treatment options include removal of the tonsils and adenoids if they are enlarged, weight reduction if overweight or obese, and/or nightly continuous positive airway

pressure therapy to prevent intermittent upper-airway collapse.[140]

Narcolepsy and Idiopathic Hypersomnia

Narcolepsy is a neurologic disorder associated with inappropriate control of REM sleep. Recent studies have suggested that the pathophysiology of narcolepsy involves depletion of the neuropeptide hypocretin known as orexin.[141] The true prevalence of narcolepsy is unknown, but 4 to 10 people per 10 000 in the United States may have the disorder.

Core symptoms of narcolepsy reflect the components of REM sleep, specifically loss of muscle tone and an extremely active cortex during sleep, leading to vivid dreams. Patients with narcolepsy may have sleep attacks in which they inappropriately doze off. Many will have cataplexy, which is a sudden loss of muscle tone typically precipitated by anger or laughter. In severe cases, the patient may lose all tone and collapse to the floor. Symptoms may even present as unexplained syncope. If left alone, the patient will fall asleep and be in REM sleep. The presence of cataplexy is diagnostic of narcolepsy.[142] People with narcolepsy may also experience sleep-onset paralysis as they are starting to fall asleep; they suddenly become paralyzed while they are still awake. Sleep paralysis may also occur on awakening, but this may happen in people without narcolepsy as well, either sporadically or on a familial basis. Patients with narcolepsy may also experience hypnagogic hallucinations (vivid, dreamlike visual images) before falling asleep. Only a minority of patients present with the complete tetrad of symptoms.[91,143]

Although the symptoms of narcolepsy typically begin during adolescence and young adulthood, adults with narcolepsy frequently report that the diagnosis was not established for several more years.[144] Frequently, adolescents with narcolepsy have behavioral and emotional disturbances. Dahl and colleagues[91] found that 12 of 16 adolescents with narcolepsy had emotional problems, and 4 patients were misdiagnosed as having a psychiatric disorder. Delayed and mistaken diagnoses may contribute to adult psychosocial dysfunction.[143,145]

A definitive diagnosis of narcolepsy can be made if cataplexy is present. Testing for narcolepsy includes overnight PSG to exclude other causes of daytime sleepiness, such as SDB. In addition, patients will undergo MSLT testing the following day. There is no specific test for cataplexy. Adolescents with narcolepsy will be pathologically sleepy with a mean sleep latency across all naps of less than 6 minutes, compared with a normal mean sleep latency of 15 minutes. Patients with narcolepsy, as well as patients with insufficient sleep, may demonstrate REM sleep in at least 2 of the naps, whereas normal subjects have no REM sleep.

Treatment includes stimulant drugs such as methylphenidate and dextroamphetamine to decrease daytime sleepiness, wake-promoting agents such as modafinil, and REM-suppressant agents such as tricyclic antidepressants and serotonin reuptake inhibitors to control cataplexy. Recently, sodium oxybate has become available to prevent cataplexy.[146] In addition, education, counseling, and working closely with both family and school personnel are essential. Regular sleep/wake schedules need to be established, and daytime restorative naps may be helpful.

"Idiopathic hypersomnia" is a term used to describe patients who are excessively sleepy for no apparent cause and who do not have cataplexy.[147] Despite adequate sleep time for age and despite a normal all-night PSG result, these patients have MSLT results in the sleepy range, yet do not demonstrate any episodes of REM sleep in individual naps or any of the associated symptoms of narcolepsy. It is possible that these patients may be found to have narcolepsy in the future. Patients with idiopathic hypersomnia are frequently treated with stimulant medications such as methylphenidate and dextroamphetamine, although the response to treatment is usually less effective than it is with narcolepsy.

Periodic Limb Movement During Sleep and RLS

Periodic limb movements during sleep are repetitive contractions of the anterior tibialis muscles occurring during sleep. Although more common in aging adults, leg movements may also be seen in adolescents and young adults. Periodic limb movements may be an incidental finding on all-night PSG, but they can also be a cause of subconscious sleep disruption, leading to daytime sleepiness, or a trigger for full awakenings and subsequent insomnia.

