

Maturation of Normal Sleep Patterns - Childhood through Adolescence

Avi Sadeh, D.Sc

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The evolution of normal sleep patterns in children is one of the most fascinating maturational processes that attracts the attention of parents and professionals involved in child-care and developmental research. Although many aspects of this maturational process are visible to the interested observer, some of them require special methods of peeking into the “night life” of the child.

Sleep plays a major role in children’s well being. Sleep is strongly influenced by the child’s health status, psychological stress and family issues as well as by multiple aspects of his or her culture and environment. Also, children’s sleep patterns affect their well being within the same wide range of health and psychosocial phenomena. This chapter contains a brief description of the maturation of sleep-wake patterns in normal children and adolescents and a discussion of the factors influencing and influenced by the evolving sleep patterns of the child.

I. Consolidation of Nocturnal Sleep

Full-term newborns spend an average daily amount of about 16 hours in sleep. Their sleep is distributed around the clock, day and night, across a number of sleep episodes (five to six on average) with relatively short intervals of wakefulness between them (1,2,3).

Interestingly, significant variability in sleep consolidation exists already in newborns where the longest continuous sleep episode ranges between 50 and 300 minutes (3).

Although it may appear to the observer that newborns’ sleep is evenly distributed across the day and night, studies have shown that sleep is more concentrated in the nighttime hours than in daytime hours (3,4). During the first year of life a rapid maturational process leads to a clear preference to sleep during the night and the consolidation of a prolonged nighttime sleep episode also referred to as “sleeping through the night”, is achieved by most infants during this period (2,5-7). This maturational process is influenced by multiple biological and psychosocial factors. From the biological perspective it has been suggested that the synchronization of the sleep-wake cycle with

the light-dark cycle is mediated by the secretion of the pineal hormone melatonin which rises in the dark hours and drops in response to light exposure. It has been shown that maturation of the melatonin secretion occurs during the first 6 months of life (8), a period that corresponds with the accelerated process of sleep consolidation during the night. Furthermore, a significant association between melatonin secretion patterns and sleep-wake patterns has been demonstrated in 6-8 month old infants, suggesting that the two systems are indeed interrelated (9). In this study, sleep onset was associated with measures of melatonin secretion levels during the evening hours and sleep fragmentation was associated with inappropriate peak time of melatonin secretion. The relationship between the sleep-wake cycle and melatonin secretion patterns has also been demonstrated in older children (10). In blind children, disrupted sleep patterns have been associated with deviate melatonin secretion patterns (11). It has also been shown that exogenous melatonin administration may normalize sleep in blind children (12) and in some neurologically impaired children with severe sleep disruptions (13). However, a failure to achieve favorable therapeutic effects with melatonin has also been reported (14).

Beyond these melatonin-related and possibly other underlying biological mechanisms the infant is exposed to many environmental cues and psychosocial pressures that favors sleeping during the night and wakefulness during the day. Some of these psychosocial influences are described in chapter (McKenna's chapter).

Another way to describe the maturation of the sleep-wake system is to address the changes in daytime napping. As sleep consolidates during the night and sleep needs decrease, daytime sleep gradually disappears. A recent longitudinal study of naps in children from 6-months to 7 years of age indicates that the percentage of children who nap, the number of napping episodes and the time spent in naps decreases with age (15). In this study, most infants were napping two (83.7%) or three (16.3%) times per day at 6 months of age. By 3 years of age all the children were napping only once a day. Napping

almost completely disappeared at 7 years of age. No gender differences were found with regard to the napping habits of the children.

Interestingly, napping often reappears during adolescence as teenagers compensate on a sporadic basis for their accumulated sleep debt due to their exaggerated curtailment of nighttime sleep (see below).

The process of nighttime sleep consolidation is associated with night-waking problems that are the most prevalent sleep complaints of early childhood. Studies have shown that most infants learn to “sleep through the night” during the first year of life (2,5-7).

However, surveys indicate that 20-30% of young children suffer from night-waking problems (16-20). Using actigraphy (activity-based monitoring) it has been shown that, on average, normal infants wake up about twice per night but they usually quickly soothe themselves back to sleep with or without parental intervention (21). In contrast, sleep-disturbed infants wake up about 4 times per night and usually require significant parental help to resume sleep. The maturational trend of sleep consolidation continues throughout early childhood and some of these night-waking problems may be spontaneously resolved. Surveys based on parental reports usually find that these problems improve with age. For instance, in a study of 2,889 Italian infants and children Ottaviano et al. found that the prevalence of night-waking problems decreases from a range above 34% during the first two years of life to 13.4% in 4-6 year olds (22). However, recent studies suggest that night-waking problems are persistent and may turn into a chronic sleep problem if not treated (23-28).

The prevalence of night-waking problems in older children and adolescents is still unclear. Although surveys suggest that the problem is less frequent compared to that of early childhood (26,29,30) there is data to suggest that subjective reports underestimate this phenomenon in older children since they are less likely to require parental intervention (31). A recent survey of sleep problems in school-age children reported night-waking problems in 6.5% of the children (29); In a second survey of preadolescents “poor sleep” was reported in 14% of the children (32). In an EEG study, it was found that

children between 6-11 years of age had an average of 1-3 brief night-wakings (33). Unfortunately there are no systematic naturalistic studies of night-waking problems using objective measures in older children. A recent naturalistic study that used actigraphic monitoring to assess sleep in normal school-aged children found that they woke up more than twice per night on average, and that 18% of these children could be characterized as “poor sleepers” based on their fragmented sleep patterns (34).

