Physiological Evolution of Sleep

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Introduction

Education on sleep has become a priority for many communities, social services, medical institutions, and government and educational bodies. Parallel to this, sleep disorders have become an important problem for doctors and patients alike. A study of the prevalence of neurological problems in Europe ranked sleep disorders third in terms of prevalence and impact, after headaches and anxiety disorders. The study estimated that the costs secondary to sleep disorders were over US $35 billion in 2010—more than the costs associated with epilepsy and multiple sclerosis put together.

Children are particularly affected by respiratory disorders and alterations in normal sleep architecture. In itself, the total time spent sleeping does not correlate directly with academic performance. The

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physiological mechanisms underlying these associations remain unclear. It is thought that intermittent hypoxemia decreases brain blood flow to specific areas, and alterations in the architecture of sleep contribute significantly.

Given that there are no clear algorithms of study, doctors should use clinical tools to diagnose and treat patients with sleep disorders. There are few doctors specializing in sleep medicine or centers that can provide more in-depth studies. Consequently, there are long waiting lists for sleep studies in several countries. Waiting lists in the USA for polysomnography are 3–6 weeks long, while in Canada there are waiting lists of more than a year to get into a tertiary center capable of diagnosing sleep disorders with methods that are accepted as standard today. Consequently, it is critical that the treating physician—whether he or she is a neurologist, pediatrician, bronchopulmonary specialist, or child psychiatrist—is informed as to how to diagnose these very prevalent disorders, which leave many sequelae. In current pediatric practice, we need to know where patients with suspicion of sleep disorders should be referred to. Education on healthy and safe sleep should be an important theme in the training of pediatricians and doctors who work with children. Despite this evident need, studies show that education on sleep occupies less than 4.4 hours in total in the average training of a pediatric resident in the USA.

There is a high prevalence of sleep disorders, comparable to that of bronchial asthma. It is estimated that 12% of children experience daily problems associated with sleep, while 76% have occasional problems. Certain populations have a greater risk of developing sleep-related problems, such as children with Down syndrome, neuromuscular diseases, craniofacial alterations, epilepsy, intracranial tumors, and late development.

History and Anamnesis

The first step in diagnosing a possible sleep disorder is to study and understand the child in wakefulness. An example of this is the low yield of the symptom of snoring to predict obstructive sleep apnea in a child, in contrast to what happens in adults. Questions should be directed at academic performance, daytime sleepiness, naps, sleep routines and hygiene, eating habits, attentional conflicts, and hyperactivity. Children often present symptoms and problems of concentration, disruptive behavior, and lack of concentration in school and at home, rather than evident somnolence (unlike adults). There are numerous questionnaires that investigate these symptoms, such as the Pediatric Daytime Sleepiness Scale (PDSS), which is a tool with broad support for application by parents who have noted drowsiness and tiredness in their children.

The investigation into sleep hygiene and symptoms suggestive of insomnia should be presented in a directed way in an anamnesis. The use of televisions, radios, cell phones, computers, and various portable devices is associated with poor sleep and numerous sleep disorders. Special attention should be given to questions regarding the number of hours these devices are used for. The family context should also be explored; the working routines of the parents and the family composition themselves imply an implicit adaptation of the children's sleeping habits.

Age and Development

Given that sleep disorders in children can manifest themselves in a number of ways, search and screening tools should be adapted specifically to age groups and the type of sleep disorder being considered. The first step in developing these kinds of tools is to define what is normal at each age level. In the following sections, we summarize the main characteristics of sleep at different ages.

The Fetal Stage

It has been possible to obtain information about sleep at the early stages of life. Apart from what can be observed in premature newborns, noninvasive techniques have been used to study neurophysiological waves at the fetal stage to allow for the study of sleep in children before their birth.

Prechtl defined four stages of fetal sleep as measured by ultrasound:

1. Regular and slow heart rate, twitching, absence of eye movements
2. Irregular heart rate, eye movements, occasional body movement
3. Regular and rapid heart rate, eye movements, absence of body movements
4. Irregular and rapid heart rate, eye movements, continuous body movements

Premature Newborns

Premature newborns have more complex sleeping patterns in parallel with development. Active and quiet sleep can be distinguished between 33 and 34 weeks of gestational age, with greater reactivity evident around 35–36 weeks.

