Hypersomnia in Children

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KEYWORDS

• Hypersomnia • Children • Adolescents • Narcolepsy • Kleine-Levin syndrome • Hypocretin

KEY POINTS

- Further refinements are needed in the diagnostic testing of childhood daytime sleepiness.
- It is not known whether the multiple sleep latency test in childhood should use 4 or 5 naps.
- Longitudinal studies are needed to determine if treating sleepiness by medications corresponds
 with improved neuropsychological function. Given the small number of patients with childhood
 narcolepsy at each sleep center, a consortium-based approach is needed for gathering prospective
 high-quality evidence regarding optimum treatment measures, be they pharmacologic or nonpharmacologic in nature.

Daytime sleepiness is an important symptom of impaired health during childhood and adolescence. It is consequent to a set of diverse pathophysiologic circumstances. The initial manifestations are often underrecognized by parents, school authorities, and health professionals alike. The consequences of daytime sleepiness are significant, especially from the standpoint of its impact on mood, learning, behavior, and dexterity. The purpose of this article is to provide an overview of childhood daytime sleepiness, with an emphasis on clinical assessment and management. In areas where there is insufficient evidence in childhood, the author has extrapolated information from adult sleep literature.

HOW PREVALENT IS CHILDHOOD DAYTIME SLEEPINESS

Nevéus and colleagues³ conducted a questionnaire survey in 1413 Swedish children aged 6 to 10 years and found a 4% prevalence rate for daytime sleepiness. When Yang and colleagues⁴ conducted the validated School Sleep Habits Survey in a sample of 1457 Korean school children aged 9 to 19 years, they found that 6.6% of the respondents admitted to daytime sleepiness being a "very big problem." As seniority in school increased from the 5th to the 12th grade, so also did the prevalence of daytime sleepiness. The increase in prevalence of daytime sleepiness with advancing grade levels in children and adolescents is also supported by Ohayon and colleagues.⁵ Using a telephone survey, the investigators sampled 1125 French, British, German, and Italian adolescents aged 15 to 18 years.⁵ A prevalence rate of 19.9% was found for daytime sleepiness, with 11.9% of the sample complaining also of difficulty waking up in the morning. Although Yang and colleagues⁴ believe that sleepiness was slightly more prevalent in girls than in boys, gender differences in prevalence of childhood daytime sleepiness have not been definitively established. The key point established by survey instruments is that daytime sleepiness is a significant pediatric health problem, with prevalence ranging from 4% in preadolescents to almost 20% in high school seniors.^{3–5}

WHAT FACTORS PREDISPOSE TO CHILDHOOD DAYTIME SLEEPINESS

A convergence of multiple factors increases the vulnerability of teenagers to daytime sleepiness.

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Concurrent with maturation, children and adolescents show a tendency to sleep fewer hours at night. In a longitudinal study of 493 healthy children and adolescents, Iglowstein and colleagues⁶ found that the mean sleep duration in 10-year-old children was 9.9 hours (standard deviation [SD], 0.6), whereas the mean sleep duration in 16-year-old adolescents had decreased to around 8.1 hours (SD, 0.7). When this reduced total sleep time is juxtaposed with the need for teenagers to wake up between 5:30 AM and 6:30 AM to arrive at school by around 7:30 AM, the end result can be daytime sleepiness.

Dim-light melatonin onset (DLMO) is a physiologic marker for the time of sleep onset. DLMO shifts to a later time in the evening in older adolescents in comparison with preadolescents. In a study by Taylor and colleagues,7 the mean DLMO time was found to be 2033 hours (SD, 49 minutes) in 9 prepubertal children who were of Tanner stage I sexual development (mean age, 11.1 years), whereas the mean DLMO time had shifted to 2129 hours in 11 pubertally mature adolescents who were of Tanner stage V (mean age, 13.9 years). The resulting physiologic delay in sleep onset to a later time of the night predisposes to sleep deprivation, especially on school nights. Wolfson and Carskadon⁸ surveyed about 3000 high school students in New England using the School Sleep Habits Survey and found that students who self-reported higher grades reported more total sleep time and earlier bedtimes on school nights than children with lower grades (P<.001). Furthermore, early school start times are associated with decreased total sleep time, increased daytime sleepiness, and poorer school performance.9

The intrusion of technological devices, such as televisions, computers, cell phones, video games, and the Internet, into the bedroom tends to further postpone the sleep-onset time on school nights and leads to insufficient night sleep, with consequent daytime sleepiness. Sadeh and colleagues¹⁰ have evaluated the effect of relative sleep restriction by an average of 41 minutes on school children. The investigators found that even a very moderate but accumulated sleep deficit (eg, watching one more television show) can have adverse neurobehavioral effects, especially when it comes to executive functioning.

A disruption of key central wake-promoting mechanisms, as seen in narcolepsy-cataplexy, can also lead to excessive daytime sleepiness (EDS). Patients with narcolepsy-cataplexy lose hypocretin (orexin)-secreting neurons from the dorsolateral region of the hypothalamus. These hypocretin neurons have widespread projections to

the forebrain and brainstem. Hypocretin promotes alertness and locomotor activity. 11,12 Immunemediated or anatomic lesions of the hypothalamus and rostral midbrain (such as neoplasms, inflammation, or trauma) can be associated with hypersomnia by virtue of alterations in the balance between the sleep-enhancing and wakefulnesspromoting influences. Saper and colleagues¹³ have proposed the concept of autonomic regulation via a sleep-wake switch. The investigators postulate that the tuberomammillary nucleus (histaminergic in nature), the locus coeruleus (noradrenergic in nature), and the raphe system (serotonergic in nature) work together to enhance alertness. They have a reciprocal relationship with the sleep-promoting neurons of the ventrolateral preoptic (VLPO) nucleus, which are γ -aminergic in nature. Some cells in the VLPO (termed VLPO cluster) promote non-rapid eye movement sleep, whereas other cells (VLPO extended) facilitate rapid eye movement (REM) sleep. Hypocretin (orexin) serves to stabilize the relationship between these 2 sets of physiologically opposing influences.

WHAT ARE THE CONSEQUENCES OF DAYTIME SLEEPINESS

Similar to adults, adolescents with daytime sleepiness manifest changes in behavior and a decline in performance. 2,14 Conversely, the treatment of sleep disruption by improving sleep hygiene or treating specific sleep disorders results in improvements in daytime performance. 15 Ha and colleagues 16 performed objective computerized testing on 24 patients with narcolepsy (being treated with stimulants, mean age 30.7 ± 12.8 years, mean intelligence quotient of 119, 79% male) and 24 matched controls. The patients with narcolepsy performed more frequent omission and commission errors on a vigilance test and more omission errors on a continuous performance test.

Stores and colleagues¹⁷ studied psychosocial problems in children with narcolepsy (n = 42) and nonnarcolepsy hypersomnia (EDS, n = 18) along with 23 age-matched controls. The investigators observed that patients with both narcolepsy and EDS exhibited more behavioral difficulties than controls on the Strengths and Difficulties Questionnaire. Both groups (patients with narcolepsy and nonnarcolepsy hypersomnia) showed more depressed mood on the Child Depression Inventory and lower quality of life on the Child Health Questionnaire's mental health subscale when compared with controls. These findings suggest that deficits in mood, behavior, and quality of life were more related to hypersomnia than specifically to narcolepsy. Furthermore, patients

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