II. Sleep Onset and Sleep Duration

Another conspicuous maturational process is related to significant reduction in sleep duration across development. The most dramatic changes occur during the first 3 years of life, but the slow monotonic decrease in sleep duration continues through adolescence. From an average of 16 hours of sleep per day as a newborn, to an average of 7-8 hours as a young adult, the ratio between sleep and wake hours is virtually reversed. However, these age-group means do not reflect the entire picture. The individual differences in sleep duration or sleep needs are striking and preclude simple answers to questions such as how much sleep is appropriate for a child at a certain age. For example, it has been shown that in newborns, during their first 48-hours in the nursery, sleep duration ranges between 10 hours and 22 hours per day (3).

Another way to look at this issue is to address the duration of sleep during nighttime hours. It has been reported that by 1 year of age the longest sleep period reaches 8 to 9 hours (6). The longest sleep period grows to about 10 hours per night at 5 years of age and remains quite stable until puberty, at which point it continues to decline. During this 5-10 year period, although there is relative stability in the nighttime sleep duration there is a decline in the overall 24-hour sleep period, which is mainly due to the decrease in daytime napping.

The issues of sleep onset time and sleep onset difficulties are among the factors determining sleep duration. There are conflicting reports with regard to what maturation

does to sleep latency during early and middle childhood. One study reported a trend of a gradual increase in the time taken to fall asleep with age (35). Another study reported a steady significant decrease in sleep latency from early infancy to 6 years of age (22).

When sleep latency over 30 minutes long was considered as the criterion the percentages of children meeting this criterion dropped from 13.7% during the first few months of age to 2.2% within the age range of 4-6 years. It has been estimated that 10 to 15% of children 1 to 8 years of age have difficulties going to bed or falling asleep (16,26, 36).

For most children, child-care, school or parental requirements and demands determine morning rise time. In contrast, sleep onset time is much more negotiable and it is determined by the child's physiologic needs and by multiple psychosocial factors of the child and his or her family. Therefore, The maturational trend of reduced sleep duration is primarily determined by the tendency to delay sleep onset. This delay of sleep onset could result from biobehavioral factors associated with the sleep-phase shift or from psychosocial factors ranging from separation difficulties and fears in early childhood to social incentives and academic and work demands in later development (e.g., interaction with parents, siblings or peers, TV, Internet, work, etc.).

From a psychosocial perspective it is clear that at around 2 to 3 years of age the child is becoming increasingly aware of and involved in the social life of the family. Sleep rituals are extended and settling problems become more prevalent. Between 3-5 years nighttime fears and nightmares also increase in prevalence and may complicate going to bed which is associated with darkness and separation from the parents and family social life (20,38). The pervasive belief is that children's fears subside when they reach school age (the "latency period"). However, Kahn and colleagues reported nighttime fears in as much as 15% of their large sample of preadolescents (32). These fears were associated with difficulties in initiating sleep (i.e., increased sleep latency). In this age group, the bedtime struggles are often associated with the evolving tendency of the child to delay sleep and with parental difficulties in limit setting.

In adolescence, the delay of the sleep-phase appears to accelerate. Carskadon and colleagues have demonstrated that the phase shift (a tendency to delay sleep onset and morning rise time) that characterizes adolescence is associated with the pubertal status of the youngster (38,39). They suggested that this is a biobehavioral process that is not only influenced by the psychosocial demands of adolescence but also driven by underlying biological mechanisms.

Furthermore, Carskadon and others have convincingly demonstrated that the continued reduction of sleep time in adolescence, which in this age group results mainly from delayed sleep onset leading to chronic sleep debt and an accompanied increase in daytime sleepiness (40-43). Surveys indicate a rising prevalence of direct subjective complaints of fatigue and daytime sleepiness during adolescence (40-45). In a recent study as much as 63% of the teenagers reported a need for more sleep during weekdays (45). In addition to the subjective reports, compelling evidence comes from studies that have shown that during school days adolescents sleep significantly less than on free days (42-47). It appears that school start time plays a major role in restricting sleep due to the delayed sleep onset (42,47). In a recent study, Carskadon and colleagues investigated the transition from 9th to 10th grade which led to an earlier school start-time (47). The earlier school start-time was associated with earlier rise time. No change occurred in sleep onset time and, consequently, the adolescents slept less. This transition was associated with delayed dim-light saliva melatonin onset phase, shorter sleep latencies on the Multiple Sleep Latency Test (MSLT) and intrusions of REM sleep episodes into the MSLT. The results of this study suggest that earlier school start-time with the resultant earlier rise time are associated with increased signs of sleep deprivation and daytime sleepiness. Thus, it appears that the only way adolescents can compensate for their accumulated sleep debt during school days is by increasing their sleep duration on non-school nights. Another line of evidence for the direct manifestation of this cumulative sleep debt is the growing daytime sleepiness that characterizes adolescents as documented objectively by the MSLT. It has been shown that adolescents' increased sleepiness is similar to the clinical level of sleepiness presented by patients with severe sleep disorders (40,41).

The normal sleep-phase delay that occurs in adolescents can turn to an exaggerated and dysfunctional pattern that is clinically labeled as the Delayed Sleep-Phase Syndrome. This syndrome is characterized by an inability to fall asleep before the very late hours of the night (or the early hours of the morning) with the associated inability to rise for normal activities at a reasonable morning hour (48). It has been shown that the emergence of this syndrome is often associated with the adolescent period (49,50).

