


Evaluation and Treatment of Children and Adolescents With Excessive Daytime Sleepiness

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Introduction

Excessive daytime sleepiness (EDS), a common presenting symptom among children and adolescents, is caused by a wide range of sleep disorders and other conditions, and it may impair health, development, and daily function.¹⁻³ Studies have reported rates of EDS due to various etiologies ranging from 10% to 20% in prepubertal children⁴⁻⁸ and 16% to 47% in adolescents.^{7,9,10} Sleep problems that can cause EDS are present in an estimated 25% to 40% of children and adolescents,¹¹ encompassing behavioral, neurologic, and respiratory disorders. Other conditions affecting sleep (eg, chronic pain, nocturnal seizures, and prescription and/or illicit drugs) are important contributors in some children, as reviewed elsewhere.^{2,12-17}

Untreated pediatric sleep problems and associated EDS may lead to behavioral problems, mood disturbances, depression, dysregulation of affect/emotion, impairments in neurocognitive function, increased risk for alcohol and drug use in teenagers, declining academic performance, and safety concerns.¹⁸⁻²⁰ Long-term chronic sleep loss, which results in EDS, adversely affects physiologic systems such as carbohydrate metabolism and endocrine function, potentially increasing risk for type 2 diabetes and cardiovascular dysfunction, with associated hypertension/risk of atherosclerosis and pro-inflammatory response.¹⁸ Epidemiologic data also suggest that short sleep duration is associated with an increased risk of obesity (a significant health problem itself) in children and adolescents.²¹ Other risks associated with chronic sleep loss in children and adolescents include increased unintentional injuries,²² sports-related injuries,²³ and automobile crashes.^{22,24} Specific sleep disorders frequently accompanied by EDS have also been associated with health-related adverse effects; for example, obstructive sleep apnea (OSA) in children and adolescents has been correlated in multiple studies with growth failure and insulin resistance, as well as hypertension and inflammatory changes in systemic and central nervous system vasculature.^{25,26} Children and adolescents with EDS are also more prone to being

bullied, to being regarded as “lazy,” “inattentive,” or “unmotivated,” and to having low self-esteem.^{2,12}

Excessive daytime sleepiness in children and adolescents is underreported by parents and underdiagnosed by physicians, possibly due to lack of recognition.^{3,27} Externalizing symptoms such as hyperactivity and oppositional behavior in children may be attributable to EDS, and children may not recognize or be able to verbalize the internal state of “sleepiness.”³ Thus, children and adolescents with EDS often present to primary care pediatricians when parents become concerned about behavioral, mood, and academic issues that they may not attribute to underlying sleep problems. Given the high prevalence and potential health implications, prompt detection, diagnosis, and management of EDS is an essential component of primary pediatric care.

Presentation and Screening

Excessive daytime sleepiness has classically been defined as a subjective sense of sleepiness, or increased tendency to fall asleep, occurring at times and in situations when the individual would be expected to be awake and alert.^{28,29} However, a presenting complaint that may be related to EDS must first be assessed in the context of common developmental manifestations of daytime sleepiness, as well as age-related/developmentally appropriate nocturnal sleep and daytime napping.³ Recommended ranges of optimal sleep amounts according to standards from the American Academy of Sleep Medicine vary with age and include daytime sleep periods (naps) in young children (Table 1).³⁰

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Table 1. American Academy of Sleep Medicine Recommendations for Amounts of Sleep to Promote Optimal Health by Pediatric Age Group.³⁰

Age Group	Optimal Sleep Recommendations per 24 Hours
Infants (4-12 months)	12-16 hours (including naps)
Children (1-2 years)	11-14 hours (including naps)
Children (3-5 years)	10-13 hours (including naps)
Children (6-12 years)	9-12 hours
Teenagers (13-18 years)	8-10 hours

Furthermore, daytime sleepiness may be considered excessive when it involves a pattern of increased nocturnal sleep and/or more daytime napping compared with the normal range for children of the same age group (ie, not just a self-limited response to acute sleep curtailment/disruption by illness, change in routine, etc). Difficulty waking in the morning and frequently falling asleep during the day in inappropriate circumstances (short car rides, watching television, playing) may also be considered excessive sleepiness. The propensity to sleep longer than usual when given the opportunity (ie, on weekends, during school vacation) is also an important but underrecognized sign of chronic insufficient sleep and EDS.³¹

Manifestations of EDS in children and adolescents can be nuanced, deceptive, and apparently paradoxical.^{3,32} In prepubertal children, manifestations of EDS may include restlessness, hyperactivity, emotional lability, irritability, aggression, and behavior problems in school, which can be similar to, and possibly attributed to, attention deficit hyperactivity disorder.^{3,33} Prepubertal children with EDS may also present as quiet, listless, inattentive and unfocused, or withdrawn and isolated because they have missed social events due to their sleepiness.^{3,33} Considering that alertness in school-aged children is normally high, practitioners should have a very low threshold for investigating complaints of overt sleepiness in this age group.