A related disorder is RLS, in which patients complain of an uncontrollable feeling in their lower legs at rest, either lying or sitting. The sufferer may become so uncomfortable that he or she has to move around or get up and walk to control the symptoms. Symptoms will typically disappear when the patient starts moving around. Restless-legs complaints are more common with increasing age but may be seen in younger patients as well, as with certain medical conditions such as renal failure and diabetes mellitus or under special circumstances such as pregnancy. Although most patients with restless-legs complaints will have periodic limb movements during sleep, the inverse correlation is very uncommon. RLS has been associated with insomnia and recently has been found to be associated with ADHD in children and adolescents.[87–89]

Treatment typically is directed at increasing central nervous system dopamine concentrations with agents such as carbidopa-levodopa, pergolide, or pramipexole. Gabapentin and benzodiazepines are also used to treat RLS.

Effect of Medications/Substances

Many common medications may have a marked effect on sleep and sleep patterns. A detailed summary is beyond the scope of this review. Examples include the use of extremely long-acting stimulants for ADHD. These agents may paradoxically increase sleepiness and augment problems with attention, concentration, and mood during the daytime. By causing overstimulation, these agents may actually have a negative effect on sleep and decrease actual sleep time. Similarly, medications used for depression may have a profound effect on sleep quality.

TABLE 1. "BEARS": A Sample Sleep History

B =	Bedtime problems (Do you have any problems falling asleep at bedtime?)
E =	Excessive daytime sleepiness (Do you feel sleepy a lot during the day? In school? While driving?)
A =	Awakenings during the night (Do you wake up a lot at night?)
R =	Regularity and duration of sleep (What time do you usually go to bed on school nights? Weekends? How much sleep do you usually get?)
S =	Sleep-disordered breathing (Parent: Does your teenager snore loudly or nightly? Patient: Has anyone ever told you that you snore loudly at night?)

Even over-the-counter cold and allergy medications may be overstimulating (eg, pseudoephedrine) or oversedating (eg, diphenhydramine). Adolescents or young adults abusing prescription or illegal drugs are at high risk of significant adverse sleep effects.

Alcohol is a potent short-term sedating substance.