III. Sleep State Organization

The maturational changes in sleep structure are subtler than those discussed thus far and require special observation or recording methods for detection. Interestingly, REM sleep was discovered during infant observation and was the breakthrough finding that led to the establishment of the field of sleep research. This finding is not surprising since among the unique characteristics of infant sleep are the facts that: (a) young infants either fall asleep directly with the onset of REM sleep or with a very short REM latency compared to older children or adults; and (b) infants spend a high proportion of their sleep time in REM sleep. These two unique features are the ones that undergo the most prominent maturational processes (see also Chapter 1- Scher's chapter).

Roffwarg, Muzio and Dement (51) published the earliest systematic scientific inquiry of the ontogeny of sleep structure in 1966. Since then, a limited number of polysomnographic studies have contributed to our knowledge on the normal maturation of sleep organization in children and adolescents (33,52-61). As indicated by Kahn and colleagues (52) the methods used by different laboratories differ in many aspects and, therefore, the results do not always overlap. Nevertheless, consistent maturational processes have been identified and are reported in the following subsections.

REM-NREM Sleep Distribution

During their earlier months of life infants spend around 50% of their sleep time in REM sleep (almost 8 hours per day). By the time they are 1-year-old they spend only

around 30% of their sleep time in REM sleep and this proportion only slightly decreases during the next few years to the adult proportion of 20-25%. Indeed, the major component of the outstanding decrease in sleep duration from 16-hours in the newborn period to 7-8 hours in adults is the reduction in REM sleep. As stated earlier, newborns experience an immediate onset of REM sleep when they fall asleep but this tendency changes quickly during the first few months as quiet sleep becomes more dominant during the early phases of sleep. There is scientific support to the hypothesis that REM sleep is so predominant in infancy because it facilitates information processing and brain maturation of the human infant who is born with a small brain in comparison to its mature brain size (51,62). It is important to note that during the early childhood period (ages 2-5 years) most children nap during the day and their state distribution might be affected by their napping sleep structure (51,53-56). For instance, it has been suggested the lower amount of NREM sleep in infants in comparison to older children results from the fact that they spend much more time in NREM sleep during their naps.

Dahl and colleagues have reported a significant association between REM sleep parameters and reproductive hormones in school-age children during their pubertal development (57). Higher levels of reproductive hormones were associated with shorter REM latency, lower REM activity and lower REM density.

In addition to the maturational changes in REM-NREM sleep, the distribution of the NREM sleep stages also undergoes significant developmental process. The differentiation of slow-wave sleep stages 3-4 becomes possible during the second half of the first year of life (52). During the school years stage 4 NREM sleep appears to decrease from 18% in 6- to 7-year-olds to 14% in 11-year-olds. This reduction is accompanied by an increase in NREM stage 2 (33). Similar findings have recently been reported by Acebo et al (61). During adolescence it has been reported that slow-wave sleep decreases in a linear progression across the Tanner stages (52). Bes and colleagues (60) have compared the distribution of slow-wave sleep (SWS) across the night in infants (aged between 20 weeks and 1 year), children (1-6 years of age) and adults (20-36 years). They found that SWS reached its peak in all age groups during the first NREM sleep

episode. Following the first NREM episode, SWS percent decreased across the night in the children and the adults but not in the infants. Another maturational tendency observed in this study was the transition from a recurrence of SWS in *alternate* REM-NREM cycles in infants to a recurrence of SWS on *consecutive* REM-NREM cycles.

REM-NREM Cycle

The REM-NREM state cycle also undergoes maturational changes. Since different studies use different criteria the reports are somewhat discrepant although the trends are similar (52). In newborns and very young infants the cycle lasts approximately 40-60 minutes. In one study it has been reported that by two years of age the cycle increases to 75 minutes and continues to increase to an average of 84 min in 5-year-olds. Another study reported an increase of the cycle from 40 minutes at 2 years of age to 60 minutes at 5 years of age (53). The length of the REM-NREM cycle continues to gradually increase until it reaches the 90-100 minute adult-like cycle during adolescence. Another important aspect that should be noted is that in early infancy, the EEG distinction between REM and NREM sleep is not as sharp as in older children and adults. This is demonstrated by the relatively large proportion of sleep scored as “indeterminate” or “transitional” in infancy. Bes and colleagues have shown that when REM recurrence times are considered in the assessment of the sleep cycle there is a clear peak of recurrence time in infants, children and adults (60). This peak recurrence time increases from about 50 minutes in infants to almost 100 minutes in adults. However, when SWS recurrence is considered there is no clear peak in infants or children, and only in adults there is a peak recurrence time of about 100 minutes. This pattern appears to result from the fact that infants and children often skip SWS in their NREM sleep episodes.

IV. Factors Influencing Children’s Sleep

The literature on the multiple factors influencing children’s sleep is quite extensive and I can only highlight some of the solid findings and directions emanating from this research field. For some phenomena the cause and effect between sleep and associated child or

environmental characteristics is unclear. For convenience purposes, I'll divide these factors into medical and psychosocial factors, although such clear distinction does not always exist, particularly in children.

Medical Factors

Sleep is very sensitive to the medical and physiological status of the child. Any common flu or congestion of the upper airways could lead directly to severe disruptions of sleep. Other, more serious and chronic common medical problems of infancy and childhood have been associated with poor sleep. For instance, in early childhood, allergy to cow milk (63), an esophageal reflux (64,65), Colic (66), atopic dermatitis (67,68) otitis media (69), headaches (70) and neurological disorders (69), are among the conditions that may exert negative effects on sleep. Asthma is another very common childhood disorder that has been repeatedly associated with poor sleep and increased sleepiness (71-73). Furthermore, blindness (74-77) and pervasive developmental disorders and other neurological impairments have also been associated with severe sleep disorders in children (78).