Adolescents as a group have high levels of sleepiness for various reasons, including developmental puberty-related changes in circadian rhythms, chronic sleep curtailment, and use of electronic media before bed and during the night with resultant sleep disruption. Adolescents with EDS may appear to be lethargic or moody, disinterested, lacking in motivation, bored, and depressed.^{29,31,32,34} They may fall asleep in class or while completing homework and perform poorly in academics or sports. Given that EDS is nearly ubiquitous in adolescents, the distinction between “normal” sleepiness related to environmental and lifestyle factors and “pathologic” sleepiness that might be a manifestation of an underlying primary central nervous system

hypersomnia is important but not always easy to determine. Nonetheless, the consequences of environmentally induced sleepiness (often called “behaviorally induced insufficient sleep syndrome”) can be highly significant³⁵; manifestations of behaviorally induced insufficient sleep syndrome in adolescents include car crashes and other unintentional injuries, depression, and risk-taking behavior.³¹

Another challenge in screening for EDS is the need to distinguish it from fatigue. Fatigue, while related and often a co-complaint with EDS, does not by strict definition involve increased sleep propensity, decreased alertness during waking hours, and/or a short time to fall asleep at night (sleep latency). Rather, fatigue is a subjective sense of lack of energy and an abnormal level of exhaustion following normal activities.^{29,32} Fatigue is more likely to be associated with psychiatric and mood-related conditions, such as depression and anxiety, and chronic medical conditions, such as chronic infection, hypothyroidism, chronic fatigue syndrome, and personality disorders.^{28,29} However, the distinction between “sleepiness” and “fatigue” in clinical situations may be unclear, and use of both subjective and objective methods to quantify increased sleep propensity may assist in identifying these overlapping conditions.

Screening instruments can help confirm the presence of EDS by probing for sleepiness in specific situations relevant to children and adolescents (Table 2).^{13,36}

Widely used and well-validated screening instruments for pediatric sleep problems include the BEARS (B = Bedtime Issues, E = Excessive Daytime Sleepiness, A = Night Awakenings, R = Regularity and Duration of Sleep, S = Snoring) 5-item questionnaire,³⁷ Children’s Sleep Habits Questionnaire,^{36,38} Children’s Report of Sleep Patterns–Sleepiness Scale,³⁹ and Pediatric Sleep Questionnaire.⁴⁰ Instruments used specifically to screen for EDS in the pediatric population include the Pediatric Daytime Sleepiness Scale⁴¹ and Modified Epworth Sleepiness Scale for Children and Adolescents.^{42,43} Another screening tool that is useful in identifying potential causes of EDS is a 24-hour, 2-week, parent-recorded (or self-recorded for adolescents) sleep diary.¹³ Sleep diaries allow detailed documentation of variations in sleep patterns and sleep-wake cycles in real time compared with descriptive histories recalled in the clinic.¹³

Diagnosing Causes of EDS

Once the presence of EDS has been confirmed, its diagnosis requires a systematic approach, given the many conditions that may result in daytime sleepiness in children and adolescents.^{2,3,12} Sleep-related causes of EDS may be conceptualized under 4 broad categories:

Table 2. Instruments to Screen for and Measure Subjective EDS and Sleep Problems in Children and Adolescents.

Instrument	Description	Validation/Correlation Data
BEARS Sleep Screening Tool ³⁷	<ul style="list-style-type: none"> 5 questions (BEARS): bedtime issues, excessive daytime sleepiness, night awakenings, regularity and duration of sleep, and snoring Questions adapted/targeted to 3 age ranges: toddler/preschool (2-5 years), school aged (6-12 years), and adolescent (13-18 years) Questions aimed at both children/adolescents and parents/caregivers 	<ul style="list-style-type: none"> Clinical use of BEARS was correlated with increased detection/diagnosis of sleep problems in children 2-12 years of age (N = 195)³⁷
CSHQ ³⁶	<ul style="list-style-type: none"> Parent-report survey for school-aged children 4-10 years of age Includes 45 items relating to major sleep quality domains/complaints for age group in 8 subscales: bedtime resistance, sleep-onset delay, sleep duration, sleep anxiety, night wakings, parasomnias, sleep disordered breathing, and daytime sleepiness Each item rated from 0 (usually) to 3 (rarely); some items are reversed to make higher score reflect more disturbed sleep 	<ul style="list-style-type: none"> Showed adequate/acceptable internal consistency in both community sample (n = 469) and clinic sample (n = 154) of school-aged children³⁶ Subscales showed no correlation with PSG or actigraphy, except for one (night wakings) with actigraphy only in children 6-12 years of age (N = 30)³⁸
CRSP-S ³⁹	<ul style="list-style-type: none"> Self-report measure for school-aged children 8-12 years of age 5-item survey scored from 1 (never) to 5 (always) for situations where children should not feel sleepy (eating, talking with someone else, at school, playing, riding in the car or a bus for <20 minutes) 	<ul style="list-style-type: none"> Showed internal consistency and test-retest reliability in children 8-12 years of age³⁹ Correlated with other measures, including actigraphy, parent report, sleep hygiene level, and others³⁹
PSQ ⁴⁰	<ul style="list-style-type: none"> 22-item scale focused on assessment for SRBDs Includes 4 subscales: snoring/breathing problems (8 items), daytime sleepiness (4 items), inattention/hyperactivity (6 items), and other symptoms (4 items: nocturnal enuresis, morning headache, delayed growth, and obesity) 	<ul style="list-style-type: none"> Validated in children 2-18 years of age for good consistency (n = 162) and good test-retest reliability (n = 21)⁴⁰
PDSS ⁴¹	<ul style="list-style-type: none"> 8 questions scored 0 (never) to 4 (always) regarding sleepiness in the morning, at school, doing homework, and during the day Additional questions available to probe for other signs/impacts of EDS (academic problems) and parental report/observations 	<ul style="list-style-type: none"> Showed internal consistency and correlation with negative academic outcomes in children 11-15 years of age (N = 450)⁴¹
ESS-CHAD ⁴²	<ul style="list-style-type: none"> Based on the well-known/validated ESS often used in adults; includes 8 questions scored 0 (would never fall asleep) to 3 (high chance of falling asleep) in situations adapted for children (eg, sitting in a classroom) 	<ul style="list-style-type: none"> Rasch analysis showed reliability and internal validity in children 12-18 years of age (N = 297)⁴³
<i>Parent/self-assessments to assist in screening</i>		
Sleep diaries ¹³	<ul style="list-style-type: none"> 24-hour, 2-week sleep diary Completed by parent or adolescent Typical sleep parameters recorded include bedtime; sleep-onset latency; number, duration, and timing of awakenings during the night; morning wake and rise times; total sleep duration; time in bed; sleep efficiency (time asleep/time in bed); number, duration, and timing of daytime sleep periods; and differences in sleep patterns on school and nonschool days Graphic sleep diaries are available for download at websites hosted by the American Academy of Sleep Medicine (yoursleep.aasmnet.org/pdf/sleepdiary.pdf) and the National Sleep Foundation (sleepfoundation.org/sites/default/files/SleepDiaryv6.pdf), among other online sources 	