Movements, blinking, smiles, and nonspecific facial movements can be observed during active sleep. Jerking and twitching occur at the onset of sleep, which should not be interpreted as pathological. This sleep is not equivalent to REM in older children. While there are common phenomena, studies in newborn rats have found that lesions in the nuclei of the anterior raphe result in either the absence of rapid eye movement (REM) at the adult stage or no alteration, depending on the gestational age when the lesions occur. Given the above, it is unclear if REM sleep emerges subsequently as an independent stage per se. Non-REM (NREM) or slow-wave sleep is not present in newborns. It develops between the second and sixth weeks after birth, parallel to the appearance of greater brain voltages. Quiet sleep in newborns is equivalent to NREM.

The development of the sleep/wakefulness cycle in the brain includes the mesencephalic reticular formation, the posterior hypothalamus, and the medullary magnocellular nucleus. These structures are present at birth and, over the course of the first year of life, they mature concomitantly with their myelination, which begins with the posterior structures and proceeds anteriorly through the cerebral cortex.

There is evidence from animal models that somatostatin promotes the development of REM sleep. It is detectable in human at 120 days of gestation. This hormone reaches its maximum concentration at birth, whereupon it decreases progressively until it reaches 15–30% of its initial concentration in adulthood. However, because numerous neurotransmitters have been identified in different species of mammals, it is not clear which of these is responsible for the development and differentiation of sleep in humans. Among the neurotransmitters involved in the sleep/wakefulness cycle are glutamate, glycine, and γ-aminobutyric acid (GABA).

An electroencephalography (EEG) study of newborns should include at least 16 channels. The reading is highly complex and distinctly different from one for an adult, in terms of both voltage and EEG morphology. A premature patient can demonstrate periods of bursts of delta activity, followed by more than 25 μV of amplitude. This has classically been called an alternating trace pattern.

The *tracé discontinue* refers to a discontinuous pattern of alternating bursts of activity and intervals of apparent inactivity, which can be seen in premature newborns of less than 32 weeks of gestational age. This pattern has an amplitude of less than 25 μV and is transformed into quiet sleep at 36 weeks. At around 36 weeks the pattern termed "continuous slow-wave sleep" emerges, which predominates by 36 weeks (Table 5.1).

Bilateral synchrony has bimodal development; it is initially synchronic (until 30 weeks of gestational age), then becomes 25% asynchronic at up to 35 weeks, and then becomes synchronic again at 40 weeks. The symmetry is considered normal if the ratio of the interhemispheric amplitude is no greater than 2:1.

Newborns

There are many sleep-related situations in the neonatal period that can be stressful for parents or caregivers. The normal sleep patterns at this age should be explained to them, with screening to identify postpartum depression among mothers. One of the most common questions relates to the impression of inversion of the sleep–wakefulness cycle between night and day.

Table 5.1 Description of sleep patterns of newborns and premature newborns

Type of pattern	Description	State	Gestational age
Tracé alternant	Interburst intervals >25 μV	Alert	34–37 weeks
Tracé discontinue	Interburst intervals < 25 μV	Quiet	32–36 weeks
Slow-wave sleep	High-amplitude delta and theta waves	Quiet	36–45 weeks
Activité moyenne	Low voltage, irregular, continuous	Wakeful, active	>36 weeks
Sleep spindles	12–14 Hz, central	Quiet	44–49 weeks
Delta bursts	0.3–1.5 Hz, high-amplitude waves (50–250 μV), occipitotemporal, asynchronous	Quiet	29–33 weeks

At birth, babies have a very basic and initial circadian rhythm that is sensitive to changes in light in relation to melatonin secretion. Although parents may not note a significant difference in the distribution of hours of sleep between day and night, it has been demonstrated that there is a tendency from the first days of life for the baby to sleep more during night hours. It is estimated that term newborns sleep on average for 16 hours a day. Newborns present a polycyclic pattern that ranges between 1 and 4 hours, with the need for feeding between the cycles. The alternation between the sleep stages is shorter in newborns—on average, 50–60 minutes—in contrast to the 90-minute alternation in adults.

It is estimated that 50% of newborn sleep is REM and that REM sleep latency is shorter in newborns than in adults.