Psychosocial Factors

The early environment of the child appears to have a significant influence on the child's developing sleep-wake patterns. During infancy and early childhood, parental personality and psychopathology (particularly maternal), interaction style and bedtime practices have been associated with infants' and toddlers' sleep patterns and sleep disruptions (see 20, 79-82 for recent reviews). A comprehensive review of parental and cultural influences on children sleep is presented in chapter 6 (McKenna's chapter).

Children's sleep is also very vulnerable to psychosocial stressors (see 83,84 for reviews). There is a growing body of literature suggesting that children's sleep is directly affected by a variety of stressors ranging from minor stressors - sleeping in a sleep laboratory, also known as "the first night effect" - to extreme ones such as trauma and abuse.

A review of the literature suggests that there are two modes by which children adapt to stressors: one mode has been defined as the “turn on” response and the other, the “shut off” response. The “turn on” response is observed usually under circumstances of acute, external stress or threat that lead infants and children to become hyperalert and vigilant, a physiological state that is incompatible with extended consolidated sleep. This response is manifested in difficulty initiating and maintaining sleep (e.g., inability to relax and fall asleep, multiple night-wakings). The “shut off” response is usually seen under conditions of chronic and/or uncontrollable stress. Under these circumstances the child often appears to turn away from the stressful environment and spends more time in sleep, which is deeper and characterized by elevated arousal threshold.

Sleep is closely linked to the child’s emotional status and psychopathology. The cause-and-effect relationship between these phenomena is quite complex since sleep characteristics can modulate the emotional regulation of the child and vice versa (85). The relationship between sleep and psychopathology is discussed in chapter 19 (Wolfson’s chapter).

Gender Differences

The issue of gender differences in children’s sleep and sleep maturation is still a very confusing research topic due to many conflicting and inconsistent reports in the literature. It is beyond the scope of this chapter to extensively review this topic and only a few examples based on objective measures of sleep will be presented here.

An actigraphic sleep-wake study of more than 200 newborns during their first 48 hours of life did not reveal any significant gender differences (3). A study of 9-24 month-old infants using the same methodology revealed significant gender differences with boys presenting higher proportion of active sleep than girls (21).

An earlier EEG study of children, 6-12 years of age, reported a different gender difference: boys 10 years of age and older had higher percentage of stage 3 sleep and a higher delta ratio compared to girls (33). Carskadon and colleagues

have reported higher absolute amounts and percentages of SWS in boys than in girls (58).

Based on a longitudinal EEG study of children during their pubertal development, Dahl and colleagues found significant gender differences (57). Girls spent less time in REM sleep, had lower REM activity and lower REM density. The authors also reported a significant age-by-sex interaction for bedtime and sleep onset time. Interestingly, Acebo and colleagues (61) also reported age-by-sex interactions for SWS measures that suggest that there may be distinct maturational trajectories for boys and girls.

In a naturalistic actigraphic study of school-age children (34) significant gender differences were found on measures of true sleep time (excluding all wakefulness after sleep onset time) and percent of quiet sleep (motionless sleep). In a recent study of adolescent sleep using actigraphic measures it was found that girls spent more time than boys in sleep during school and non-school nights. However, in a laboratory study of children, 9.7-16.8 years of age, it was found that boys spent more time in bed than girls (61).

It is difficult to depict a clear gender-related picture from the pattern of results described above. It appears that gender differences vary with age and thus the differences appear or disappear in various studies. It is also possible that some findings on gender differences are related to psychosocial factors associated with the methods of the study. For instance, it is possible that girls and boys have a different emotional reaction to the issues associated with coming and sleeping in a sleep laboratory. Such possible differential emotional reactions may lead to variations found on the sleep measures. A close inspection of the differential “first-night effect” seen in boys and girls in the data reported by Carskadon et al (58) lend some support to this hypothesis.

V. Maturation of Sleep and Cognitive Function in Children

Beyond the complex relationships between sleep and emotional regulation and psychopathology in children, sleep is also closely associated with cognitive functioning, learning and attention. The sleep-wake system is a biobehavioral system that lends itself to a scientific investigation from the earliest stages of life and the principles of its organization, which are well-documented, may shed light on other brain systems that their maturation is behaviorally manifested only in later phases of development. Therefore, it appears that it is particularly important to understand the relationships between the developing sleep-wake systems and the maturation of other neurobehavioral systems.

Insufficient sleep and sleep disruptions have been associated with compromised neurobehavioral functioning in numerous studies in adults. It is surprising how limited the work on this topic in children where the maturation of sleep parallels and is interrelated to the maturation of brain systems responsible for information processing, response inhibition and modulation, attention, and regulation of motivational systems. A number of studies have associated infant sleep-wake patterns with neurobehavioral development (86-90). For instance, Scher et al., demonstrated that EEG sleep measures of newborns can predict mental and motor maturation as measured by Bayley mental scores at 12 and 24 months of age (90). Lower Bayley mental scores were associated with higher spectral EEG correlations, lower spectral EEG energies in the beta frequency ranges, fewer arousals per minute, lower rapid eye movements per minute, and shorter sleep latencies from awake state to active sleep. Due to the small number of children studied in these “prediction” studies it is difficult to conclude if the early organization of the sleep-wake system is indeed a predictor of later neurobehavioral organization. Nevertheless these studies suggest that this is an important area for future systematic investigation. Additional support comes from other studies in infants that have demonstrated a concomitant relationship between inadequate sleep and short attention span or attention problems in infants (91,92).

