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A Brief Primer on Sleep for Pediatric and Child Clinical Neuropsychologists

Dean W. Beebe, Ph.D.
Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio. Department of Pediatrics, University of Cincinnati College of Medicine, Cincinnati Ohio

Abstract

Sleep problems are common in the children seen by pediatric and child clinical neuropsychologists, and these problems have the potential to significantly impact the child and his or her family. All are treatable to some degree, and some respond extremely well to existing treatments. This paper provides a brief overview of the impact, nature, screening, and treatment for childhood sleep problems, with a particular emphasis on issues relevant to practicing neuropsychologists.

Although some of the landmark discoveries on sleep were made via the study of children (e.g., Aserinsky & Kleitman, 1953), pediatric sleep medicine has arguably advanced most dramatically in the past two decades, and professional education has struggled to keep pace. Few medical training programs offer more than 5 hours of concentrated teaching on sleep, and the modal exposure is much less (Mindell, Moline, Zendell, Brown, & Fry, 1994; Rosen et al., 1998). Similarly, a recent survey found that only 5–8% of clinical psychology graduate or internship training programs offered formal coursework on sleep and about half offered no instruction on the topic whatsoever, even through symposia or individual lectures (Meltzer, Phillips, & Mindell, 2009). While the impact of this paucity of training is not known for clinical psychologists, the parallel training concern in medicine has resulted in many pediatricians incorrectly answering basic sleep knowledge questions and feeling unsure of their ability to address sleep concerns in their patients (Owens, 2001; Papp, Penrod, & Strohl, 2002).

There are several reasons why pediatric and child clinical neuropsychologists should develop foundation knowledge of pediatric sleep disorders. First, the children and adolescents with whom they work are at high risk for sleep pathology. Children with a wide range of medical, neurological, psychiatric, and developmental conditions are at high risk for sleep disorder (Bandla & Splaingard, 2004; Cortese, Faraone, Konofal, & Lecendreux, 2009; Cortesi, Giannotti, Ivanenko, & Johnson, 2010; Dahl & Harvey, 2007; Dorris, Scott, Zuberi, Gibson, & Espie, 2008; Kothare & Kotagal, 2011; Rosen, Shor, & Geller, 2008), with sleep problems occurring in perhaps 75–80% of children with complex neurodevelopmental disabilities (Jan & Freeman, 2004). Second, sleep pathology can signal an unaddressed problem in the child, family, or environment, such as deteriorating neurological state or parental misunderstanding of the child's needs. Third, a child's sleep disorder can have a major impact on the family, with sleep disruption occurring in 15–85% of parents of children with chronic illnesses, and parental sleep quality mediating the

Contact Information: Dean Beebe, Ph.D., Division of Behavioral Medicine and Clinical Psychology (ML3015), Cincinnati Children's Hospital Medical Center, 3333 Burnet Avenue, Cincinnati, OH 45229, USA. Phone: 513-636-3489, Fax: 513-636-7756, dean.beebe@cchmc.org.

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relationship between child sleep and parental mood, stress, and fatigue (Meltzer & Mindell, 2006, 2007; Meltzer & Moore, 2008). Sleep deprivation is known to impair daytime cognitive and emotional functioning in adults (Goel, Rao, Durmer, & Dinges, 2009). In light of the other challenges they face, using resources to cope with their own fragmented sleep is not a luxury that parents of special-needs children can afford. Fourth, as will be detailed later, there is reason to believe that childhood sleep problems can cause or contribute to deficits in daytime neuropsychological function. Finally, there is increasing evidence that childhood sleep disorders can cause or contribute to other health problems. For example, untreated obstructive sleep apnea (OSA) can result in cardiovascular and metabolic abnormalities (Gozal, Capdevila, & Kheirandish-Gozal, 2008; Gozal & Kheirandish-Gozal, 2008). Early screening by all health care professionals, neuropsychologists included, may play an important role in obtaining appropriate treatments to prevent or reverse morbidity.

The goal of this paper is to help bridge the gap between the limited knowledge base on pediatric sleep that is possessed by many neuropsychologists and that needed for clinical care. The gap is too wide to cover in a single paper, so some topics are only briefly summarized or omitted entirely here in favor of topics more relevant to clinical practice. This paper begins by briefly summarizing the neurophysiology of sleep and arousal. It then reviews the evidence linking childhood sleep problems to waking neuropsychological functioning, summarizes the most common sleep problems seen in children, reviews the primary sleep assessment procedures, and offers suggestions for what can be done once a sleep problem is identified. In the process, the paper provides the clinician with new knowledge and tools to more effectively detect and work with the sleep problems of the children they see in daily clinical practice.

Overview of the Neurophysiology of Sleep and Arousal

While superficially straightforward, the regulation of sleep and arousal turns out to be remarkably complex. Over 40 chapters and 500 pages in the benchmark text *Principles and Practice of Sleep Medicine* (Kryger, Roth, & Dement, 2011) speak to the diverse genetic, physiological, chronobiological, pharmacological, and psychobiological aspects of sleep. Only a brief overview can be provided here, but readers who are interested in relatively targeted additional information are directed to the Sleep Research Society's *Basics of Sleep* text (Sleep Research Society, 2009) and Kothare and Kotagal's edited volume *Sleep in Childhood Neurological Conditions* (2011).

Regulation of sleep-wake states originates in a complex network of regions from the brainstem through the hypothalamus, via at least four major pathways. The two arousal-promoting elements of the classic reticular activating system (RAS) include (1) glutaminergic and cholinergic neurons originating in the upper brainstem that project to and promote activity in thalamic relay centers, which in turn excite widespread cortical regions, and (2) primarily noradrenergic and dopaminergic neurons from the upper brainstem and caudal hypothalamus that more directly ascend to and promote activity in broad regions of the cortex. Arousal is further promoted by (3) histominergic projections from the posterior hypothalamus to the cortex and (4) relative de-activation of primarily GABA-ergic projections from the ventrolateral preoptic nucleus of the anterior hypothalamus. Projections from the posterior hypothalamus also excite the sympathetic nervous system, impacting respiration, blood pressure, and heart rate. Orexin/hypocretin-producing neurons within the lateral hypothalamus stabilize the system; loss of these neurons causes sleep-wake instability and the intrusion of features of rapid-eye-movement (REM) sleep into waking periods, resulting in the symptoms of narcolepsy (described later).

As healthy individuals move into the lightest stages of Non-Rapid-Eye-Movement Sleep (N1 and N2), GABA-ergic pathways from the anterior hypothalamus become more active, inhibiting the brainstem origins of the RAS. Anterior hypothalamic regions also stimulate the parasympathetic nervous system, resulting in slowing of respiration, blood pressure, and heart rate. The electroencephalogram (EEG) shows progressive slowing, marked by thalamically-mediated bursts of rapid activity (sleep spindles) and intermittently spiking "K-complex" waveforms that characterize N2 sleep. Slow-wave sleep (SWS or N3) is characterized by large-amplitude, low frequency waveforms that result from synchronized firing of large neuronal groups, organized via thalamo-cortico-thalamic circuits. The individual is least able to be aroused during SWS, with forced arousals often accompanied by long delays before the individual is fully alert, and partial arousals often accompanied by parasomnias (see below).