Abbreviations: BEARS, B = Bedtime Issues, E = Excessive Daytime Sleepiness, A = Night Awakenings, R = Regularity and Duration of Sleep, S = Snoring; CSHQ, Children's Sleep Habits Questionnaire; CRSP-S, Children's Report of Sleep Patterns–Sleepiness Scale; EDS, excessive daytime sleepiness; ESS-CHAD, Epworth Sleepiness Scale–Child Adolescent; PDSS, Pediatric Daytime Sleepiness Scale; PSG, polysomnography; PSQ, Pediatric Sleep Questionnaire; SRBDs, sleep-related breathing disorders.

Table 3. Conditions That May Cause EDS in Children or Adolescents and Management Options.^{3,13,51,53-60}

Disorder	Prevalence ^a	Therapies (Potential Uses/Indications)
<i>Insufficient sleep/sleep deprivation</i>		
Insomnia	20% to 30%	<ul style="list-style-type: none"> Behavioral management, sleep hygiene advice, family counseling (insomnia) Alpha agonists: clonidine 0.05 mg at bedtime, guanfacine 1 mg at bedtime (insomnia; off-label) Antihistamines: diphenhydramine 6.25-50 mg maximum (insomnia; off-label)
Behavioral sleep-onset association disorder		
Limit-setting disorder		
Psychiatric medical disorder		
Poor sleep hygiene		
Sleep initiation and maintenance insomnia		
RLS with associated delayed sleep onset		
<i>Fragmented/disturbed sleep</i>		
Behavioral		<ul style="list-style-type: none"> Behavioral management, sleep hygiene advice, family counseling
Sleep-onset association disorder		
SRBDs		<ul style="list-style-type: none"> Adenotonsillectomy (first-line treatment for OSA, with weight reduction in obese children) CPAP during sleep (OSA, with weight reduction in obese children) Weight reduction (SRBDs in obese children)
Snoring	16%	
Obstructive sleep apnea	1% to 5%	
Upper airway respiratory syndrome		
Hypoventilation		
Central sleep apnea		
Movement disorder		<ul style="list-style-type: none"> Iron supplementation (for patients with ferritin levels <50 ng/mL) Gabapentin 5-15 mg/kg at ≤ 1.5 hours before bedtime (symptomatic relief of RLS and PLMD; off-label) Low-dose clonazepam 0.5-4 mg/day (symptomatic relief of RLS and PLMD; off-label) Clonidine 0.1-0.3 mg/day (symptomatic relief of RLS and PLMD; off-label) Standard treatment of symptoms by condition
RLS	6% to 26%	
PLMD	2% to 4%	
Bruxism (teeth grinding)		
Head banging, body rocking		
<i>Medical problems disturbing sleep</i>		
Asthma		
Eczema		
Cystic fibrosis		
Gastroesophageal reflux		
Epilepsy		
Environmental disturbances		<ul style="list-style-type: none"> Family counseling
Noise, light co-sleeping, crowding		
<i>Circadian misalignment</i>		
Circadian rhythm disorder	7% ^b	<ul style="list-style-type: none"> Light therapy, chronotherapy (circadian rhythm disorder) Melatonin 0.5-3 mg 2-3 hours before bedtime (circadian rhythm disorder, unregulated OTC medication)
Delayed sleep-wake phase syndrome		
Non-24-hour sleep-wake schedule		
Sleep-entrainment problems		
<i>Increased need for sleep</i>		
Neurologic injury/disorder		<ul style="list-style-type: none"> Improved sleep hygiene with regular sleep-wake schedules; strategic napping (hypersomnia) Sodium oxybate, pediatric nightly dosage, 2-9 g in divided doses, based on body weight and time of administration^c (indicated for treatment of cataplexy or EDS in patients ≥7 years of age with narcolepsy)
Head trauma		
Increased intracranial pressure		
Temporary hypersomnia		
Medical illness, drug use (illicit, prescribed)		
Depression		
Recurrent hypersomnia		<ul style="list-style-type: none"> Modafinil 100-400 mg/once daily or divided (hypersomnia; off-label for children)
Kleine-Levin syndrome	Rare	
Persistent hypersomnia		<ul style="list-style-type: none"> Methylphenidate extended-release 5-20 mg/day (hypersomnia; off-label) Dextroamphetamine 10-30 mg/day; for children 6-12 years of age: starting at 5 mg/day, titrated at 5 mg weekly until optimal dose attained; for children ≥ 12 years of age: starting at 10 mg/day, titrated at 10 mg weekly (indicated for treatment of narcolepsy) Clomipramine, 3 mg/kg/day (cataplexy; off-label) Venlafaxine extended-release 37.5-150 mg/day (cataplexy; off-label)
Narcolepsy, type 1 or type 2	0.03% to 0.05%	
Idiopathic hypersomnia		
Inherited disorders (eg, Prader-Willi syndrome)		
Hypothalamic lesions (eg, astrocytoma, craniopharyngioma, degenerative, infection, traumatic, vascular)		