In older children, severe sleep disruption has been associated with attention and cognitive problems (93-96). Sleep disruptions have often been implicated in Attention Deficit

Hyperactivity Disorder (ADHD) and it has been repeatedly suggested that ADHD-like symptoms could result from insufficient or disordered sleep and the resultant decrease in arousal level (see 97; for review). Recent studies have shown that inattention and other ADHD-like symptoms are among the common correlates of sleep-related problems like sleep apnea (95); periodic leg movements (96) and snoring (94). In a recent intervention study it was demonstrated that children suffering from sleep-disordered breathing improve their academic achievements following surgical intervention of tonsillectomy and adenoidectomy (98). In normal children, early school start time and shortened sleep duration correlated with subjective complaints of sleepiness and attention problems at school (43). Similarly, in adolescents, irregular sleep patterns, delayed sleep onset and shorter sleep time have been associated with poor academic performance (42).

In a recent study of school-age children, based on an actigraphic assessment of sleep patterns and computerized neuropsychological tests, Sadeh and colleagues reported a significant correlation between fragmented sleep and poor performance on specific attention and learning tasks (99). Furthermore, a subsequent study demonstrated that extending or restricting sleep of school-age children by 30-60 minutes for 3 consecutive nights have direct positive or negative effects (respectively) on their neurobehavioral functioning (99).

Finally, Randazzo et al., studied the effects of acute sleep restriction on cognitive functioning in 16 children 10-14 years of age (100). The sleep-restricted children had shorter sleep latencies the following morning in comparison to the non-restricted children. Adverse effects of the sleep restriction were found on the cognitive measures of verbal creativity (i.e., fluency, flexibility) and the Wisconsin Card Sorting Test. No effects were found on less-complex cognitive functions. The authors concluded that higher cognitive functions in children are vulnerable to sleep restriction of one night whereas other cognitive functions can be maintained with no detected impairments. In sum, it appears that the close ties between the evolving sleep-wake system and the neurobehavioral maturation of the child gradually unfold and suggest that inappropriate or disturbed sleep patterns may adversely affect the maturing brain and the concurrent

neurobehavioral functioning of the child. These findings reemphasize the critical role of sleep in child development.

VI. Summary and Conclusions

The maturation of sleep-wake patterns during child development consists of a complex dynamic of change and balance. While sleep time dramatically decreases with age, the relationships between various components of sleep also change to meet what seems to be the changing needs of the maturing brain. These maturational processes appear to be influenced by multiple biological and psychosocial factors. Specific sleep disorders appear to be age-specific or at least peak in prevalence concomitantly with age-specific maturational trends (e.g., night-wakings in infancy or phase-delay in adolescents).

Because the maturation of the sleep-wake system is an ongoing process and, at specific periods, a very rapid and dramatic phenomena, the knowledge of normal sleep development is essential to any evaluation and understanding of clinical phenomena.

The maturation of the sleep-wake system exerts significant influences on the psychosocial and neurobehavioral functioning of the child. Thus, the early identification and treatment of childhood sleep disorders or inappropriate sleep patterns is essential for the child's well-being.

Finally, although the focus of this chapter was not on methodological issues, it is important to note that the representation of many of the maturational and clinical sleep phenomena depends on the instruments used to measure the phenomena (82,101,102). For instance, it has been demonstrated that parents are reliable informants of their infant sleep schedule, but do poorly when sleep quality measures are involved (i.e., night-wakings; 103). It is, therefore, important that the evaluation of information on sleep phenomena include critical consideration of the appropriateness of the instruments used to obtain this information.

References

1. Hoppenbrouwers T. Sleep in infants. In Guilleminault C, ed. *Sleep and Its Disorders in Children*. New York: Raven Press, 1987:1-15.
2. Coons S. Development of sleep and wakefulness during the first 6 months of life. In Guilleminault C, ed. *Sleep and Its Disorders in Children*. New York: Raven Press, 1987:17-27.
3. Sadeh A, Dark I, Vohr BR. Newborns' sleep-wake patterns: The role of maternal, delivery and infant factors. *Early Hum Dev* 1996; 44:113-126.
4. Freudigman KA, Thoman E. Ultradian and diurnal cyclicity in the sleep states of newborn infants during the first two postnatal days. *Early Hum Dev* 1994; 30:67-80.
5. Anders TF, Halpern LF, Hua J. Sleeping through the night: a developmental perspective. *Pediatrics* 1992; 90:554-560.
6. Anders TF, Keener M. Developmental course of nighttime sleep-wake patterns in full-term and premature infants during the first year of life. *Sleep* 1985; 8:173-192.
7. Bernal J. Night waking in infants during the first fourteen months. *Dev Med Child Neurol* 1973;15:760-769.
8. Kennaway DJ, Stamp GE, Goble FC. Development of melatonin production in infants and the impact of prematurity. *J Clin Endocrinol Metab* 1992; 75:367-369.
9. Sadeh A. Melatonin and sleep in infants: a preliminary study. *Sleep* 1997; 20:185-191.
10. Cavallo A. The pineal gland and human beings: relevance to pediatrics. *J Pediatr* 1993; 123:843-851.
11. Tzischinsky O, Shlitzer A, Lavie P. The association between the nocturnal sleep gate and nocturnal onset of urinary 6-sulfatoxymelatonin. *J Biol Rhythms* 1993; 8:199-209.
12. Espezel H, Jan JE, O'Donnell ME, Milner R. The use of melatonin to treat sleep-wake-rhythm disorders in children who are visually impaired. *J Visual Impairment & Blindness* 1996; 90:43-50.