Several times per night in healthy individuals, "REM-On" neurons in the brainstem and forebrain selectively activate the cholinergic aspects of the RAS, while aminergic and histominergic pathways remain suppressed. During REM, airway muscle tone is at a nadir, and there is almost complete absence of skeletal muscle tone due to glycine-based inhibition of motor signals travelling through the brainstem and upper spinal cord. The EEG appears superficially like wakefulness due to the activation of thalamic, limbic, and some posterior cortical regions, despite the fact that there are no outward signs of wakefulness and there is continued deactivation of anterior cortical regions (Nofzinger & Maquet, 2011).

Two major processes interact to result in the typical sleep-wake cycle evident in healthy humans past infancy (Achermann & Borbely, 2011). The first is a roughly 24-hour circadian cycle ("Process C") which most heavily promotes wakefulness in the late afternoon/early evening and promotes sleep in the early morning. The timing of this cycle is heavily influenced by bright light exposure, mediated by activity of the suprachiasmatic nucleus, a melatonin-secreting "master clock" that projects to other aspects of the hypothalamus. The second major process, the sleep homeostat or "Process S", involves a gradual accumulation of sleep pressure across periods of wakefulness. Pressure is greatest at the time of sleep onset, then declines rapidly, especially SWS. The neurological mechanism of Process S is not fully understood, but appears to be related to the accumulation of extracellular adenosine in the basal forebrain.

Special Considerations for Neuropsychologists

Even this cursory summary suggests several mechanisms by which the children seen by neuropsychologists might display significant sleep pathology. For example, because the arousal circuits are redundant, it takes considerable damage or disruption of multiple circuits to result in coma. Given that severe traumatic brain injuries (TBI) are typically defined by depth of coma, children who have sustained a severe TBI will almost certainly have experienced injuries or disruption to multiple arousal systems, putatively contributing to problems with sleep-wake regulation in both the acute and long-term recovery periods (Beebe et al., 2007). Related to treatment factors and the disproportionate location of childhood brain tumors in midline and posterior fossa regions, survivors of childhood brain tumors also may experience injuries to one or more key aspect of the sleep-arousal system, resulting in difficulties with the appropriate onset, intensity, or maintenance of sleep or wakefulness (Rosen & Stone, 2011). Among children with epilepsy, nocturnal seizures can disrupt sleep, daytime seizures can diminish arousal post-ictally, and antiepileptic medications (e.g., GABA agonists) can chronically diminish arousal (c.f., Kaleyias & Kothare, 2011)). Indeed, a wide range of medications used in children with psychiatric, neurological, or other medical conditions have known or suspected side effects on arousal, sleep, or sleep staging (Schweitzer, 2011). Even the timing of medication doses can interfere

with sleep, especially when children must be awakened for administration, resulting in inadequate completion of Process S and resulting effects of sleep deprivation.

The Impact of Sleep Problems on the Child's Neuropsychological Functioning

A comprehensive review of the evidence linking sleep problems to neuropsychological functioning in children is beyond the scope of this paper and has been published recently elsewhere (Beebe, in press). Briefly, there are four primary sources of such evidence that, when taken together, strongly suggest that the daytime functioning of children is significantly impacted by sleep that is too short or of poor quality.

The first and largest line of evidence comes from correlational studies in "typical" children and case-control studies examining specific sleep disorders. The former have demonstrated that children's sleep quantity and/or quality correlates with their levels of daytime sleepiness and performance in school (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010; Drake et al., 2003; Fallone, Owens, & Deane, 2002; Wolfson & Carskadon, 2003), even when both sleep and school performance are measured objectively (i.e., not by the same parent- or self-reporter; (Keller, El-Sheikh, & Buckhalt, 2008). Interestingly, office-based tests of academic *knowledge* appear to be less sensitive than measures of actual *classroom performance*, suggesting the presence of functional deficits that may be "controlled away" during highly structured standardized testing, analogous to what has been described for the measurement of attention and executive functioning (Beebe, in press; Beebe, Ris, Kramer, Long, & Amin, 2010; Gioia, Isquith, Guy, & Kenworthy, 2000). Consistent with this hypothesis, inadequate sleep correlates with poor attention, impulse control, and behavior regulation (Paavonen, Porkka-Heiskanen, & Lahikainen, 2009; Paavonen et al., 2009; Sadeh, Gruber, & Raviv, 2002; Steenari et al., 2003). Poor sleep quality or quantity has also been associated with risk-taking behaviors and driving accidents in adolescents (O'Brien & Mindell, 2005; Pizza et al., 2010), as well as accidental injuries across childhood (Koulouglioti, Cole, & Kitzman, 2008; Owens, Fernando, & Mc Guinn, 2005; Stallones, Beseler, & Chen, 2006).

Case-controlled studies comparing children with sleep pathology to healthy controls further support an association between poor quality sleep and daytime deficits. The largest relevant literature is on obstructive sleep apnea (OSA; see also Table 1). Untreated OSA has been linked to poor classroom grades, sleepiness, inattention, hyperactivity, oppositional behaviors, and mood dysregulation in the vast majority of relevant studies in both children (Beebe, 2006) and adolescents (Beebe, Ris, et al., 2010). Again, office-based tests appear less sensitive than functional assessments of behavior or classroom performance, though there is some evidence of IQ deficits during the preschool and early grade-school years (but not later in school) and mixed findings on tests of attention, executive functioning, and learning/memory (Beebe, 2006; Kheirandish-Gozal, De Jong, Spruyt, Chamuleau, & Gozal, 2010; Spruyt, Capdevila, Kheirandish-Gozal, & Gozal, 2009). Non-randomized studies have shown improved daytime functioning following surgical correction of uncomplicated OSA (Beebe, 2006; Garetz, 2008), though the only large-scale randomized intervention study was still in data collection at the time of this writing. Outside of OSA, childhood manifestations of Restless Leg Syndrome and Periodic Limb Movement Disorder also have been linked to hyperactivity/impulsivity and inattention (Cortese et al., 2005; L. M. O'Brien, 2009), but the research literature remains small.

The third line of evidence is quasi-experimental, in which middle- and high-school students were studied while their sleep duration was systematically influenced by changes in school start times. Although not a one-to-one correspondence, later school start times result in students getting more sleep due to later morning awakening and minimally changed

bedtimes (Carskadon, 2002; Danner & Phillips, 2008; Dexter, Bijwadia, Schilling, & Applebaugh, 2003; Hansen, Janssen, Schiff, Zee, & Dubocovich, 2005; Wahlstrom, 2002a, 2002b; Wolfson, Spaulding, Dandrow, & Baroni, 2007). Later start times are associated with less subjective and physiological sleepiness (Carskadon, 2002; Dexter, et al., 2003; Wahlstrom, 2002a, 2002b; Wolfson, et al., 2007), improved enrollment stability and better attendance among the least stable students (Wahlstrom, 2002a, 2002b), less tardiness (Wahlstrom, 2002a, 2002b), fewer teen driving accidents (Danner & Phillips, 2008), and slightly fewer sick days and depressive symptoms (Wahlstrom, 2002a, 2002b).

