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REVIEW ARTICLE

Sleep disorders in childhood

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Abstract

Objective: the aim of this article is to describe the main sleep disturbances in the paediatric age group, as well as the diagnostic criteria and management for the paediatrician. **Methods:** a non-systematic review of the current literature was made, based on the most recent international classification. **Results:** sleep disturbances are common in the paediatric age group, and can lead to a series of behavioural, social, and cognitive diurnal consequences. A sleep-directed interview is essential for suspicion and, frequently, sufficient for the diagnosis. The management is dependent on the diagnosis, as well as the severity of symptoms. **Conclusion:** the paediatrician plays an important role in the detection of sleep disturbances. Awareness of such conditions is essential for diagnosis and early management.

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INTRODUCTION

Sleep plays a fundamental role in child growth and development; sleep patterns can be observed in fetuses starting from around 26 weeks.[1] During the first year of life, marked changes occur in sleep characteristics, which mature over the course of childhood.[2] Newborns demonstrate an ultradian sleep pattern, with frequent awakenings more associated with hunger and discomfort than the time of day, whereas 1-year-old infants already have a well-established circadian cycle.[3]

Sleep duration over 24 h, which varies from 14 to 17 h in newborns, drops to 11–14 h between the first and second years of life and subsequently decreases to 10–13, 9–11, and 8–10 among preschoolers, school-aged children, and adolescents, respectively.[4] During the first years of life, reduction in total sleep mainly occurs due to decreasing daytime sleep periods. In this way, approximately half of the sleep period of a 1-month-old infant is distributed during the day, whereas a 12-month-old infant will have 1 or 2 episodes of daytime sleep lasting around 1.5 h. By 5 years of age, the need for daytime sleep disappears, with the morning nap eliminated first.[5]

As the circadian rhythm is established, the number of nighttime awakenings gradually decreases over the first year of life. Therefore, whereas a 1-month-old infant wakens 2 or 3 times per night, a 12-month-old infant usually does not wake up more than 2 times per night.[6] However, brief awakenings that follow the sleep cycle (90–120 min) continue to occur, with the child normally falling back to sleep without external intervention.[7]

Sleep disorders are prevalent in the pediatric age range[8] It is estimated that until adolescence, 20%–30% of children present some sleep abnormality, and this prevalence is higher among children with neuropsychiatric comorbidities.[9] On the other hand, sleep disorders may also in themselves increase the risk that a series of metabolic and behavioral changes may emerge, leading to attention deficits, mood disorders, weight gain, and even neurodevelopmental alterations.[10,11]

The pediatrician plays a fundamental role in orienting sleep habits, as well as in recognizing, suspecting, and managing possible disturbances. The objectives of this article are to describe the main sleep disorders in children, with an emphasis on the most prevalent, and to cite management measures according to the diagnosis.

SLEEP DISORDERS

The most recent edition of the International Classification of Sleep Disorders (ICSD-3) divides sleep disorders into seven main categories, as described in Table 1.[12]

Insomnia

Insomnia is the most prevalent sleep disorder in the pediatric age group, affecting up to 30% of children.[7] The ICSD-3 defines insomnia as difficulty initiating or maintaining sleep, waking up earlier than desired, resisting the onset of sleep, or difficulty initiating sleep without intervention by parents or caregivers in an environment conducive to sleep (without the use of television, smartphones, or tablets at bedtime).[13] Diagnosis requires daytime consequences of the difficulty described in the form of drowsiness or fatigue and changes in performance at school or at work, in intellectual capacity, or in mood or behavior. These consequences may be described for the child as well as the main caregiver. Insomnia is defined as chronic if present at least 3 days per week for at least 3 months.[9,13]

Table 1. Classification of Sleep Disorders according to the International Classification of Sleep Disorders (ICSD)*

1. Insomnia
2. Sleep-Related Breathing Disorders
3. Centrally Originating Hypersomnias
4. Circadian Rhythm Sleep and Wakefulness Disorders
5. Parasomnias
6. Sleep-Related Respiratory Disorders
7. Other Sleep Disorders

*Adapted from Sateia MJ.6

The most prevalent causes of insomnia vary according to age range and are described in Table 2.