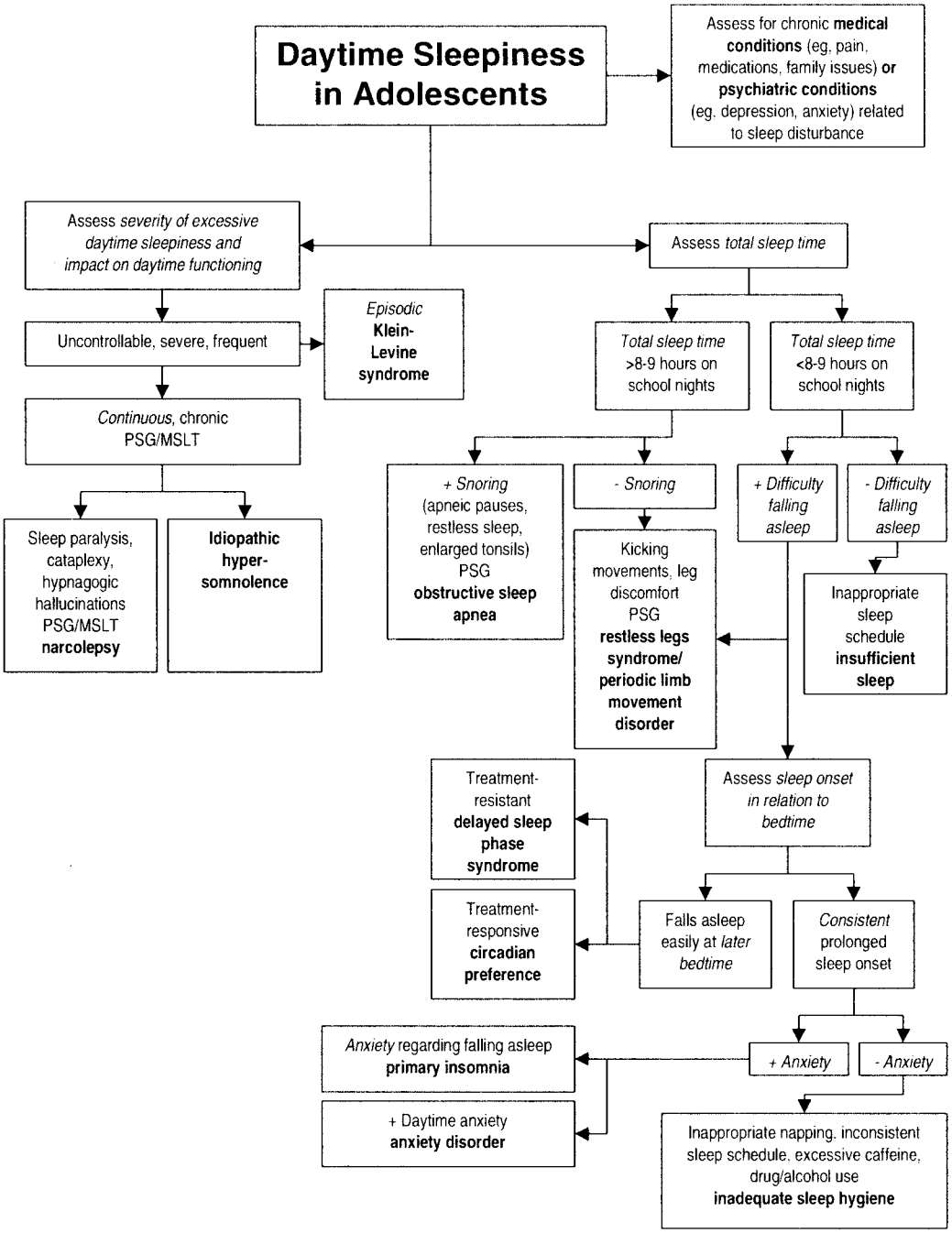

Fig 1. Sample clinical assessment flowchart. PSG indicates polysomnography; MSLT, Multiple Sleep Latency Test. (Reproduced with permission from Mindell JA, Owens JA. *A Clinical Guide to Pediatric Sleep: Diagnosis and Management of Sleep Problems*. Philadelphia, PA: Lippincott Williams & Wilkins; 2003:56.)

Although it may induce sleep, once the blood alcohol concentrations have dropped low enough, one may develop acute rebound insomnia. In addition, alcohol causes pharyngeal dilator muscle relaxation and hence precipitates snoring and even sleep apnea in susceptible individuals.[148,149]

Caffeine is ubiquitous in coffee, tea, chocolate, and soft drinks and may lead to insomnia or subconscious sleep disruption and subsequent daytime drowsiness, which in turn may lead to an increased need for caffeine the next day. Excessive use of caffeine, nicotine, or stimulants under conditions of sleepiness may provide apparent short-term gain but long-term negative consequences on sleep and circadian health.

IMPLICATIONS FOR CLINICAL PRACTICE

Excessive sleepiness is a significant problem in adolescents and young adults. In most cases, it results from insufficient sleep caused by insufficient time in bed and is associated with intrinsic changes in the sleep/wake cycle as well as extrinsic pressures to go to bed later and get up earlier. At a minimum, clinicians evaluating individuals in this age range need to ask questions routinely about sleep patterns and how much sleep an individual is receiving as well as whether there are any sleep-related symptoms. Specific tools such as the "BEARS" Pediatric Sleep History (Table 1) have been used in younger children and adolescents[150] and can serve as a template for the development of a routine review of systems for clinicians in asking sleep questions. This instrument is designed to provide a practical and user-friendly vehicle for incorporating pediatric sleep history into the standard histories and physical examinations in both outpatient and inpatient settings. The "BEARS" instrument is divided into 5 major sleep domains and provides a comprehensive screen for the major sleep disorders affecting children in the 2- to 18-year age range. Each sleep domain has a set of age-appropriate "trigger questions" for use in the clinical interview.

The key message for clinicians is that insufficient sleep (time in bed) occurs commonly but that this is not the only process that may be present. Consideration, therefore, needs to be given to possible depression, obstructive sleep apnea, insomnia, narcolepsy, and other sleep disorders as well as to medications or stimulants such as caffeine as the cause of impaired sleep quality and excessive daytime sleepiness.

There is not an established and validated algorithm that all clinicians use for diagnosing and treating sleep disorders in this age range. One sample assessment tool is shown in Fig 1. Treatment should be directed at any potentially reversible process. At a minimum, adolescents and young adults need to be counseled about normal age-appropriate sleep needs and the detrimental effects of sleep loss on performance and overall health.

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