The fourth line of evidence is experimental. In contrast to the hundreds of publications on experimental sleep deprivation that have been conducted on adults, only 8 studies had been published on children or adolescents at the time of this writing. Compared to when they are well-rested, experimentally sleep-deprived children and adolescents (1) are subjectively and physiologically sleepier (Beebe et al., 2008; Carskadon, Harvey, & Dement, 1981a, 1981b; Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001; Fallone, Acebo, Seifer, & Carskadon, 2005; Sadeh, Gruber, & Raviv, 2003; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010); (2) show more inattentive behaviors (Beebe, et al., 2008; Beebe, Rose, & Amin, 2010; Fallone, et al., 2001; Fallone, et al., 2005) but mixed effects on one-on-one attention tests (Beebe, in press); (3) report less positive affect despite either no change in self-reported negative affect (Talbot, et al., 2010) or a parent-reported increase in angry or oppositional behaviors (Beebe, et al., 2008); (4) show diminished creativity and reasoning skills on some tasks (Randazzo, Muehlback, Schweitzer, & Walsh, 1998) and diminished executive functioning in daily life according to parent- and self-report forms (Beebe, et al., 2008); and (5) display diminished performance in actual and simulated classroom settings (Beebe, Rose, et al., 2010; Fallone, et al., 2005). Interestingly, although correlational and case-control data would seem to suggest that poor sleep quality or quantity results in hyperactivity, impulsivity, or other externalizing behaviors in children, such an effect has not been evident so far in experimental studies (Beebe, in press).

Correlational, case-control, quasi-experimental, and experimental research designs each have unique strengths and limitations, such that the most confident conclusions can be drawn by a convergence of evidence. Such a convergence strongly suggests a causal and functionally significant impact of inadequate sleep quantity or quality on the neuropsychological functioning of children and adolescents, particularly in real-world settings. Indeed, over a dozen studies to date have found longitudinal associations between childhood sleep problems (variously defined) and the development of mood, attention, and behavior problems over time (Beebe, in press).

The voluminous adult sleep deprivation literature suggests additional avenues for investigation in children. In adults, experimental sleep deprivation can induce cognitive and psychomotor slowing, diminished working memory, poor cognitive flexibility, diminished situational awareness, and weakened recognition of the facial expression of emotions (Goel, et al., 2009; van der Helm, Gujar, & Walker, 2010). Adult sleep deprivation research has also documented a significant impact on memory consolidation that extends beyond the brief period covered by published neuropsychological tests (Ellenbogen, Hulbert, Jiang, & Stickgold, 2009; Kopasz et al., 2010; Stickgold & Walker, 2007; Walker, 2010) and that may have implications for the development of children with chronic sleep pathology. Finally, adult imaging studies have demonstrated intriguing alterations in neural processing due to sleep deprivation (Goel, et al., 2009) which are only beginning to be explored in children (Beebe, Difrancesco, Tlustos, McNally, & Holland, 2009; Beebe, Rose, et al., 2010; Key, Molfese, O'Brien, & Gozal, 2009). These few pediatric studies suggest that, as in adults, the brains of healthy children and adolescents appear to respond to mild sleep restriction by launching an initial compensatory response, in which more intense or

widespread neural activation occurs than is elicited by the same tasks when the individuals are well-rested. Even so, study authors caution that findings should be considered preliminary, and very little is known about the state of the pediatric brain at the time that any such compensatory neural responses fail.

The mechanisms by which inadequate sleep impairs daytime functioning have primarily been explored in animal models and adult humans. Most proximal to behavior, there is evidence to suggest that prefrontal cortical regions may require the greatest "recovery" during sleep, and are consequently differentially sensitive to inadequate sleep (Durmer & Dinges, 2005; Horne, 1988), while others have observed more diffuse deficits associated with long, boring tasks that seem to be associated with instability in sleep-wake states (Goel, et al., 2009). Even more mechanistic hypotheses suggest that inadequate sleep alters gene regulation, inhibits neurogenesis, induces a glucocorticoid stress response, and induces systemic inflammation (Thomas, 2011). Comparatively little mechanistic work has been done in immature animals or human children. However, given the unique needs and plasticity of the developing nervous system, there have been concerns about the cumulative effect of chronic sleep problems on neural organization (Beebe, 2008, in press; Jan et al., 2010).

Special Considerations For Neuropsychologists

The vast majority of research examining the impact of sleep problems on daytime functioning has been conducted in otherwise healthy individuals. High base rates of sleep problems are seen in children with developmental or acquired neurological conditions, but there have been no published experimental sleep restriction protocols in these populations. As a result, although studies of healthy children suggest that inadequate sleep can cause inattention and daytime sleepiness, it is not clear whether children with special needs are particularly vulnerable, or whether sleep restriction has a unique role in maintaining or exacerbating symptoms in such children (e.g., whether those with autism show greater social impairment when systematically sleep-deprived).

Conversely, while there is some research to suggest that addressing sleep problems in these children can improve their daytime functioning, most samples have been small and findings have been inconsistent (Richdale & Wiggs, 2005). For example, there is good evidence that children with autism spectrum and insomnia display improved sleep with melatonin treatment, but data related to daytime symptoms and behavior are limited (Rossignol & Frye, 2011). The only relevant placebo-controlled trial to date indicated improved daytime behaviors with melatonin treatment in 17 children with autism spectrum disorders and severe sleep problems, but conclusions are limited by the small sample and concerns about generalizability to groups with less severe sleep problems (Wright et al., 2011).

Even relatively large studies have yielded mixed results. For example, in a placebo-controlled double-blinded randomized clinical trial, a sample of 105 children with attention-deficit/hyperactivity disorder (ADHD) and insomnia had earlier sleep onset and about 30 minutes more sleep per night while taking melatonin than a placebo, but without any discernible short-term effect on daytime symptoms (Van der Heijden, Smits, Van Someren, Ridderinkhof, & Gunning, 2007). However, among the 94 participants who then engaged in a follow-up study 3 years later, 71% of parents reported that melatonin improved the behavior of their child, and an even larger majority reported continued daily use of melatonin daily (65%) or occasionally (12%; Hoebert, van der Heijden, van Geijlswijk, & Smits, 2009). The available evidence strongly supports the *presence* of certain sleep problems in children with ADHD, including variable sleep onset times, restlessness during sleep, objectively-measured daytime sleepiness, and parent-reported symptoms of sleep-disordered breathing and bedtime resistance (Cortese, et al., 2009; Gruber, 2009; Owens,

2008). However, because there is very little research that addresses whether ADHD symptoms remit with successful treatment of sleep problems, at least two competing hypotheses remain viable: (a) a subgroup of children with behavioral symptoms that meet criteria for ADHD might have primary sleep disorders "in disguise", or (b) some or all of the relationship between sleep problems and clinically-defined ADHD might be due to some other factor (e.g., neurotransmitter anomalies or arousal dysregulation affecting both sleep and waking states; (Owens, 2008).

Treatment trials for otherwise healthy children with sleep apnea have yielded results suggestive of improved behavior and objectively-measured attention (Beebe, 2006; Garetz, 2008). However, none of the published work in this area has involved treatment randomization or placebo control in any sample, let alone one comprised of children with special needs. In a promising development, adults with both epilepsy and OSA show improved seizure control with OSA treatment (Parisi et al., 2010). Although the impact of OSA on seizure frequency in children with epilepsy is not fully known, a recent open-label trial of melatonin in children with intractable epilepsy and insomnia symptoms reported improvements both in sleep and in seizure control (Elkhayat et al., 2010).