While the main causes among infants are reflux, excessive ingestion of liquids, and inappropriate associations for the onset of sleep, among adolescents, physiological delay of sleep phase, psychiatric comorbidities, and family pressure are significant.[7]

Sleep onset association disorder, one of the types of behavioral insomnia, is one of the most prevalent disorders among infants and preschoolers. In this case, the child requires certain external conditions to fall asleep. This inevitably requires intervention from parents or caregivers such as swaddling or breastfeeding the child. In this way, there is a psychological wakening at the end of each sleep cycle when the intervention must be repeated for sleep to resume. When this associated factor is absent, there is a loss of sleep for both the child and caregiver.

Sleep-Related Respiratory Disorders

This classification includes pathologies associated with breathing and ventilation abnormalities during sleep; in the latest edition of the ISCD, these comprise obstructive sleep apnea (OSA), central apnea syndromes, sleep-related hypoventilation, and sleep-related hypoxemia.[13] Of these, OSA is the most prevalent and relevant for pediatric patients.

OSA is characterized by partial or complete obstruction of the upper airways, leading to increased respiratory effort, hypoxia, and hypercapnia.[14] It affects 1%–5% of the pediatric population, with peak incidence between 2 and 8 years, and the main cause is adenotonsillar hypertrophy.[15-17] Risk factors are male sex, black race, family history of OSA, prematurity, obesity, allergic rhinitis, asthma, presence of neurological diseases such as Down syndrome, Prader–Willi syndrome,

Table 2. Causes of Insomnia According to Age Group*

< 2 years	Inadequate associations for onset of sleep
	Gastrointestinal alterations (gastro-esophageal reflux, food allergies, infant colic)
	Excessive ingestion of liquids
	Acute infectious diseases
	Chronic diseases
2–3 years	Inadequate associations for onset of sleep
	Fear or anxiety about separation from parents
	Long naps or naps at inappropriate times
	Acute infectious diseases
	Chronic diseases
Pre-school and school-aged children	Lack of establishment of limits
	Fear or nightmares
	Acute infectious diseases
	Chronic diseases
Adolescents	Delayed sleep phase
	Sleep hygiene problems
	Psychiatric comorbidities
	Family and/or school pressure
	Sleep-related respiratory disorders
	Movement disorders
	Acute infectious diseases
	Chronic diseases

*Adapted from Nunes and Bruni.[7]

Chiari malformation, cerebral palsy, micrognathia, and neuromuscular diseases.[14,18] The clinical presentation varies and may include breathing difficulty at least 3 nights per week (in the absence of acute upper airway pathologies), secondary nocturnal enuresis, cervical hyperextension during sleep, morning headache, daytime sleepiness, or sensation of non-restorative sleep, symptoms of inattention and/or hyperactivity, and learning difficulties.[18] Furthermore, over the long term, this condition is associated with stature problems, hypertension, and even right ventricular hypertrophy.[16,17] The diagnostic criteria include habitual snoring, respiratory effort/obstruction, or daytime symptoms related to sleep fragmentation (excessive sleepiness, hyperactivity) and specific polysomnographic findings (≥ 1 obstructive events per hour of sleep or pCO_2 of > 50 mmHg during > 25% of sleep time, associated with snoring, paradoxical thoraco-abdominal movements, or reduced amplitude of nasal flow pressure wave).[13]

OSA is classified as mild at an apnea–hypopnea rate of 1–5 per hour, moderate when the rate is 5–10, and severe when there are > 10 events per hour.[14]

Centrally Originating Hypersomnias

Centrally originating hypersomnias are classified as narcolepsy type 1 (formerly "with cataplexy"), narcolepsy type 2 (formerly "without cataplexy"), idiopathic hypersomnia, Kleine–Levin syndrome, hypersomnia secondary to medical pathology and medication or substance, hypersomnia associated with psychiatric pathology, and insufficient sleep syndrome.[13] The common point among these pathologies, according to the ICSD-3, is daily episodes of the irrepressible need to sleep or daily sleep episodes.[13]