A sleep anamnesis of a newborn should include:

1. Gestational age and neonatal complications
2. Complications of the birth
3. Admissions to neonatological care
4. Presence of apnea or family history of any apparent life-threatening events (ALTEs; events that put the infant's life at risk, in the eyes of the observer) or sudden death syndrome
5. Environment for sleeping: where, how much, and how the baby sleeps; shared bed, exposure to smoking
6. Safe environment for sleeping: separate and strong crib, absence of stuffed animals or pillows, sleeping on her or his back, avoidance of soft surfaces to avoid the risk of suffocation
7. Sleeping position: the baby should sleep on his or her back; recommendations followed to prevent sudden death of the baby

Newborns with apnea represent a special and unique population. There are several risk factors that facilitate the development of apnea, one of which is premature birth. The gestational age marks the presence of "premature apnea," such that 7% have it at 34–35 weeks of gestational age, 15% at 32–33 weeks, 54% at 30–31 weeks, and almost 100% of newborns at less than 29 weeks. Although premature apnea is considered a self-limiting condition, it is treated to avoid possible neurocognitive consequences associated with desaturations. The underlying physiological mechanism of this phenomenon is not completely understood, and it is supposed that it is based on the immaturity of the respiratory center. In all cases of persistent apnea, the possibility of conditions such as hemorrhages, infections, intracranial lesions, gastroesophageal reflux, convulsions, and metabolic and electrolytic alterations should be considered.

One theory is that premature babies are particularly sensitive to inhibitory neurotransmitters such as GABA, adenosine, serotonin, and other prostaglandins. Adenosine is one of the key targets of treatment with methylxanthine. There is evidence that adenosine facilitates the release of GABA, leading to respiratory depression in the newborn. More recent studies have shown the presence of prostaglandins that induce the formation of proinflammatory mediators such as interleukin beta 1.

The experience of newborn admissions to neonatological care raises questions about the stressful effect that intensive services can have on sleep. The overstimulation of arousals and awakenings by sounds, and the attention related to continuous monitoring, raise concerns. There have been few studies on this issue, but simple interventions such as the use of music have been shown to reduce parental stress and improve newborn sleep stages.

Long-term follow-up has shown that newborns who have had efficient sleep are more attentive between 4 and 18 months of age than those who did not sleep efficiently at the newborn stage.

Infants

Infancy is a period of significant changes for the normal sleep pattern, and questions should be directed in particular to the following aspects:

1. Number and duration of naps: In total, these should not exceed 3–4 hours.
2. Total time spent sleeping: Nocturnal sleep around 9–10 hours, total time 12–13 hours.
3. Sleep routine and rituals: Incipient routines and rituals generally already exist at this age.
4. Presence of colic, diseases, and the use of medications.
5. Gastroesophageal reflux: This condition, which is often physiological, has been associated with an increase in arousals, although it is sometimes associated with apnea.
6. Details of any ALTEs.
7. Psychomotor development: Nonacquisition of the different stages in psychomotor development can involve concomitant problems related to sleep. This should be investigated to determine the presence of hypotony, which can affect the occurrence of hypoventilation.
8. Use of pacifiers and bottles: The American Academy of Pediatrics currently recommends the use of pacifiers to avoid sudden infant death.
9. Parents' attention and routines: In many cases, parents sleep with their infant as a result of sleep problems. Parents respond in different ways to their infant's crying and needs. Some actions can interrupt the infant's sleep, such as using a baby bottle as a pacifier, but can also help the baby go back to sleep once he or she has woken up.

At 3–4 months of age, the infant develops more consistent sleeping blocks, which can be a source of relief to the parents, who will say the baby now "sleeps the whole night through." During the first 6 months of life, melatonin secretion matures concomitantly, with a strong correlation between melatonin levels and sleep/wakefulness cycles. This correlation is not observed in children who have lost their vision. The aberrant sleep patterns of blind infants tend to improve with use of exogenous melatonin.

Nap taking begins to decrease at 6 months of age, and the majority of infants continue with two naps in the second semester of life. Most infants immediately begin with REM sleep or with significantly shorter REM latency than adults. The REM cycles get longer with the passing months. The time passed in REM sleep decreases progressively from 50% of the total sleep time in small infants to less than 30% as they reach the age of 1 year.