Systematic screening and treatment of sleep disorders in children with special needs remain relatively new, and initial efforts focused on documenting the presence of sleep problems and initial efficacy of interventions to improve sleep. It is anticipated that future research will also document the impact of sleep interventions on daytime functioning. For now, however, it seems reasonable for the pediatric or child-clinical neuropsychologist to discuss with families how sleep problems *may* contribute to daytime dysfunction and/or parental stress, but not to make promises that daytime symptoms will improve with a sleep treatment.

Common Sleep Problems in Children

Nearly 100 disorders are listed in the current *International Classification of Sleep Disorders* (ICSD-2; (American Academy of Sleep Medicine, 2005). The current paper focuses on the most common sleep problems in children.

Dyssomnias

Table 1 summarizes what, prior to 2005, had been codified as dyssomnias. Although not used in the current nomenclature, the term has some heuristic value in highlighting conditions that, because they shorten or disrupt sleep, carry a strong potential to impact daytime functioning. Indeed, for most of these conditions, the ICSD-2 explicitly requires that "the sleep disturbance is associated with impairment of social, occupational, or other areas of functioning" (American Academy of Sleep Medicine, 2005). When that impairment is related to repeated difficulty with sleep onset or maintenance despite adequate time and opportunity for sleep, the term *insomnia* is used. The *sleep-related breathing disorders* involve poor respiration during sleep due to recurrent upper airway obstruction (obstructive sleep apnea), recurrent lack of appropriate respiratory effort (central sleep apnea), or other factors. The *sleep-related movement disorders* encompass conditions in which daytime impairment results from recurrent, involuntary movements during sleep, except for restless leg syndrome, in which the movements are intentional efforts to lessen uncomfortable parasthesias. When daytime impairment is due to abnormal regulation of an individual's internal day-night "clock" or the chronic misalignment of that internal clock with environmental demands, a *circadian rhythm sleep disorder* is diagnosed. Finally, when excessive daytime sleepiness (hypersomnia) is present for other reasons, a fifth diagnostic cluster encompasses *hypersomnias of central origin not due to a circadian rhythm sleep disorder, sleep related breathing disorder, or other cause of disturbed nocturnal sleep.*

In the interests of brevity, Table 1 also lists only key features and symptoms for representative diagnoses in each of these diagnostic clusters. Even this brief summary highlights the diversity of potential contributors to sleep pathology. These include a mismatch between child factors and environmental contingencies (Behavioral Insomnias of Childhood), emotional factors (e.g., Adjustment and Psychophysiological Insomnias), and structural abnormalities of the airway (Obstructive Sleep Apnea; OSA), as well as abnormal neural control of sensory systems (Restless Leg Syndrome), smooth muscle (OSA), skeletal muscle (Periodic Limb Movement Disorder), the circadian rhythm (Delayed Sleep Phase Disorder), and stability and synchrony of various aspects of sleep and wakefulness (Narcolepsy).

Similar to the psychiatric nomenclature, ICSD-2 diagnoses are distinct, but can be comborbid. The disorders listed in Table 1 can not be the exclusive result of poor sleep hygiene, defined as the failure to adopt sleep habits that would normally promote healthy sleep. (Mindell & Owens, 2010) divided poor sleep hygiene practices for children into two groups. Practices that increase arousal – and are therefore prone to delay or disrupt sleep – include excessive or late caffeine use, smoking, stimulating play at bedtime, extended evening “screen time” (e.g., television, video games, text messaging), and excessive noise or bright light in the evening. Practices that tend to disrupt sleep organization include napping late in the day, having an inconsistent sleep-wake cycle, and frequent engagement in activities in bed that are incompatible with sleep. Sometimes, poor sleep hygiene results from well-intentioned parent, patient, or even physician-promoted attempts to address one problem in a manner that creates or maintains others. For example, a child with severe obstructive sleep apnea might consume excessive caffeine or take long afternoon naps to combat sleepiness, which in turn results in difficulty falling asleep at night, compounding the daytime symptoms.

Parasomnias

Table 2 summarizes the most common childhood parasomnias, loosely defined as undesirable events that accompany sleep (American Academy of Sleep Medicine, 2005). Although traditionally thought of as benign, these conditions warrant clinical attention for several reasons. First, the presence of these conditions can indicate other sleep problems that require treatment. For example, confusional arousals, sleepwalking, and sleep terrors involve partial arousal from SWS. They are exacerbated by conditions that result in partial arousals (e.g., OSA) or that increase the density of slow-wave sleep, such as sleep restriction or deprivation, which are worthy of clinical attention even if the parasomnias are tolerable. Second, conditions such as intense nightmares can be very distressing to the child and contribute to anxiety or reluctance around falling asleep. Third, these conditions can impact the family; while a child with chronic sleep terrors has no waking recall of the events, other family members may find them extremely disruptive to their own sleep and well-being. Fourth, some parasomnias can indirectly impact sleep. For example, nocturnal enuresis (bedwetting) can disrupt sleep if a “pull up” is not used, and also cause distress in the child and family. Fifth, in rare cases the events can have direct health consequences, such as when sleepwalking results in accidental injuries.

Finally, particularly when working with patients with neurodevelopmental conditions or brain injuries, it can sometimes be difficult to differentiate parasomnias from nocturnal seizures, which have very different pathophysiologies and treatment implications. Parasomnias arise during SWS in response to activation of subcortical pattern generators in the context of arousing stimuli, genetically-determined oversensitivity, or a combination of the two (Dhamija & Kotagal, 2011). Treatment tends to focus on parent education, matter-of-fact redirection of the child during an event, and reducing causes of increased SWS or arousals. In contrast, the seizures which tend to mimic parasomnias often arise from

epilepsy-related chaotic activation of ventral frontal cortex, often but not exclusively during lighter sleep stages (N1 and N2; Dhamija & Kotagal, 2011). Recurrent seizures are considered cause for concern and are generally treated with antiepileptic medications. Definitive differentiation of a given event can require its capture during an overnight EEG, though on rare occasions even that is insufficient (Kotagal, 2009; Manni & Terzaghi, 2010). Complicating matters, children with seizure disorders have disproportionately high rates of parasomnias (Crompton & Berkovic, 2009; Manni & Terzaghi, 2010). Fortunately, in many cases, the clinical signs, symptoms, and history factors listed in Table 3 can guide diagnostic impressions and treatment recommendations. The reader is cautioned, however, that these factors have received only limited cross-validation in adults, and no published cross-validation in children. Particularly puzzling or treatment-refractory cases should be seen by a neurologist with specialty expertise in sleep medicine.

Special Considerations For Neuropsychologists

Although neuropsychologists might occasionally conclude that a referred child has only a primary sleep disorder, often the sleep disorder is secondary to, or comorbid with an acquired or developmental neurological condition or its treatment. As noted earlier, medications or damage to key midline structures can alter the regulation of arousal and sleep-related modulation of muscle tone and respiratory control (Jan et al., 2008). Beyond such direct neural mechanisms, children with poorly-regulated endocrine conditions also commonly experience sleep problems, and sleep problems can impact aspects of endocrine functioning (e.g., OSA results in diminished nocturnal production of vasopressin, a hormone that slows urine production; (Alonso & Sheldon, 2010). Also present in some conditions, chronic or episodic pain can be extremely disruptive to sleep, and individuals with chronic pain and disrupted sleep experience particularly poor daytime functioning (Palermo, Fonareva, & Janosy, 2008). Children with craniofacial abnormalities, hypotonia, or obesity – all of which can occur in several different neurodevelopmental conditions – are at high risk for OSA (Mindell & Owens, 2010).