13. Jan GE, Espezel H, Appleton RE. The treatment of sleep disorders with melatonin. *Dev Med Child Neurol* 1994; 36:97-107.
14. Camfield P, Gordon K, Dooley J, Camfield C. Melatonin appears ineffective in children with intellectual deficits and fragmented sleep: Six "N of 1" trials. *J Child Neurol* 1996; 11: 341-343.
15. Weissbluth, M. Naps in children: 6 months-7 years. *Sleep* 1995; 18:82-87.
16. Jenkins S, Bax M, Hart H. Behavior problems in preschool children. *J Child Psychol Psychiatry* 1980; 21:5-17.
17. Johnson M. Infant and toddler sleep: A telephone survey of parents in one community. *J Dev Behav Pediatr* 1991; 12:108-14.
18. Moore T, Ucko L. Night waking in early infancy, Part 1. *Arch Dis Child* 1957; 32:333-42.
19. Richman N. Surveys of sleep disorders in children in a general population. In Guilleminault C, ed. *Sleep and Its Disorders in Children*. New York: Raven Press, 1987:115-27.
20. Sadeh A, Anders TF. Infant sleep problems: origins, assessment, intervention. *Infant Mental Health J* 1993; 14:17-34.
21. Sadeh A, Lavie P, Scher A, Tirosh E, Epstein R. Actigraphic home monitoring of sleep-disturbed and control infants and young children: A new method for pediatric assessment of sleep-wake patterns. *Pediatrics* 1991; 87: 494-499.
22. Ottaviano S, Giannotti F, Cortesi F, Bruni O, Ottaviano C. Sleep characteristics in healthy children from birth to 6 years of age in the urban area of Rome. *Sleep* 1996; 19;1-3.
23. Hauri P, Olmstead E. Childhood-onset insomnia. *Sleep* 1980; 3:59-65.
24. Kateria S, Swanson M, Trevarthin G. Persistence of sleep disturbances in preschool children. *J Pediatr* 1987; 110:642-6.
25. Monroe L. Psychological and physiological differences between good and poor sleepers. *J Abnorm Psychol* 1967; 72:255-64.

26. Richman N, Stevenson J, Graham P. Preschool To School: A Behavioral Study. London: Academic Press, 1982.
27. Salzarulo P, Chevalier A. Sleep problems in children and their relationships with early disturbances of the waking-sleeping rhythms. *Sleep* 1983; 6:47-51.
28. Zuckerman B, Stevenson J, Baily V. Sleep problems in early childhood: Predictive factors and behavior correlates. *Pediatrics* 1987; 80:664-671.
29. Blader JC, Koplewicz HC, Abikoff H, Foley C. Sleep problems of elementary school children: a community survey. *Arch Pediatr Adolesc Med* 1997; 151:473-480.
30. Rona RJ, Gulliford MC, Chinn S. Disturbed sleep: effects of sociocultural factors and illness. *Arch Dis Child* 1998; 78:20-25.
31. Anders TF, Carskadon MA, Dement WC, Harvey K. Sleep habits of children and the identification of pathologically sleepy children. *Child Psychiatry Hum Dev* 1978; 9:56-62.
32. Kahn A, Van de Merckt C, Rebuffat E, Mozin MJ, Sottiaux M, Blum D, Hennart P. Sleep problems in healthy preadolescents. *Pediatrics* 1989; 84:542-546.
33. Coble PA, Kupfer D, Reynolds CF, Houck P. EEG sleep of healthy children 6 to 12 years of age. In Guilleminault C, ed. *Sleep and Its Disorders in Children*, New York: Raven Press, 1987:29-41.
34. Gruber R, Sadeh A, Raviv A. Sleep of school-age children: objective and subjective measures. *Sleep Res* 1997; 26:158.
35. Beltramini A, Hertzig M. Sleep and bedtime behavior in preschool-aged children. *Pediatrics* 1983; 71:153-158.
36. Richman N. A community survey of characteristics of one- to two-year-olds with sleep disruptions. *J Am Acad Child Psychiatry* 1981; 20:281-291.
37. Terr L. Nightmares in children. In Guilleminault C, ed. *Sleep and Its Disorders in Children*, New York: Raven Press, 1987:231-242.
38. Carskadon MA, Viera C, Acebo C. Association between puberty and delayed phase preference. *Sleep* 1993; 16:258- 262.

39. Carskadon MA, Acebo C, Richardson GS, Tate BA, & Seifer R. Long-nights protocol: access to circadian parameters in adolescents. *J Biol Rhythms* 1997; 12:278-289.
40. Carskadon MA. Patterns of sleep and sleepiness in adolescents. *Pediatrician* 1990; 17:5-12.
41. Carskadon MA, Dement WC. Sleepiness in the normal adolescent. In Guilleminault C, ed. *Sleep and Its Disorders in Children*, New York:Raven Press, 1987:53-66.
42. Wolfson A, Carskadon M. Sleep schedules and daytime functioning in adolescents. *Child Dev* 1998; 69:875-887.
43. Epstein R, Chillag N, Lavie P. Starting times of school: effects on daytime functioning of fifth-grade children in Israel. *Sleep* 1988;21:250-256.
44. Tynjala J, Kannas L, Levalahti E. Perceived tiredness among adolescents and its association with sleep habits and use of psychoactive substances. *J Sleep Res* 1997; 6:189-198.
45. Mercer PW, Merritt, SL, Cowell JM. Differences in sleep need among adolescents. *J Adolesc Health* 1998; 23:259-63.
46. Manbar R, Bootzin RR, Acebo C, Carskadon MA. The effects of regularizing sleep-wake schedule on daytime sleepiness. *Sleep* 1996; 19:432-441.
47. Carskadon MA, Wolfson AR, Acebo C, Tzischinsky O, Seifer R. Adolescent sleep patterns, circadian timing, and sleepiness at a transition to early school days. *Sleep* 1998; 21:871-881.
48. Weitzman ED, Czeisler CA, Coleman RM, Spielman AJ, Zimmerman JC, Dement WC. Delayed sleep phase syndrome, a chronobiological disorder with sleep-onset insomnia. *Arch Gen Psychiatry* 1981; 38:737-746.
49. Alvarez B, Dahlitz MJ, Vignau J, Parkes JD. The delayed sleep phase syndrome: clinical and investigative findings in 14 subjects. *J Neurol Neurosurg Psychiatry* 1992; 55:665-670.