The response of infants to arousals matures rapidly in the first 9 months of life. The response to hypoxia with arousals decreases toward week 9, because of which responses to microawakenings gradually shift from subcortical to cortical responses.

One theory for infant sudden death is a failure to adapt an arousal response to particular stimuli or a toxic substance. It is especially important with infants to prepare guidelines to prevent sudden death: the baby should sleep on his or her back, the bed should not be shared, and exposure to cigarette smoke should be avoided. A low socioeconomic level and prematurity have also been identified as risk factors for infant sudden death. The reticular formation of the brain stem is involved in balancing inhibitory and excitatory impulses that collect sensorial, somatic, and chemical/mechanical receptor–sourced information. The progression of microawakenings in

response to stimuli, such as tactile stimuli and an increase in CO_2, have demonstrated an ascending spinal, subcortical, and cortical pattern. Waking begins with small subtle movements, following by swaying, and culminating in eye opening and full awakening, with the shift to wakefulness. These microawakenings occur in both active and quiet sleep.

Preschool Children and Older Infants (1–3 Years of Age)

The sleeping habits of small preschool children is biphasic, with sleep concentrated in the night and two naps during the day. The sleep/wakefulness cycle gradually consolidates, and by the age of 3 years the child usually has only one nap during the day. By the age of 6 years, naps have become abnormal and suggest a sleep-specific disorder. REM sleep decreases gradually at this stage, reaching 30% after the age of 1 year. By 9–11 years of age, the REM/NREM ratio is similar to that in adults. EEG spectral analysis can show the changes over time during this period.

Parallel to the decrease in REM sleep, the total sleeping time decreases from 16–18 hours to ≤9 hours at the postpuberty stage. The average number of hours of sleep at the preschool stage is 10 hours.

Between 20% and 30% of older infants and preschool children remain awake during the night, which constitutes a common reason for medical consultation. It is considered that intervention is appropriate for awakenings when the family routine is disrupted and the parents are forced to take actions such as staying with the child or forcing the child to return to his or her room. However, as actigraphy studies have shown, most preschool children wake up twice a night without their parents realizing it. In most cases, the children go back to sleep and do not alter the family's sleep routine. The data from EEG studies of children between 6 and 11 years of age who experience 1–3 three small awakenings per night suggest that the sleep of preschool children is more fragmented than we might think.

Another aspect that implies complex interaction of several factors is the initiation of sleep. In general, by 2–3 years of age, children are adapted to the routines and rituals of the family with respect to sleep. The use of audiovisual media, extended working hours of parents, or bedroom sharing with siblings can result in alteration of the time for going to sleep.

Nocturnal fears and nightmares are characteristic of this age and can often disrupt sleep.

A sleep anamnesis at this age should include the following points:

1. Sleep rituals and hygiene
2. Switching from a crib to a bed at around 2.5–3 years of age
3. Naps: their number and duration
4. Symptoms of attention deficit, misbehavior, hyperactivity, bad moods, changes in regular patterns in daycare or school
5. Nightmares or nocturnal fears
6. Shared bed, sleep space environment, use of a pacifier
7. Enuresis (which generally should not occur beyond 6 years of age)
8. Drowsiness and problems in waking up
9. Use of electronic devices (e.g., television, radio, cell phone, computer, tablet, or electronic game)
10. Allergies, dermatitis, asthma
11. Snoring

Respiratory sleep disorders begin to increase at this age.

Schoolchildren and Neuronal Plasticity

Slow-sleep-wave activity can be mapped with EEG spectral analysis, showing cortical development. Slow-wave activity in preschool children is predominant in the occipital lobes and moves progressively to the parietal and occipital regions during adolescence. The role of naps in this maturation process is not clear, but it appears that they play an important role in consolidating learning and memory. Naps remain important in

the schedules of many daycare centers and schools. In this respect, a study indicated that the use of naps resulted in a 10% improvement in learning of visual–spatial tasks by preschool children. The concomitant polysomnography record in this study showed the presence of a larger number of sleep spindles among the children, which would improve their learning.

The impact of sleep on memory is one the main concerns at this vulnerable stage of neurocognitive development. Memory development is influenced by a series of molecular factors and signals. One of the processes of memory is consolidation, which is favored by repairing sleep. It is assumed that there are regenerative processes during sleep, distributed into two stages, the first occurring immediately at the beginning of sleep, characterized by signals and protein synthesis in the hippocampus, and a second stage 4 hours later. The kinase that facilitates these processes is altered when individuals lose sleep or change their sleep schedule.