Abbreviations: CPAP, continuous positive airway pressure; EDS, excessive daytime sleepiness; OSA, obstructive sleep apnea; OTC, over the counter; PLMD, periodic limb movement disorder; RLS, restless legs syndrome; SRBDs, sleep-related breathing disorders.

^aPrevalence rates given where data were available.^{3,12,13,34,53}

^bEstimated prevalence in adolescents.

^cSee Xyrem (sodium oxybate) prescribing information⁵¹ for pediatric dosing schedule by weight.

insufficient sleep duration, fragmented/disturbed sleep, circadian misalignment, and primary disorders that increase sleep needs (Table 3).

Table 4 summarizes characteristics and causes of some of the more common and/or representative conditions.

Table 4. Descriptions of and Factors Contributing to Conditions That May Cause EDS in Children or Adolescents.

Disorder	Description/Diagnostic Criteria	Contributing Factors
Insomnia	<ul style="list-style-type: none"> Generally defined as chronic difficulty with sleep onset, short sleep duration, and reduced or inadequate sleep consolidation and/or quality resulting in impaired daytime function¹³ 	<p>Children</p> <ul style="list-style-type: none"> May include underlying problems such as physical or psychiatric conditions, or inappropriate or irregular sleeping and/or napping schedules that interfere with the child's natural sleep patterns¹³ Common comorbidities associated with behavioral insomnia include depression, anxiety, and ADHD¹³ <p>Adolescents</p> <ul style="list-style-type: none"> Often related to either DSPS or poor sleep hygiene resulting from heavy use of electronic media (television, music players, mobile devices, and video games); lack of parental monitoring and rules regarding bedtimes; after-school employment; demands of schoolwork; increased socializing; and use of alcohol and illicit drugs³¹ Correlated with enlarged tonsils and adenoids and may be associated with increased body mass index (this association is not as clear as in adults)⁶¹ Other risk factors: chronic wheezing or sinus problems, nasal allergies, neuromuscular disorders, craniofacial abnormalities, and African American race¹³ Occur most commonly in prepubertal children 2-6 years of age, when adenoidal/tonsillar hypertrophy often presents⁴⁴
Sleep-onset association disorder	<ul style="list-style-type: none"> Refers to the inability to fall asleep without specific conditions (eg, being rocked, watching television, hearing a story), or the presence and/or intervention of parents/caregivers; often resolves around 3 or 4 years of age (toddler stage)^{13,32} 	
Limit-setting sleep disorder	<ul style="list-style-type: none"> Typically occurs in preschool- and school-aged children and refers to parental difficulty in setting and enforcing bedtime limits and rules, with the child refusing to go to bed or awakening repeatedly through the night^{13,32} 	
DSPS	<ul style="list-style-type: none"> Characterized by a marked delay in the circadian timing of the urge to sleep by about 2-3 hours, with corresponding later awakening³¹ Typically results in difficulties arising in time for school or work, as well as EDS^{34,54} 	
SRBDs	<ul style="list-style-type: none"> Spectrum from snoring (mildest form) to frequent loud snoring, snorting, gasping, and pauses in breathing (OSA; most severe manifestation)^{13,61} 	
OSA	<ul style="list-style-type: none"> Characterized by presence of nocturnal symptoms, such as snoring, labored/obstructed breathing during sleep, and/or a consequence of disturbed sleep such as EDS or hyperactivity⁶² ICSD-3 criteria: PSG findings of ≥ 1 obstructive events (obstructive or mixed apnea or obstructive hypopnea) per hour of sleep, or obstructive hypoventilation as indicated by PCO_2 in arterial blood >50 mm Hg for $>25\%$ of sleep time, along with snoring, paradoxical thoracoabdominal movement, or flattening of the nasal airway pressure waveform⁶² 	
Others	<ul style="list-style-type: none"> Treatment-emergent central sleep apnea: residual OSA symptoms on PSG after resolution of OSA symptoms with CPAP treatment^{32,62} Hypoventilation: elevation of arterial PCO_2^{32,62} 	
Movement disorders	<ul style="list-style-type: none"> Characterized by stereotyped, simple movements, such as brief arm or leg jerks occurring during sleep or at its onset^{32,62} 	
RLS	<ul style="list-style-type: none"> May occur in waking states, most often when at rest; uncomfortable sensations typically described as "spiders crawling" or tickling of the legs¹³ ICSD-3 diagnostic criteria in children: urge to move the legs, sometimes with an uncomfortable sensation, which occurs primarily with rest or inactivity, is present primarily in the evening or at night, is relieved at least partially or totally by movement, and causes distress, associated sleep disturbance, and/or impairment^{32,62} 	
PLMD	<ul style="list-style-type: none"> Sleep-movement disorder (does not occur while awake) Diagnosis of PLMD in children requires a rate of >5 limb movements per hour during sleep, accompanied by sleep disturbance or other functional impairment^{32,62} 	