50. Thorpy MJ, Korman E, Spielman AJ, Glovinsky PB. Delayed sleep phase syndrome in adolescents. *J Adolesc Health Care* 1988; 9:222-227.
51. Roffwarg H, Muzio J, Dement W. Ontogenetic development of the human sleep-dream cycle. *Science* 1966; 152:604.
52. Kahn A, Dan B, Groswasser J, Franco P, Sottiaux M. Normal sleep architecture in infants and children. *J Clin Neuropsychol* 1996; 13:184-197.
53. Kahn E, Fisher C, Edwards A. Twenty-four hour sleep patterns: comparison between 2- to 3-year old and 4- to 6-year-old children. *Arch Gen Psychiatry* 1973; 29:380.
54. Maron L, Rechtschaffen A, Wolpert E. Sleep cycle during napping. *Arch Gen Psychiatry* 1996; 11:503.
55. Webb W, Agnew H. Sleep cycling within 24-hour periods. *J Exp Psychol* 1967; 74:158.
56. Ross JJ. Sleep patterns in pre-adolescent children: an EEG-EOG study. *Pediatrics* 1968; 42:324-335.
57. Dahl R, Trubnick L, Al-Shabbout, Ryan N. Normal maturation of sleep: a longitudinal EEG study in children. *Sleep Res* 1997; 26:155.
58. Carskadon MA, Keenan S, Dement WC. Nighttime sleep and daytime sleep tendency in preadolescents. In Guilleminault C, ed. *Sleep and Its Disorders in Children*, New York:Raven Press, 1987:43-52.
59. Carskadon MA, Orav EJ, Dement WC. Evolution of sleep and daytime sleepiness in adolescents. In Guilleminault C, Lugaresi E, eds. *Sleep/Wake Disorders: Natural History, Epidemiology, and Long-Term Evaluation*. New York: Raven Press, 1983:201-216.
60. Bes F, Schulz H, Navelet Y, Salzarulo P. The distribution of slow-wave sleep across the night: a comparison for infants, children and adults. *Sleep* 1991; 14:5-12.

61. Acebo C, Millman RP, Rosenberg C, Cavallo A, Carskadon MA. Sleep, breathing, and cephalometrics in older children and young adults: part I – normative values. *Chest* 1996; 109:664-672.
62. Zapelin H. Mammalian sleep. In MH Kryger, T Roth and WC Dement Principles and Practice of Sleep Medicine, 2nd edition. London: WB Saunders Company, 1994:69-80.
63. Kahn A, Mozin M, Rebuffat E, Sottiaux M, Muller MF. Milk intolerance in children with persistent sleeplessness: A prospective double-blind crossover evaluation. *Pediatrics* 1989; 84:595-603.
64. Kahn A, Rebuffat E, Sottiaux M, Dufour D, Cadranel S, Reiterer F. Arousals induced by proximal esophageal reflux in infants. *Sleep* 1991; 14:39-42.
65. Ghaem M, Armstrong KL, Trocki O, Cleghorn GJ, Patrick MK, Shepherd RW. The sleep patterns of infants and young children with gastro-oesophageal reflux. *J Paediatr Child Health* 1998; 34:160-163.
66. Weissbluth M. Colic. In Ferber R, Kryger M, eds. Principles and Practice of Sleep Medicine in the Child. Philadelphia: W.B. Saunders Company, 1995:75-78.
67. Reid P, Lewisjones MS. Sleep difficulties and their management in preschoolers with atopic eczema. *Clin Exp Dermatol* 1995; 20:38-41.
68. Stores G, Burrows A, Crawford C. Physiological sleep disturbances in children with atopic dermatitis: a case control study. *Pediatr Dermatol* 1998; 15:264-268.
69. Sheldon HS, Spire JP, Levy HB. Pediatrics Sleep Medicine. Philadelphia: Saunders Company, 1992.
70. Bruni O, Favrizi P, Ottaviano S, Cortesi F, Giannotti F, Guidetti V. Prevalence of sleep disorders in childhood and adolescence with headache: a case-control study. *Cephalalgia* 1997; 17:492-498.
71. Madge PJ, Nisbet L, McColl JH, Vallance A, Paton JY, Beattie JO. Home nebuliser use in children with asthma in two Scottish Health Board Areas. *Scott Med J* 1995; 40:141-143.