Narcolepsy is one of the most common causes of excessive daytime sleepiness, affecting approximately 1 in every 2000 individuals, and has peak incidence in the second decade of life.[19] Nevertheless, the period from the onset of symptoms to the establishment of diagnosis tends to be long, on average up to 15 years, mainly because of lack of knowledge of this diagnosis among physicians.[9,20] In addition, the symptoms of narcolepsy are commonly confused with other pathologies, leading to erroneous diagnoses of OSA, chronic fatigue syndrome, psychiatric diseases (depression, schizophrenia), conduct and learning disorders, and epilepsy.[21] In addition to excessive daytime sleepiness, classic symptoms are cataplexy (a REM sleep intrusion phenomenon consisting of sudden loss of muscle tone without loss of consciousness lasting a few second to a few minutes usually triggered by strong emotions) in narcolepsy type 1, sleep paralysis, or hallucinations at the beginning or end of sleep (hypnagogic or hypnopompic).[20])

Narcolepsy type 1 is characterized by low levels of hypocretin-1 (a neurotransmitter responsible for regulating the sleep–wake cycle, eating and reward behaviors, as well as autonomic and neuroendocrinological activities) in the cerebrospinal fluid due to the loss of hypothalamic neurons responsible for its production.[19,22] The diagnostic criteria include two episodes of early REM sleep, starting < 15 min after falling asleep (SOREMP) in the multiple daytime sleep latency test (MSLT) or one SOREMP on a polysomnography (PSG) together with SOREMP in the MSLT.[13] For the diagnosis of narcolepsy type 2, the same criteria for multiple daytime latencies must be present. However, cataplexy is absent and levels of hypocretin-1 in the cerebrospinal fluid exceed the determined levels for the diagnosis of narcolepsy type 1.[13] The diagnosis requires PSG followed by the multiple daytime sleep latency test. Narcoleptic patients, especially those who show signs of the disorder before puberty, exhibit high rates of obesity and endocrine changes (such as precocious puberty), as well as OSA and migraine and psychiatric comorbidities such as depression, anxiety, and attention deficit–hyperactivity disorder (ADHD).[23]

Circadian Rhythm Sleep and Wakefulness Disorders

Among other diagnoses, these disorders include phase delay and advancement and jet lag (usually transient and

caused by trips across time zones). Most commonly seen in the pediatric population, more precisely in adolescents (and may affect up to 16% of the population in this age group), is delayed sleep phase syndrome.[24] This disorder is characterized by a delay in the time of sleep onset, usually by > 2 h in relation to the time desired by the individual needed to fulfill social commitments.[25] This causes the adolescent to have difficulty getting up in the morning as well as daytime sleepiness, which may also affect school performance. Several factors may be involved in pathogenesis, from social pressure (exacerbated by access to electronic media at bedtime) to issues of homeostatic development and circadian rhythm associated with puberty, which may be evaluated through anamnesis or objective methods, such as actigraphy.[26-28]

Parasomnias

Parasomnias are more common in childhood and represent a dissociation between wakefulness and sleep and REM and non-REM sleep, with superposition of the characteristics of one state onto the other, resulting in undesirable behavioral phenomena.[29,30] One longitudinal study in Canada, which included approximately 1500 individuals aged 2.5–6 years, found that almost 90% of participants had at least one parasomnic episode during the study period.[31] Parasomnias are classified as non-REM sleep-related (most commonly in childhood, confusional arousal, sleepwalking, and night terrors), REM sleep-related (nightmares), and other parasomnias including nocturnal enuresis.[13]

Confusional arousals are most frequent among infants, preschoolers, and school-aged children and are characterized by partial awakening from slow-wave deep sleep within the first 2–3 hours of sleep; the child tends to sit up in bed and despite being unresponsive, screams and appears terrified and demonstrates motor agitation. The duration of the episodes varies (average of 15 min, but may last for hours), after which the child falls back to sleep and wakes up the next day with no memory of what happened.[29,32]