(continued)

Table 4. (continued)

Disorder	Description/Diagnostic Criteria	Contributing Factors
Hypersomnias		
Narcolepsy	<ul style="list-style-type: none"> • EDS characterized by frequent and extreme drowsiness most often occurring during quiet or passive activities (eg, reading quietly, sitting in class, or sitting in a car); sleep attacks lasting from a few minutes to ≥ 90 minutes; sleep drunkenness or sleep inertia on forced awakening (presenting as extreme confusion, irritability, or even aggressive behaviors) • Other symptoms of narcolepsy include cataplexy^a (type 1 narcolepsy), sleep paralysis, and hypnagogic and hypnopompic hallucinations, which may represent intrusions of REM sleep into the waking state⁶⁴ • Nocturnal sleep disturbances after falling asleep quickly at bedtime (fragmented sleep and at times extended night awakenings) • Children can present with dramatic weight gain at the onset of symptoms⁶⁴ • Diagnostic criteria—type 1 narcolepsy ($\approx 70\%$ of patients): EDS > 3 months, and either (1) cataplexy plus MSLT with MSOL ≤ 8 minutes and ≥ 2 SOREMPs or (2) CSF hypocretin level $< 110 \mu\text{m/L}$⁴⁹ • Diagnostic criteria—type 2 narcolepsy ($\approx 30\%$ of patients): EDS > 3 months, MSLT with MSOL ≤ 8 minutes and ≥ 2 SOREMPs, no cataplexy, CSF hypocretin $> 110 \mu\text{g/mL}$, hypersomnolence not better explained by other condition⁴⁹ 	<ul style="list-style-type: none"> • Predisposing genetic factor—type 1 narcolepsy: HLA DQB1 06*02 is found in $> 90\%$ of patients (however, the presence of this HLA has low specificity as it is also present in $\approx 25\%$ of the general population without narcolepsy)⁵³ • Loss of hypocretin (a neurotransmitter involved in wakefulness) underlies type 1 narcolepsy; the relationship of type 2 narcolepsy with hypocretin loss is less clear⁶⁴ • Secondary hypersomnias can be due to neurologic disorders, psychiatric disorders, and medication or other substances⁵³
Idiopathic hypersomnia	<ul style="list-style-type: none"> • MSLT with MSOL ≤ 8 minutes but no more than 1 SOREMP⁴⁹ • Clinical symptoms include long nighttime sleep, severe, prolonged sleep inertia, and daytime naps that leave the individual unrefreshed 	
Secondary hypersomnias	<ul style="list-style-type: none"> • Kleine-Levin syndrome is characterized by recurrent episodes of EDS lasting from 2 days to 4 weeks, cognitive and behavioral disturbances, and hyperphagia and hypersexuality⁵³ 	

Abbreviations: ADHD, attention deficit hyperactivity disorder; CPAP, continuous positive airway pressure; CSF, cerebrospinal fluid; DSPS, delayed sleep-wake phase syndrome; EDS, excessive daytime sleepiness; HLA, human leukocyte antigen; ICSD-3, The International Classification of Sleep Disorders, 3rd edition; MSLT, Multiple Sleep Latency Test; MSOL, mean sleep-onset latency; OSA, obstructive sleep apnea; PCO_2 , partial pressure of carbon dioxide; PLMD, periodic limb movement disorder; PSG, polysomnography; REM, rapid eye movement; RLS, restless legs syndrome; SOREMP, sleep-onset rapid eye movement period; SRBDs, sleep-related breathing disorders.

^aCataplexy is defined as a sudden, brief, and transient partial or complete loss of muscle tone, often precipitated by strong positive emotions. Patients are fully conscious during episodes and aware of their surroundings. Cataplexy may manifest as weakness of the head and facial muscles, leading to head drop, jaw slackening, tongue protrusion, slurred speech, or head nodding, or weakness of the knees.^{32,64}

Figure 1 provides a diagnostic algorithm.