Even the behavioral insomnias of childhood, often considered to stem from parent behaviors, take on new dimensions when working with children who have neurodevelopmental disabilities. Problems with sleep onset or maintenance can result from a child's ritualistic behaviors, idiosyncratic pursuit or avoidance of sensory experiences, poor understanding of social cues or parental communications around sleep, problems communicating their comfort needs, and difficulties executing calming routines (Goldman & Malow, 2011; Jan, et al., 2008). Global or specific cognitive delays can also impact the effectiveness of a given parenting strategy. Emotional dysregulation, especially "activating" emotions such as anger or fear/anxiety, in a child with neurological impairments may make it extremely difficult for them to settle into sleep, or may result in fragmented sleep. Similarly, evening behavior problems (e.g., oppositionality associated with ADHD) can result in delayed sleep onset (Owens, 2005).

By combining information on sleep with that already obtained during a comprehensive neuropsychological evaluation (e.g., current behaviors and cognitive functioning, contextual/environmental factors, and medical/neurological, developmental and family histories), the neuropsychologist is an excellent position to address these diagnostic complexities in the service of formulating an effective intervention plan. At times, these complexities may be reflected in ICSD-2 diagnoses for sleep disorders secondary to another condition (Table 4).

Assessment Procedures

Table 5 introduces the most common sleep assessment procedures and summarizes key strengths and weaknesses of each. As also occurs in traditional neuropsychological assessment, the procedures that provide the most objective data tend to sacrifice the breadth of data collected or focus on gathering data outside of the natural setting. Conversely, those that emphasize the assessment of sleep in the natural setting generally sacrifice objectivity, precision, and information on sleep physiology.

Polysomnography (PSG) and actigraphy are used almost exclusively by sleep specialists, but it is good for neuropsychologists to know what these tools are well- and poorly-suited to assess. PSG is essential for the definitive assessment of sleep-related breathing disorders, sleep-related movement disorders, and some hypersomnias. PSG can also assist with the detection of nocturnal seizures if augmented with an expanded EEG montage, neurologically-trained scorer/interpreter, and altered timeframe for review (usual sleep scoring is done at a “paper speed” that obscures epileptiform discharges). Drawbacks of PSG relate to the fact that children must sleep in an unfamiliar setting while wearing monitoring equipment, which can fundamentally alter a patient’s sleep-related behaviors, sleep latency, onset, offset, and even arousals. At-home PSG is designed to allow the child to sleep in his or her home setting, but does not eliminate concerns about the impact of monitoring equipment, often includes only a limited array of sensors and, especially if unattended by a technician, may lose data due to sensor removal (Gay & Selecky, 2010).

Actigraphy involves wearing a wristwatch-sized device that records movements in a manner that (a) is generally unobtrusive and well-tolerated, (b) can provide a reasonable estimate sleep onset, offset, and duration in healthy populations, and (c) can record continuously while a patient undergoes their normal routines for several weeks or longer (Sadeh, 2011; Sadeh, Hauri, Kripke, & Lavie, 1995; Sadeh, Sharkey, & Carskadon, 1994). However, because most units record a single channel (movement), they miss a wide array of physiological variables measured by PSG, provide little information on behaviors leading up to sleep or during waking episodes, can miss PSG-verified awake states when the individual is lying or sitting still, and are prone to artifacts when sleep co-occurs with movement (e.g., sleeping in a moving car, parasomnias). Further, the data obtained can be compromised by poor patient adherence to wearing the devices.

Because both PSG and actigraphy require special equipment and training, it is unlikely that neuropsychologists who do not conduct sleep research or work frequently with sleep clinic patients will routinely use these in their own practice. In contrast, neuropsychologists will find that sleep diaries, questionnaires, and interviews are more readily integrated with their assessments. Of the many published sleep questionnaires, only two met all of Spruyt and Gozal’s (2010a, 2010b) psychometric quality criteria. Both are multi-dimensional parent-report questionnaires that generally take <10 minutes to complete. The Sleep Disturbance Scale for Children (Bruni et al., 1996) was written in Italian but subsequently translated to English (available at no cost from Dr. Bruni), but only Italian norms are available. The Sleep Disorders Inventory for Students (Luginbuehl & Kohler, 2009; http://www.sleepdisorderhelp.com/) was written in English and subsequently translated to Spanish, with American norms available across ages 2–18.

Sleep questions can also be integrated into clinical interviews. Dr. Judy Owens developed the “BEARS” acronym to cue pediatricians to ask about Bedtime problems, Excessive daytime sleepiness, Awakenings during the night, Regularity and duration of sleep, and Sleep-disordered breathing. This approach has been shown to result in better documentation of sleep issues and, importantly, to be feasible within a busy pediatric clinic (Owens &

Dalzell, 2005). If pediatricians can integrate acronym-based sleep screeners into their brief clinical interviews, neuropsychologists seem reasonably capable of doing so as well. Indeed, the author has found in his own practice that a similar brief sleep screener generally adds less than 5 minutes to a clinical interview and is well-tolerated by parents. Instead of using the BEARS approach, however, the author typically asks about sleep in chronological order, using the acronym BOWS: Bedtime, Overnight, Waking, Sleepiness. Table 6 provides suggested screening questions and potential follow-up questions. The questions are worded as if speaking to parents, but can be modified for discussions with children and adolescents, or to accommodate unique social, physical, or developmental issues.

Special Considerations For Neuropsychologists

Experienced pediatric and child-clinical neuropsychologists are accustomed to adapting their assessment procedures to the specific needs of the child and family being assessed. This need for flexibility is no less for sleep assessments. Children with neurological or developmental disorders sometimes do not tolerate PSG, resulting in failed or technically limited studies. Sleep centers that work regularly with such children have specialized technicians and equipment, and it can be helpful for parents to bring familiar objects to increase the child's comfort. Because it is similar to wearing a wristwatch, actigraphy is typically well-tolerated by patients seen by neuropsychologists, and procedures are available to ensure that the units are worn by neurologically impaired individuals (Ancoli-Israel, 2000). However, in rare cases tactile sensitivities may require the unit to be worn over clothing or the procedure may not be tolerated. Sleep diaries, questionnaires, and interviews are less directly affected by acquired or developmental neurological conditions. However, clinicians are advised to consider whether a given sleep question or scale maintains its original meaning in special populations. In children who are non-ambulatory, for example, remaining in the bedroom is a function of the child's physical condition, and limit-setting problems can still be evident in vocalizations.

Expectations for a given behavior also change developmentally, and the clinician needs to consider a child's neurodevelopmental and chronological age when comparing to published norms. The reader is referred to recent excellent reviews for more comprehensive coverage of developmental changes in the sleep of healthy children (e.g., Jenni & Carskadon, 2009; Mindell & Owens, 2010). Briefly, large-scale surveys (Iglowstein, Jenni, Molinari, & Largo, 2003; National Sleep Foundation, 2004, 2006) have placed the 25th and 75th percentiles for sleep in a 24-hour period at roughly 10 – 12 hours during the preschool years and 9 – 10 ½ hours during elementary school. Most of that decline is attributable to the loss of daytime naps, which occur daily or near-daily in a quarter of healthy 4-year-olds, but in less than 2% of US children ages 6 and older. Average sleep durations on non-school nights decline only slightly from elementary school into adolescence, but school-night sleep drops to 7 – 8 ½ hours on average, largely related to later bedtimes but stable or earlier school start times. The rates of specific symptoms change developmentally as well. For example, the dramatic decline in SWS that occurs across adolescence is accompanied by a reduction in partial-arousal parasomnias.