72. Kales A, Kales JD, Sly RM, Scharf MB, Tand TL, Preston TA. Sleep patterns of asthmatic children: all night EEG studies. *J Allergy* 1970; 46:300-308.
73. Sadeh A, Horowitz I, Wolach-Benodis L, Wolach B. Sleep and pulmonary function in children with well-controlled, stable asthma. *Sleep* 1998; 21:379-384.
74. Okawa M, Nanami T, Wada S, Shimizu T, Hishikawa Y, Sasa H, Nagamine H, Takahashi K. Four congenitally blind children with circadian sleep-wake rhythm disorder. *Sleep* 1987; 10:101-110.
75. Sadeh A, Klitzke M, Anders T F, Acebo C. Sleep and aggressive behavior in a blind retarded adolescent: a concomitant schedule disorder? *J Am Acad Child Adolesc Psychiatry* 1995; 34:820-824.
76. Tzischinsky O, Skene D, Epstein R, Lavie P. Circadian rhythms in 6-sulfatoxymelatonin and nocturnal sleep in blind children. *Chronobiol Int* 1991; 8:168-175.
77. Palm L, Blennow G, Wetterberg L. Long-term melatonin treatment in blind children and young adults with circadian sleep-wake disturbances. *Dev Med Child Neurol* 1997; 39:319-325.
78. Okawa M, Sasaki H. Sleep disorders in mentally retarded and brain-impaired children. In Guilleminault C, ed. *Sleep and its Disorders in Children*. New York: Raven Press, 1987:269-290.
79. Mindell JA. Sleep disorders in children. *Health Psychol* 1993; 12:151-162.
80. Wolfson A. Sleeping patterns of children and adolescents: developmental trends, disruptions and adaptations. *Psychiatr Clin North Am* 1996; 5:685-700.
81. Anders TF, Eiben LA. Pediatric sleep disorders: A review of the past 10 years. *J Am Acad Child Adol Psychiatry* 1997; 36:9-20.
82. Sadeh A, Gruber R. Sleep Disorders. In Bellack AS, Hersen M, eds. *Comprehensive Clinical Psychology*. New York: Pergamon, 1998:629-653.
83. Sadeh A. Stress, trauma and sleep in children. *Psychiatr Clin North Am* 1996; 5:685-700.

84. Sadeh A, Gruber R. Stress and sleep in adolescence: A clinical-developmental perspective. In Carskadon MA, ed. Adolescent Sleep Patterns: Biological, Social, and Psychological Influences. New York: Cambridge University Press, in press.
85. Dahl RE. The regulation of sleep and arousal: development and psychopathology. *Dev Psychopathol* 1996; 8:3-27.
86. Thoman EB. Sleeping and waking in infants: A functional perspective. *Neurosci Biobehav Rev* 1989;14:93-107.
87. Thoman EB. Sleep and wake behaviors in the neonates: consistencies and consequences. *Merrill-Palmer Quart* 1975; 21:295-314.
88. Freudigman KA, Thoman E. Infant sleep during the first postnatal day: an opportunity for assessment of vulnerability. *Pediatrics* 1993; 92:373-379.
89. Thoman EB, Denenberg VH, Sievel J, Zeidner LP, Becker P. State organization in neonates: developmental inconsistency indicates risk for developmental dysfunction. *Neuropediatrics* 1981; 12:45-54.
90. Scher MS, Steppe DA, Banks DL. Prediction of lower developmental performances of healthy neonates by neonatal EEG-sleep measures. *Pediatr Neurol* 1996;14:137-144.
91. Sadeh A, Lavie P, Scher A. Maternal perceptions of temperament of sleep-disturbed toddlers. *Early Educ Dev* 1994;5:311-322.
92. Weissbluth M, Liu K. Sleep Patterns, attention span and infant temperament. *J Dev Behav Pediatr* 1983; 4:34-36.
93. Dahl RE. The impact of inadequate sleep on children's daytime cognitive function. *Semin Pediatr Neurol* 1996b; 3:44-50.
94. Chervin RD, Dillon JE, Bassetti C, Ganoczy DA, Pituch KJ. Symptoms of sleep disorders, inattention, and hyperactivity in children. *Sleep* 1997;20:1185-1192.
95. Hansen DE, Vandenberg B. Neuropsychological features and differential diagnosis of sleep apnea syndrome in children. *J Clin Child Psychol* 1997; 26:304-310.

96. Picchietti DL, Walters AS. Restless Legs Syndrome and Periodic Limb Movement Disorder in children and adolescents: comorbidity with Attention-Deficit Hyperactivity Disorder. *Psychiatr Clin North Am* 1996; 5:729-751.
97. Corkum P, Tannock R, Moldofsky H. Sleep disturbances in children with attention-deficit / hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 1998; 37:637-646.
98. Gozal D. Sleep-disordered breathing and school performance in children. *Pediatrics* 1998; 102:616-620.
99. Sadeh A, Raviv A, Gruber R. Sleep and neurobehavioral functioning in school children. Paper presented at the 14th Congress of the European Sleep Research Society, Madrid, September, 1998.
100. Randazzo AC, Muehlbach MJ, Schweitzer PK, Walsh JK. Cognitive function following acute sleep restriction in children ages 10-14. *Sleep* 1998; 15:861-868.
101. Thoman EB, Acebo C. Monitoring of sleep in neonate and young children. In Ferber R, Kryger M, eds. *Principles and Practice of Sleep Medicine in the Child*. Philadelphia: W.B. Saunders Company, 1995:55-68.
102. Ferber R. Clinical assessment of child and adolescent sleep disorders. *Psychiatr Clin North Am* 1996; 5:569-579.
103. Sadeh A. Evaluating Night-Wakings in Sleep-Disturbed Infants: A Methodological Study of Parental Reports and Actigraphy. *Sleep* 1996; 19:757-762.