Sleep disorders vary somewhat in prevalence by age category.⁸ Specific forms of behavioral insomnia, such as settling problems and night waking due to inappropriate sleep-onset associations and/or inadequate caregiver limit setting, for example, are most common in children younger than 3 years; and delayed sleep-wake phase disorder and restless legs syndrome are more typically associated with adolescents.^{13,32} OSA and sleep-related breathing disorders, although prevalent in the pediatric population as a whole, are most commonly reported

between ages 2 and 6 years in association with development of adenoidal and tonsillar hypertrophy; however, additional risk factors occurring in older children and adolescents, such as obesity, have emerged as important contributors in recent decades.^{4,44}

History and Physical Examination

A comprehensive and detailed history and physical examination are essential for diagnosing causes of EDS in children and adolescents and may include input from

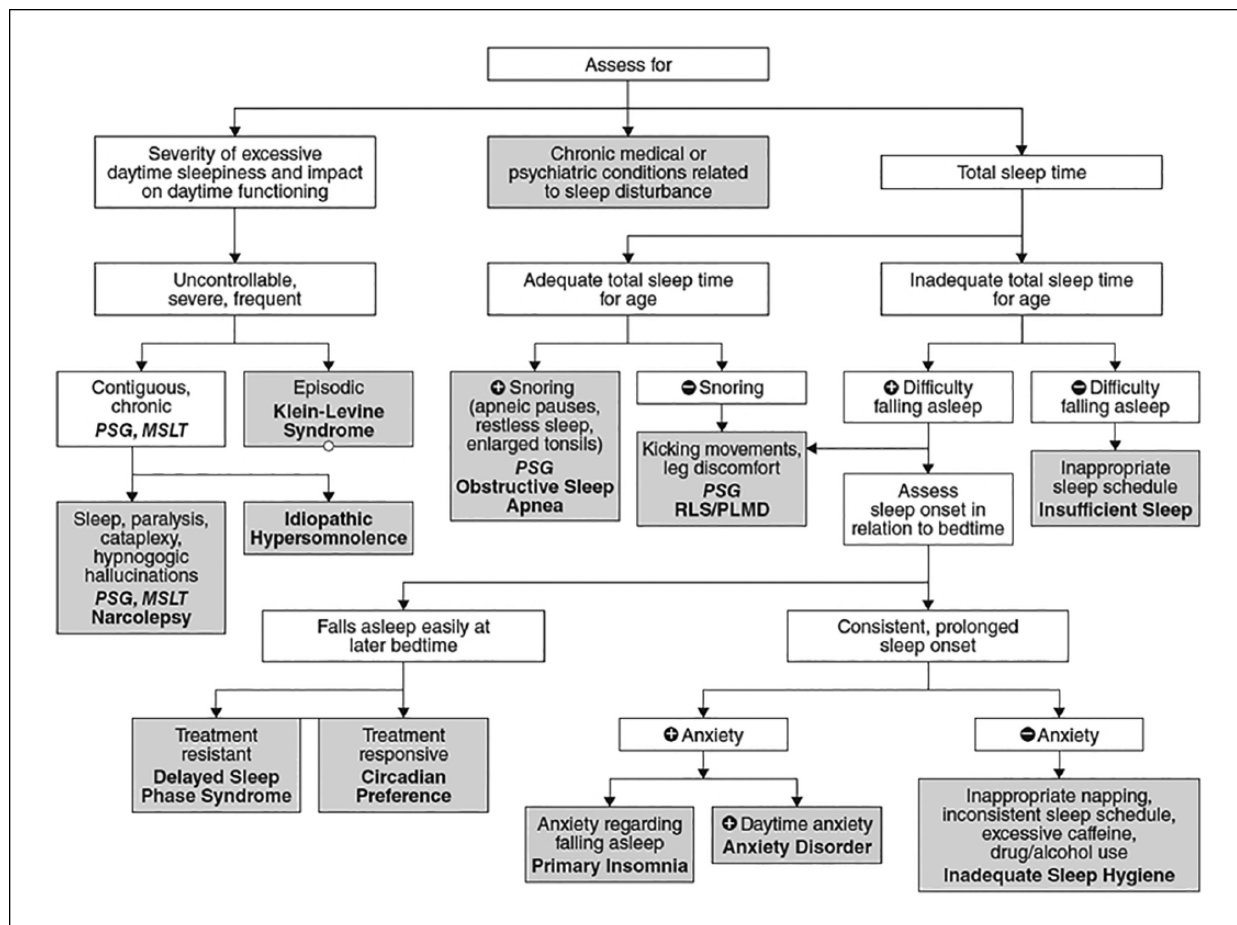


Figure 1. Evaluation and diagnosis of excessive daytime sleepiness in school-aged children and adolescents. Bold, italic text = diagnostic tests; bold, not italic text = diagnoses; shaded boxes = testing/diagnostic endpoints.

Abbreviations: MSLT, Multiple Sleep Latency Test; PLMD, periodic limb movement disorder; PSG, polysomnography; RLS, restless legs syndrome.

classroom teachers, as well as patients and parents/caregivers.^{2,13,29} Suggested points for the workup are summarized in Table 5.