Age-appropriate expectations for sleep may not hold well in a child with an acquired or developmental neurological condition, and it may be more important to determine the degree to which a behavior promotes or detracts from optimal functioning. For example, insisting on "normal" sleep duration in a child whose sleep need has been increased due to a brain injury may, in fact, cause or exacerbate daytime dysfunction. Careful questioning around the sleep patterns that parents believe have facilitated better or worse daytime functioning, perhaps combined with intentional 3–5 - day trials of different sleep regimens, can provide insight into the most functional sleep expectations for a given child, even if it is not demographically "normal."

What To Do If You Identify a Sleep Problem

Sleep Hygeine

Whenever a sleep problem is identified, the first step should be to examine whether poor sleep hygiene is contributing. As noted earlier, poor sleep hygiene involves behaviors that interfere with adequate sleep duration or quality, and that typically either increase arousal around bedtime or diminish sleep organization (Mindell & Owens, 2010). Table 7 summarizes the recommended elements of good sleep hygiene. Although in some ways intuitive, adherence to these sleep hygiene recommendations is entirely volitional and rarely complete. Working with parents to establish and behaviorally reinforce appropriate adherence to good sleep hygiene can yield impressive results. Good sleep hygiene is also essential to the success of other interventions.

Sleep Treatments

When improved sleep hygiene alone is insufficient, the final column of Tables 1 and 2 list empirically-supported intervention techniques for each condition. Prominent sleep experts, including physicians who have received material support from pharmaceutical companies, have concluded that *for the large majority of pediatric sleep conditions, the treatment of choice is behavioral or cognitive-behavioral therapy, and medication is an option only when accompanied by behavioral interventions* (Gringras, 2008; Jan, et al., 2008; Mindell & Owens, 2010; Owens, 2009; Owens et al., 2005). There is a paucity of both safety and outcomes research on sleep medications in children and, as of this writing, no FDA-regulated medication had been approved for use as a hypnotic in children. Owens (Mindell & Owens, 2010; Owens, 2009) recently reviewed the available evidence on efficacy, side effects, and risks associated with the pharmacotherapy of pediatric insomnia, laying out general guidelines for use when such treatment is clinically indicated.

Melatonin deserves special mention because it has received considerable popular exposure and is available over-the-counter in the United States as a non-FDA-regulated dietary supplement. Exogenous melatonin mimics the endogenous hormone produced by the pineal gland in response to decreased light exposure in the healthy circadian rhythm. Although large-scale randomized controlled trials are still lacking, early studies support its efficacy in treating insomnia symptoms associated with autism spectrum disorders (Miano & Ferri, 2010; Rossignol & Frye, 2011), ADHD (Bendz & Scates, 2010), and intractable epilepsy (Elkhayat, et al., 2010). In non-neurological populations, melatonin in relatively low doses (<3 mg) shifts the sleep phase earlier on the clock, with the greatest impact when taken several hours before bedtime (Lack & Wright, 2007). At higher doses (5–10 mg), melatonin appears to have mild short-lasting hypnotic effects (Owens, 2009). Although short-term safety findings have been favorable, long-term safety has not been established. Importantly, because there is no FDA regulation, the purity and dose of melatonin in the products sold over the counter can vary (Owens, 2009).

Several recent reviews provide detailed expositions of key principles, methods, and empirical support for behavioral and cognitive-behavioral treatments for sleep disorders in children (Dahl & Harvey, 2007; Gordon, King, Gullone, Muris, & Ollendick, 2007; Meltzer, 2010; Meltzer & Mindell, 2004; Mindell, Kuhn, Lewin, Meltzer, & Sadeh, 2006), including their use in children with neurodevelopmental conditions (Jan, et al., 2008; Richdale & Wiggs, 2005). While many neuropsychologists will find that follow-up sleep interventions are outside of their scope of practice, others – particularly those outside of larger tertiary care centers that have strong pediatric psychology or sleep medicine programs – may find themselves needing to be more directive in their recommendations about follow-up care. To allow the reader to make empirically-supported recommendations, a brief overview is

provided here. For the behavioral insomnias of childhood, interventions typically use teaching and operant behavioral principles (e.g., positive reinforcement, selected ignoring, limiting choices) to promote parents' ability to set a consistent age-appropriate bedtime, develop a bedtime routine, and teach a child to fall asleep independently. Nighttime fears and nightmares have been successfully addressed via behavioral reinforcement, desensitization, relaxation, positive self-talk and imagery, reduction of frightening stimulation prior to bed, and deferring detailed discussion and instruction in coping until daytime (only brief cues given at night). For psychophysiological insomnia, cognitive-behavioral treatments are used to reduce sleep-related arousal (relaxation training, positive imagery) and classical conditioning principles employed to replace negative associations to the bed (as a place of frustration and anxiety) with more restful associations via intentional sleep restriction (faded over time) and limiting use of the bed.

There are some conditions for which physical interventions are clearly indicated, but with adjuvant behavioral interventions. OSA is sometimes treated with nasal positive airway pressure (PAP), for which the child must wear a snug facial interface during sleep; behavioral treatments are often needed to ensure adherence (Koontz, Slifer, Cataldo, & Marcus, 2003). While narcolepsy often warrants medication intervention, behavioral therapies can be helpful to maximize the quantity and quality of planned sleep, to educate the patient, family, and school, and to build practical strategies for coping with the condition. Delayed Sleep Phase Disorder usually requires a combination of physiological interventions (bright light therapy in the morning, melatonin several hours before bed) with behavioral treatments using stimulus control methods.

Referrals

Most neuropsychologists develop a list of favorite providers to which they can refer patients for follow up and specialty care. Sleep specialists should be on that list. A referral should be made whenever the neuropsychologist is unwilling or unable to treat a child with a sleep disorder, or when a clinician concludes that: (1) a PSG and/or Multiple Sleep Latency Test is needed (e.g., chronic loud snoring, suspicious nocturnal movements, unexplained daytime sleepiness, other narcolepsy symptoms); (2) a sleep problem is treatment-resistant or baffling; (3) a sleep problem is extremely disruptive to the family or associated with marked functional deficits; (4) there is a concerning risk of injury; or (5) a medication intervention may be needed. Table 8 lists major information sources for clinicians who are not specialists in sleep medicine, but who are interested in learning more or in referring to a board-certified provider or clinic.

Concluding Statements

Sleep problems are common in the children seen by pediatric and child clinical neuropsychologists, and these problems have the potential to significantly impact the child and his or her family. All are treatable to some degree, and some respond extremely well to existing treatments. It is imperative that clinicians learn more about these conditions and their potential causes and consequences, routinely screen for them in clinical practice and, as appropriate, treat or refer for appropriate follow-up care.