Adenosine inhibits the hippocampus, which reduces neuronal plasticity. The oscillating theta rhythm in the hippocampus can be shown both in REM sleep and in wakefulness during the execution of neurocognitive learning and memory tasks. It was initially believed that REM sleep was responsible for memory consolidation, but in the 1980s it was shown that slow-wave NREM sleep affects memory consolidation. The increase in slow-wave NREM sleep and the density of sleep spindles subsequent to training and learning has been demonstrated in studies with human and with animal models. A "dual hypothesis" is currently proposed and assumes that NREM sleep is responsible for declarative memory consolidation in the hippocampus, while REM sleep tends to regulate functional (procedural) and emotion-related memory. However, this vision is possibly too simplistic and cannot explain a series of much more complex interactions.

Nevertheless, it has been clearly demonstrated that sleep deprivation affects memory and changes the cytoarchitecture of the hippocampus. Considering how both REM and NREM sleep decrease over the course of one's life, the preschool and early school stages should be when more effort is made to protect neuronal homeostasis and the consolidation of memory in the hippocampus.

Attention deficit–hyperactivity disorder (ADHD) has been linked directly to sleep disorders, especially at the preschool and school stages, where it is also more evident to teachers. Treatment of this syndrome can alter the normal sleep of the child. Changes have been evidenced in slow-wave and REM sleep in children with ADHD in a manner similar to what was described above in relation to memory consolidation. The frontal brain activity of children with ADHD is slow during wakefulness and presents an anteroposterior imbalance in slow-wave activity during sleep. In particular, alterations have been found in the frontal circuits associated with control of emotions and memory regulation in children with ADHD. These circuits change over time and cease to be as important in adulthood, which could explain the lower prevalence of ADHD in adults than in children. This change with age could also explain why children show more hyperactive symptoms in response to poor sleep, while adults show more drowsiness.

Adolescents

The sleep patterns of adolescents are very similar to those of adults and, on average, adolescents sleep for 8–9.5 hours per night, 25% of which is REM sleep. Neuronal pruning continues, and it is supposed that this is the main mechanism producing changes in the sleep architecture seen in this population. In particular, there is a shift in the sleep phases at this age; thus, adolescents go to sleep later and wake up later. The normal phenomenon does not change the total number of hours of sleep. Distinct chronotypes can be distinguished at this age, and common complaints from parents and teachers are difficulty in waking up adolescents and their morning drowsiness. The idea of starting school at a later hour for adolescents than for preadolescents has been discussed in some parts of the USA and Canada.

An anamnesis at this age should include:

1. School performance: drowsiness can change performance and behavior in school
2. Depression and psychiatric illnesses
3. Hours slept
4. Daytime drowsiness: sleeping in classes or while doing tasks
5. Dental history: bruxism or cavities
6. Consumption of coffee, cola, energy drinks, or sports supplements
7. Headaches, blows, or falls

The interaction between sleep, puberty, and hormones is complex. Even when adolescents are not tired, they experience changes in melatonin secretion that invariably lead to a shift in the hours of the sleep phase. It is believed that this is linked to sexual hormonal development in adolescents. Endocrine changes during sleep follow fixed patterns specific to each hormone. Growth hormone is secreted in the first hours of sleep, reaching its maximum exactly an hour after the rise in prolactin. Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) also follow fixed patterns, and their release is delayed in adolescence. In contrast, the time when cortisol is released does not change during adolescence, and its maximum release is always kept for dawn at the end of sleep.

The maturation of the cerebral cortex in adolescents is not explained simply by an EEG phenomenon or change. One of the maturational mechanisms of the cerebral cortex is sleep spindles, which change with age and increase in frequency toward the frontal area. A lower frequency of spindles in children shows better neurocognitive performance, while the reverse has been demonstrated in adults, with a higher frequency of sleep spindles indicating better neurocognitive performance.

The cyclic alternating pattern (CAP) is an EEG and polysomnography pattern for assessing sleep stability. CAP alterations are associated with conditions such as Asperger syndrome and poor cognitive performance.