If medical problems are identified, the child should be referred to the relevant specialist for further evaluation and treatment. After appropriate therapy, the presence of EDS should be reevaluated.

Objective Measures

Objective measurement of sleep may be considered if subjective screening instruments, history, and physical examination fail to produce adequate clinical information for diagnosis.² Such measures include actigraphy, polysomnography (PSG), and the Multiple Sleep Latency Test (MSLT); the Maintenance of Wakefulness Test may be used in the context of treatment (Table 6).

Actigraphy, which uses small, validated portable devices similar to wristwatches to record the presence or

absence of limb movement (indicating wakefulness or sleep), is useful primarily to evaluate insomnia and circadian rhythm disorders.^{45,46} Actigraphy is also used to confirm sufficient nighttime sleep before the PSG and MSLT (insufficient sleep can skew MSLT results). Overnight, in-laboratory PSG is particularly helpful for diagnosing OSA, periodic limb movement disorder, and narcolepsy in children and adults,^{45,47} but it is not useful or indicated for evaluation of behavioral sleep disorders, including insomnia.¹³ The MSLT assesses the propensity to fall asleep and is the standard test for quantifying EDS.^{45,48} In addition to the presence of EDS, a mean sleep-onset latency ≤ 8 minutes and ≥ 2 sleep-onset rapid eye movement periods as assessed by the MSLT are diagnostic for narcolepsy.⁴⁹ However, the applicability of the MSLT for children is unclear, because normative values have not been established for children younger than 8 years, and mean values appear to vary across pediatric age categories.⁴⁵ The Maintenance of

Table 5. Considerations in EDS Workup.

Type of Assessment	Examples
Sleep behaviors ^{2,12,13}	<ul style="list-style-type: none"> • Daily sleep duration and patterns (sleep-wake scheduling, napping) • Difficulty of morning awakenings • Behaviors, impressions, routines, and expectations of the family/child related to sleep
Medical history ^{2,12,13,65}	<ul style="list-style-type: none"> • Asthma • Eczema • Epilepsy • Migraine/headaches • Neuromuscular disorders • Autism • ADHD • BMI that is high (85th to 94th percentile) or indicates obesity (≥ 95th percentile)
Sociocultural factors/potential differences in family attitudes toward sleep ⁶⁶	<ul style="list-style-type: none"> • Bed and room sharing • Bedtimes and napping • Parental perception of sleep problems
Sleep hygiene (potentially detrimental factors) ^{2,12,64}	<ul style="list-style-type: none"> • Noise, light, snacking, or television watching before bed • Use of mobile devices • Variable sleep schedules • Engaging in mentally or physically stimulating activities too close to bedtime • Other causes of disruption or discomfort
Use of medications or substances affecting sleep ¹³	<ul style="list-style-type: none"> • Stimulants (including caffeine) • Prescription and over-the-counter hypnotic and sedating medications • Alcohol and drugs (prescription or recreational/illicit)
Family history of sleep disorders ¹³	<ul style="list-style-type: none"> • Narcolepsy with cataplexy • RLS • OSA
Witnessed reports or video of nocturnal disturbances ¹³	<ul style="list-style-type: none"> • Snoring/gasping or pauses in breathing • Awakenings caused by medical problems such as asthma, eczema, and epilepsy • Symptoms of RLS (sensations in the legs at bedtime relieved by movement) • Twitching, kicking of legs in sleep (possibly indicative of PLMD) • Potential symptoms of narcolepsy, including episodes of cataplexy, sleep paralysis, and hallucinations on going to sleep or awakening
Physical examination ¹²	<ul style="list-style-type: none"> • Assessments of growth and development (including Tanner stage and nutritional status) • Presence of dysmorphisms indicating any genetic conditions • Physical signs of endocrinologic derangement such as thyroid disease, hormonal imbalance (ie, polycystic ovary syndrome), and metabolic syndrome • Neurological function • Ear, nose, and throat examination
Laboratory assessments ^{49,61}	<ul style="list-style-type: none"> • Lateral neck X-rays (to further characterize adenoidal enlargement, which is associated with increased risk for pediatric OSA) • Tests for iron deficiency (associated with RLS and PLMD) • Chemistry and hormonal panels (if such conditions are suspected)

Abbreviations: ADHD, attention deficit hyperactivity disorder; BMI, body mass index; EDS, excessive daytime sleepiness; OSA, obstructive sleep apnea; PLMD, periodic limb movement disorder; RLS, restless legs syndrome.