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Table 1

Common childhood sleep disorders that result in inadequate and/or disrupted sleep

Condition	Key Features/Symptoms	Primary Evaluation	Primary Intervention(s)
Behavioral Insomnia of Childhood (BIC), Limit Setting Type	• Child noncompliant behavior at bedtime or refusal to return to bed after night waking • Caregiver not setting/enforcing adequate limits	Clinical Interview	• Parent training using behavior management principles
BIC, Sleep Onset Association Type	• Sleep onset at bedtime and after night wakings requires highly specific environmental conditions	Clinical Interview	• Parent training using behavior management principles
Adjustment Insomnia	• Problems with sleep onset or maintenance temporally associated with a stressor	Clinical Interview	• Reduce stress, build coping tools • Treat underlying anxiety
Psychophysiological Insomnia	• Difficulties with sleep onset or maintenance • Heightened negative arousal (e.g., physical tension, anxiety) regarding sleep • Better sleep when away from own bed • Intrusive thoughts/rumination prevent sleep	Clinical Interview, Sleep Diary	• Stimulus control (bed for sleep only) • Cognitive Restructuring • Sleep restriction faded over time • Relaxation techniques • In limited cases, medications
Obstructive Sleep Apnea (OSA)	• Chronic, loud snoring most nights • Sometimes witnessed breathing pauses despite continued respiratory effort	Polysomnography (PSG)	• If structural cause identified, surgery may work (e.g., adenotonsillectomy) • Positive Airway Pressure
Restless Leg Syndrome	• Uncomfortable parasthesias in legs, generally worse during inactivity and relieved by movement, which can delay sleep onset	Clinical Interview	• Iron supplements if serum ferritin low • Distraction techniques, massage • In limited cases, medications
Periodic Limb Movement Disorder	• During sleep, repetitive stereotyped limb movements usually 20–40 seconds apart	PSG	• Iron supplements if serum ferritin low • In limited cases, medications
Delayed Sleep Phase Disorder	• Late sleep onset that doesn't change even with effort and good sleep hygiene • Marked difficulty rising in morning • Good sleep quality and quantity if sleeps late	Clinical Interview, Sleep Diary	• Bright light in morning • Melatonin around dinner time • Chronotherapy

Condition	Key Features/Symptoms	Primary Evaluation	Primary Intervention(s)
Narcolepsy	• Extreme daytime sleepiness, fluctuating arousal • Characteristic pattern during Multiple Sleep Latency Test (MSLT) and PSG • Usually Cataplexy (sudden loss of muscle tone in response to strong emotions) • Often hypnagogic hallucinations, sleep paralysis, nocturnal sleep disruption	Clinical Interview, PSG, MSLT	• Medication often used, typically a stimulant and REM suppressant • Alter behavior patterns to maximize quantity and quality of planned sleep • Educate child, parent, school • Build pragmatic coping strategies and provide support

Table 2

Common childhood parasomnias

Condition	Key Features/Symptoms	Primary Evaluation	Primary Intervention(s)
Confusional arousals	• Partial arousal from sleep characterized by confusion, difficulty waking, sometimes agitation • Child does not recall event later when awake	Clinical Interview, in some cases PSG and Sleep Diaries are helpful in differential diagnosis	• Ensure safety • Screen for and treat potential causes • Provide parents with reassurance, education, and behavioral guidance • Avoid waking and redirect to bed or comfortable position in bed (if need to urinate seems to precipitate partial arousal, redirect to bathroom) • Avoid next-day discussions • In rare cases, medication
Sleepwalking (somnambulism)	• While still asleep or with altered consciousness, walking or engaging in nonsensical behaviors or routine behaviors in the wrong context • Child does not recall event later when awake		
Sleep terrors (aka “night terrors”)	• Sudden episode of both behavioral and autonomic nervous system symptoms of terror while still asleep or with altered consciousness • Child does not recall event later when awake		
Nightmares	• Child awakens from sleep with recall of frightening dream, perhaps crying or screaming • Reasonably coherent and oriented • May take time to calm enough to return to sleep • Child can later recall period of awakening	Clinical Interview	• Reduce frightening or stressful events, especially close to bedtime • Provide reassurance • Build self-soothing skills and tools • In rare cases, medication
Sleep Enuresis (“bedwetting”)	• Involuntary voiding during sleep at least twice per week in children > 5 years of age. • Designated “primary” if never been dry >6 mos, “secondary” if starts after dry period >6 mos	Clinical interview	• Screen for and treat possible causes (e.g., neurological, urological, OSA) • Reassurance and use of pull-up if not a concern in family • Enuresis alarms + behavior therapy • In limited cases, medication

Table 3

Clinical Patterns of Parasomnias versus Nocturnal Seizures.

	Parasomnias	**Epilepsy**
Frequency per night	1 or less per night	Can cluster in several events per night
Timing	First third of night	Varies
Sleep stage from which events arise	N3 (slow-wave sleep)	Generally N1 or N2 (“light” sleep)
Typical duration	>5 minutes	<5 minutes
Wandering outside of bedroom	Common in sleepwalking	Uncommon
Complex, directed behaviors (e.g, picking up objects, dressing)	More common	Uncommon
Dystonic posturing	Uncommon	More Common
Stereotyped events or automatisms	Uncommon	Common
Later recall of event when awake	No	More common
Family history	Very often includes parasomnias, rarely epilepsy	May include epilepsy or parasomnias

Adapted from (Crompton & Berkovic, 2009; Derry et al., 2006; Kaleyias & Kothare, 2011; Kotagal, 2009; J.A. Mindell & Owens, 2010). It is important to keep in mind that these patterns are not fully sensitive or specific, nor have they been cross-validated in pediatric samples. Patients for whom significant questions remain regarding a set of clinical symptoms should be referred to a pediatric neurologist with expertise in sleep medicine.

Table 4

Sleep Disorders Secondary to Other Conditions

Circadian Rhythm Sleep Disorder due to Medical Condition	Sleep-Related Movement Disorder due to Drug or Substance
Other Circadian Rhythm Sleep Disorder due to Drug or Substance	Sleep-Related Movement Disorder due to Medical Condition
Insomnia due to Mental Disorder	Parasomnia due to a Medical Condition
Insomnia due to Drug or Substance	Parasomnia due to Drug or Substance
Insomnia due to Medical Condition	Other Physiological (Organic) Sleep Disorder
Hypersomnia due to Medical Condition	Narcolepsy due to Medical Condition
Hypersomnia due to Drug or Substance	

Table 5

Primary Sleep Measurement Tools

Tool	Description	Strong at Measuring	Weak at Measuring
Inpatient Polysomnography (aka PSG, polysomnogram)	Overnight sleep study in a closely-monitored and well-controlled sleep laboratory. Includes monitoring of a limited-montage EEG with electro-oculogram, respiratory effort, air exchange and CO_2 content, blood oxygenation, heart rate, limb movements, and snoring.	• Sleep Stages • Sleep-disordered breathing • Periodic limb movements • EEG-based arousals • Some seizure activity, but only if using an expanded EEG montage and with the right reviewing timeframe	• Typical sleep latency, onset, offset, behaviors around sleep • Sleep in children sensitive to artificial setting and monitoring • Infrequent events • Seizure activity when traditional PSG montage and scoring are used
At-Home PSG	As above, but with more limited monitoring. Nature of at-home PSG varies by manufacturer.	• Usually a subset of the above factors are assessed well, and less expensively and in a more natural sleep setting.	• Same as above (except in more natural setting), plus any given data lost if sensors slip off or are placed incorrectly
Multiple Sleep-Latency Test	A standardized series of EEG-monitored nap opportunities over the course of a day	• Excessive daytime sleepiness • Sleep-onset REM, which is helpful in narcolepsy diagnosis	• Data not listed as strengths • Sleepiness in children sensitive to artificial setting and monitors
Actigraphy	Small device worn on the wrist or ankle that records movement, from which sleep/wake can be estimated very well in healthy individuals.	• General sleep-wake patterns and movement-related arousals across multiple nights • "Natural" sleep patterns in a relatively unobtrusive fashion	• Respiration, EEG during sleep • Sleep while moving (e.g., in car, parasomnias, seizures) • Anything if the unit not worn
Sleep Diaries	Patient/parent prospectively asked to record bedtime, sleep onset, sleep offset, and important events during sleep (e.g., night wakings)	• Sleep across multiple nights • Infrequent events • Sleep-related behaviors and events in the natural setting	• Respiration, EEG, cardiac functioning during sleep • Events not witnessed or recalled well by reporter
Questionnaires	Self- or parent-report scales that ask for retrospective report of observed sleep behaviors/habits	• Sleep patterns over broad spans in the natural setting • Infrequent events	• Same as with sleep diaries, but with added concerns about precision and recall biases
Clinical Interviews	Unstructured or semi-structured interviewed about selected aspects of sleep	• Same as questionnaires, but allowing for examiner follow-up on areas of ambiguity or particular concern	• Same as questionnaires, but with added concerns if interviewer is not thorough or talented