Risk Factors and Academic Performance

It should be kept in mind that there is a series of risks in relation to adolescents; an examining physician should ask about these and should be vigilant when dealing with an adolescent patient with a sleep disorder. Risky sexual behavior, eating habits, abrupt changes in mood, and use of medications or other drugs should immediately be associated with base diseases or mental health compromise. It is during adolescence that diseases such as depression, bipolar disorder, and (less commonly) Kleine–Levin syndrome appear. The latter is characterized by hypersomnia, hallucinations, hyperphagia, and hypersexuality. The syndrome is episodic and tends to resolve 5–10 years after its appearance.

Anxiety, mood, and depression disorders are often associated with sleep disorders. Adolescents who get less sleep (an average of 6.75 hours) than normal (an average of 8.25 hours) have been reported to have more mood disorders. Adolescents who are defined as “night persons” are more often reported as having symptoms of anxiety than those defined as “daytime” persons.

Mood disorders can result from even brief periods of sleep deprivation. It has been shown that patients with anxiety disorders in adolescence often had early sleep problems in childhood, including before the age of 4 years. One study found that adolescents with obsessive–compulsive disorder had shorter hours of sleep, reduced NREM sleep, and less REM latency than control subjects. Another study found more sleep fragmentation, microawakenings, and decreased slow-wave sleep among adolescents with anxiety disorders than among control subjects. In particular, adolescent males with depression have demonstrated decreased latency in REM sleep, an increase in the N1 sleep stage, a larger number of microawakenings, and decreased short-wave sleep.

Academic performance correlates significantly in many studies with sleep-related problems. Treatment of these problems has been

shown to improve poor academic performance. A study of first graders found that students who had undergone surgical treatment had improved their grades. Similar results from other studies suggest that good school performance is associated with regular long hours, while daytime drowsiness is associated with lower grades.

Specific Sleep-Associated Symptoms

There is a long list of symptoms that are understood as physiological and normal in sleep. Among the most common are the following:

1. Benign childhood myoclonus, which refers to local or diffuse twitching in otherwise healthy children (with normal EEG readings) that stops immediately upon the child waking up.
2. Movements at the beginning of sleep, related to small tremors in the transition between wakefulness and sleep.
3. Somniloquy or sleep-talking, which occurs particularly during NREM sleep.
4. Nonepileptic nocturnal psychogenic con vulsions.
5. Sandifer syndrome, which is nocturnal waking with rigidity and movement of extremities, associated with the presence of gastroesophageal reflux.
6. Sleep paralysis, which generally occurs during the transition from sleep to wakefulness. Paralysis that occurs at the beginning of sleep is termed hypnagogic, and that which occurs upon waking is termed hypnopompic. While sleep paralysis is benign in most cases, it is associated with epilepsy in a small percentage of cases.
7. *Jactatio capitis*, which is rhythmic head movement that is sometimes accompanied by similar body movement. This is typically found in patients with Down syndrome or neurocognitive disabilities.
8. Parasomnias, which occur in up to 80% of children between 2 and 6 years of age. This commonly occurs during NREM sleep and is distinguished among types of parasomnia by the frequency of disorders of the "arousal" or awakening type. The latter tend to be benign and are associated with premature birth and difficulties during pregnancy or birth.
9. Confusional arousal, which can emerge from deep NREM sleep and has an autonomic component ranging from the very simple to the complex.
10. Somnambulism or sleepwalking, which can occur at any age but is most common in adolescents. Episodes occur during NREM sleep, with general rhythmic activity of 2–7 Hz. An autonomic component is generally absent.
11. Nocturnal terror, which is one of the most common reasons for medical referral, owing to the dramatic nature of these episodes. The child awakens in an agitated state, screaming in terror, and parents often report an inconsolable cry. Diagnosis is generally possible with polysomnography or a video EEG. Seizures, which are the differential diagnosis, frequently occur during stages 1 and 2, while most parasomnias occur during stage 3. Children do not remember nocturnal terrors from one day to another.
12. Nightmares, which are disagreeable dreams, typically in childhood. They occur in the early hours of dawn during REM sleep, and a memory of this type of dream almost always remains the following day.
13. Bruxism, which is the habit of moving and grinding one's teeth. It is most common among children between 7 and 10 years of age. It rarely continues beyond adolescence, except in association with stress or obstructive sleep apnea.

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