Wakefulness Test is a test of wakefulness used primarily to measure response to therapy for narcolepsy and hypersomnia (ie, effects on EDS), and it may also help judge the risk of vehicle crashes or other injury in patients with EDS.^{2,48}

Treatment

Treatments for the various causes of EDS are shown in Table 3. Therapy for insomnia and other causes of

insufficient sleep often begins with behavioral and non-pharmacologic approaches, with pharmacotherapy used as an adjunct, based on the common nature of the problems (eg, behavioral and habitual). The first step in treating EDS is always the optimization and maintenance of good sleep hygiene. Specific treatment options for patients with OSA include adenotonsillectomy, weight reduction, and continuous positive airway pressure. For children with movement disorders resulting in insufficient and/or disrupted sleep, such as restless legs

Table 6. Objective Sleep Measurement Instruments.^{2,28,45-48}

Instrument	Purposes/Indications	Description
Actigraphy	<ul style="list-style-type: none"> Records sleep duration and patterns Evaluation for insomnia and circadian rhythm disorders and monitor response to treatment for these conditions Estimates total sleep time (if PSG is not available) 	<ul style="list-style-type: none"> Actigraphs (or actimeters) are small, computerized devices similar to wristwatches worn by the patient around the wrist or ankle Records limb movement (indicating wakefulness) and absence of movement (indicating sleep)^a Allows up to several weeks of recording
PSG	<ul style="list-style-type: none"> Gold standard for evaluation of EDS and sleep disorders Pediatric indications include evaluation for <ul style="list-style-type: none"> EDS Narcolepsy and other hypersomnias OSA, central apnea Monitor and titrate CPAP treatment Epilepsy Parasomnias PLMD Chronic pain or rheumatologic disorders disturbing sleep 	<ul style="list-style-type: none"> Usually an overnight, in-laboratory assessment of nocturnal sleep attended by a technician^b Measures total sleep time, sleep latency, arousals, and leg movements Records sleep stages/architecture via EEG to mark brain wave activity, EMG to record skeletal muscle movement, and EOG for eye movements (to identify REM sleep) EEG lead can also record seizures Chest and abdominal belts monitor respiration, including oronasal and mouth breathing for OSA evaluation Includes pulse oximetry and end-tidal CO₂ to monitor oxygen, CO₂, and gas exchange
MSLT	<ul style="list-style-type: none"> Measures propensity to fall asleep and for entry to REM sleep In conjunction with PSG, the gold standard for evaluation of EDS Indicated in children for evaluation of narcolepsy and other hypersomnias 	<ul style="list-style-type: none"> Usually performed in sleep laboratory on the day following nocturnal PSG Consists of 5 nap opportunities of 20 minutes each in a darkened room given at 2-hour intervals Patients asked to lie quietly, close eyes, and try to fall asleep Sleep latency is defined as the time from lights out to stage I of sleep^c
MWT	<ul style="list-style-type: none"> Measures ability to remain awake Used primarily to monitor response to therapy for a sleep disorder, and for safety with regard to driving 	<ul style="list-style-type: none"> Conducted during patient's usual period of wakefulness Consists of 4 tests of 20 or 40 minutes each given at 2-hour intervals Patients asked to sit still and remain awake as long as possible^d

Abbreviations: CPAP, continuous positive airway pressure; EDS, excessive daytime sleepiness; EEG, electro-encephalography; EMG, electromyography; EOG, electro-oculography; MSLT, Multiple Sleep Latency Test; MWT, Maintenance of Wakefulness Test; OSA, obstructive sleep apnea; PLMD, periodic limb movement disorder; PSG, polysomnography; REM, rapid eye movement; RLS, restless legs syndrome.

^aCannot differentiate movement during sleep such as RLS from wakefulness, or wakefulness from sleep while the patient is lying awake but motionless.

^bThe need for in-laboratory assessment using multiple wires connecting the patient to monitors may undermine the ability to replicate normal sleep at home.

^cNormative values for sleep latency in children <8 years of age are unclear; mean values are particularly long in prepubertal children (up to 26 minutes) and shorter in adolescents.

^dNormative values for this test are not available for children/adolescents.

syndrome and periodic limb movement disorder, iron supplementation in those with ferritin levels <50 ng/mL is advised; pharmacotherapy with agents such as gabapentin or clonazepam may be recommended in cases unresponsive to iron supplementation.

Treatment plans for children with narcolepsy and idiopathic hypersomnia include education, behavioral changes, and medication, with the goal of improving their quality of life. Children's families, other caregivers, and friends require education about the disorder. The school should be notified of the child's need for specific accommodations (eg, planned naps, extended time on examinations), commonly included in an

Individualized Education Program/504 plan. Behavioral changes are essential and include regular sleep-wake schedules, short planned naps 1 to 3 times daily, increased physical activity, and weight management. As there is no cure for narcolepsy and idiopathic hypersomnia, a number of medications are prescribed off-label to control EDS (eg, modafinil, methylphenidate) or cataplexy (eg, antidepressants).⁵⁰ Notably, sodium oxybate is Food and Drug Administration approved for the treatment of EDS or cataplexy in patients ≥7 years of age with narcolepsy⁵¹ based, in part, on a recent phase 3, randomized, placebo-controlled study in children and adolescents.⁵²

Conclusions

Excessive daytime sleepiness is common in children and can have serious adverse effects when undiagnosed and untreated. Improper diagnosis can lead to inappropriate use of medications that could worsen sleepiness and associated disruptive behaviors. Although EDS has many potential causes in children, the correct diagnosis may be identified through a systematic and thorough approach. Behavioral and educational interventions are preferred for treatment of most sleep disorders in children; and drug therapies may be effective adjuncts.

Author Contributions

All authors drafted the manuscript and have contributed equally to this work. All the authors have seen and approved the submission of this version of the manuscript and take full responsibility of the manuscript.


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