Table 6

Suggested Sleep Screening Questions (Mnemonic: BOWS)

Domain	Parent Screening Questions (reword for patient)	Possible Follow-Up Questions (reword for patient)
Bedtime	• Does _____ have problems getting to sleep? • What time does _____ go to sleep on school nights? How about weekends?	• What happens when you try to get _____ to bed? • What do you think is causing the problem? • Is there a family routine in the evening? Tell me about it. • Is there "screen time" in the evening, like TV, video games, instant or text messaging, or internet activity?
Overnight	• Once asleep, does _____ wake up or get out of bed before morning? • Does _____ sleep soundly, or is there snoring, crying out, sleepwalking, or restlessness?	• What exactly happens? How many nights per week? • What do you do when this happens? • Does _____ seem awake at these times? • How long does it take for _____ to go back to sleep? • Are certain times of night worse than others?
Waking	• What time does _____ wake in the morning on school days? How about weekends? • How hard is it for _____ to wake in the morning? • Are the bedsheets pulled out and tangled, like _____ was very restless at night?	• What does it take to get _____ out of bed in the morning? • How late would _____ sleep if allowed to do so? • What does _____ do in the morning once awake?
Sleepiness	• Does _____ seem sleepy, tired, or "spaced out" during the day? • Does _____ nap or do other things to cope with sleepiness, such as have caffeinated drinks?	• How often does this happen? • Does it get in the way of learning or enjoying life? • Does it seem to change depending on how much sleep _____ gets or how calm that sleep seems to be? • When and how long does _____ nap? • When does _____ consume caffeine? How much?

Table 7

Key Elements of Good Sleep Hygeine

Key Principle	Additional Notes
Avoid caffeine late in the day or at high doses	The half-life of caffeine is ~6 hours, so two “energy drinks” at noon are equivalent to one such drink at 6 pm. Children with neurological disorders may have unusual responses to caffeine or its withdrawal.
During the day, get out in the light and move around, but avoid bright light and intense exercise in the evening.	Bright light during the day – especially sunlight – is important for helping to set the circadian clock to the typical day-night schedule. Similarly, vigorous daytime activity helps maintain a healthy weight (overweight is associated with sleep problems) and may influence both the circadian and homeostatic aspects of sleep propensity. However, bright light in the evening inhibits melatonin secretion, which prepares the body for sleep, and residual arousal after evening exercise may delay sleep onset.
Avoid cigarette smoke, especially at night	“Second-hand smoke” also counts. Nicotine can both delay and disrupt sleep, and smoke can irritate the upper airway, contributing to breathing obstruction.
Have a predictable evening “wind-down” routine	A subdued evening routine can help reduce arousal and build positive behavioral associations with bedtime.
Screen time should be minimized during the evening “wind-down”	Although superficially relaxing, screen time (e.g., TV) has been shown to delay sleep onset. There is a huge variety of stimulating entertainment options that are available via electronics, all of which are designed to maintain interest. If distraction is needed before bed, time-limited relaxing music, reading (or being read to by a parent) can suit the bill and are better at preparing the body for sleep.
Make sure the sleep setting is comfortable	While it is intuitive to recommend comfortable bedding and a mildly cool, quiet, and generally dark sleep space, children with neurological conditions may also have unique needs by virtue of motor limitations, sensory concerns, and even issues with regulation of body temperature.
Maintain a consistent sleep schedule	This may be particularly important for individuals prone to developing non-24-hour cycles, as environmental conditioning may need to take an even greater role.
Cater sleep duration and napping to the individual child’s needs	Napping may be helpful, especially for children who are young, have neurologically-mediated arousal difficulties, or are early in their recovery from brain injuries. However, long or late naps can interfere with sleep onset, so if a napping child is having difficulty falling asleep at night, naps may need to be made earlier, shortened, or eliminated altogether.

Adapted from (Jan, et al., 2008; J.A. Mindell & Owens, 2010)

Table 8

Sources for Referral Options and Additional Information

Source	Information Available	How to Access (as of time of print)
American Board of Medical Specialties	Searchable Index of Board-Certified Physicians with subspecialization in sleep (certified since 2007)	http://www.abms.org/Who_We_Help/Consumers/verifying.aspx Ph: (866) 275–2267
American Board of Sleep Medicine	Searchable Index of Board-Certified Sleep-Medicine Physicians (certified before 2007)	http://www.absm.org/listing.aspx Ph: (630) 737–9700
	Index of Board-Certified Behavioral Sleep-Medicine Specialists	http://www.absm.org/bsmspecialists.aspx Ph: (630) 737–9700
American Academy of Sleep Medicine	Searchable Index of AASM-Certified Sleep Medicine Centers	http://www.sleepcenters.org/ Ph: (630) 737–9700
	Topical, Searchable Links to Key Topic Areas	http://www.sleepeducation.com/ Ph: (630) 737–9700
	Information on Membership, Structure, and Initiatives	http://www.aasmnet.org/ Ph: (630) 737–9700
Sleep Research Society	Information on Membership, Structure, and Initiatives	http://www.sleepresearchsociety.org/ Ph: (630) 737–9702
National Sleep Foundation	Topical, Searchable Links in Key Topic Areas, e-mail update service, searchable listing of sleep doctors who are members	http://www.sleepfoundation.org/ Ph: (202) 347–3471
National Center for Sleep Disorders Research	Educational materials and contacts for research funding	http://www.nhlbi.nih.gov/about/ncsdr/ Ph: (301) 435–0199
Professionally-Oriented Books for Non-Sleep Specialists Interested in Sleep	Mindell, JA and Owens, JA (2010). *A Clinical Guide to Pediatric Sleep: Diagnosis and Management of Sleep Problems* (2nd Ed). Philadelphia, PA: Lippincott Williams and Wilkins.	Various online and “brick-and-mortar” booksellers
	Durand, V. M. (2008). *When Children Don’t Sleep Well: Interventions for Pediatric Sleep Disorders Therapist Guide*. New York: Oxford University Press.	
	Sleep Research Society (2009). *Basics of Sleep Guide* (2nd Ed). Darien, IL: Author	http://www.sleepresearchsociety.org/ Ph: (630) 737–9702