Night terrors also occur in the first third of the night, usually in children between 3 and 10 years of age.[29] In these episodes, the child is extremely agitated, appears confused or frightened, and usually cries or screams, and there is also autonomic activation in the form of sweating, tachycardia, tachypnea, and mydriasis. The child may get out of bed and run aimlessly.[9] The episodes are short, lasting a few minutes, and as with confusional arousals, the child has no memory of what happened.[32] Night terrors are part of the same spectrum of sleepwalking, and consequently children with a history of night terrors are twice as likely to sleepwalk.[9] This is characterized by ambulation and stereotypical behavior and may eventually lead to accidents such as falls.[33]

Nightmares, on the other hand, usually occur in the second half of the night when REM sleep predominates.[34] These dreams are associated with negative emotions and lead the child to wake up and remember them; they are more prevalent in children between 5 and 10 years of age.[35] Although they occur sporadically in most school-aged children, in recurrent and clinically significant cases, they are strongly associated with psychopathological conditions, especially post-traumatic stress disorder, depressive disorders, and substance abuse.[36,37]

Primary nocturnal enuresis is defined as persistent urinary incontinence during sleep after 5 years of age, without an interval of at least 6 months of nighttime continence.[29] The prevalence is 5%–10% of children at age 7 years and is more common in boys.[38] This condition has a strong genetic predisposition; when both parents have a history of enuresis, three-fourth of their offspring will have the disorder; if one parent has a history of the condition, half of their offspring will also inherit it.[39] This condition differs from secondary nocturnal enuresis, where symptoms return after a period of at least 6 months of nighttime continence, and may be associated with sleep breathing disorders, diabetes, and epilepsy. Important in investigation is routine urinalysis and culture, if necessary.[29]

Sleep-Related Movement Disorders

These are simple movements that occur during sleep with stereotyped frequency.[13]

Restless leg syndrome (RLS) affects 2%–4% of school-aged children and adolescents and can influence not only sleep quality but also mood and quality of life.[40] It is characterized by a need to move the legs, usually accompanied by discomfort, and is triggered or exacerbated by rest at night and is partially or completely relieved with movement; it impacts daytime energy, behavior, or mood.[40] Periodic limb movement disorder (PLMD) is related to RLS, and a child with a diagnosis of PLMD may eventually develop RLS. A diagnosis of PLMD requires the presence of clinical symptoms of insomnia, difficulty maintaining sleep, or excessive daytime sleepiness associated with polysomnographic documentation of > 5 periodic limb movements per hour of sleep, which cannot be explained by other pathology (such as OSA) or pharmacological effects (such as from antidepressants).[40]

Children with RLS or PLMD frequently have low iron levels, and consequently, it is important that blood count and serum iron and ferritin levels be tested.[41] Furthermore, RLS may be present in up to 44% of children with ADHD.[42]

Another common movement disorder is bruxism and may affect up to approximately 40% of children; this disorder involves repetitive involuntary jaw muscle activity characterized by clenching or grinding teeth.[29,43] It may be associated with local factors such as temporomandibular joint pathologies or malocclusion, but extrinsic factors are also present, such as anxiety, stress, and ADHD.[32]

Benign sleep myoclonus of childhood may start in the neonatal period and extend into the first 6 months of life. The infant exhibits clusters of myoclonic movements during sleep, generally at onset of sleep, during any phase (although it is less frequent during active sleep); all four limbs may be affected, and the condition resolves upon awakening.[44] The

main differential diagnosis is epileptic-origin myoclonus. In this case, the anamnesis or electroencephalography can rule out epilepsy by investigating events of the previous night or possible changes in neuropsychomotor development.[45]

Rhythmic movements when falling asleep are physiological in the first years of life, generally resolve between 3 and 4 years of age, and are more prevalent among boys. They may also occur during non-REM and REM sleep. The most common types are lateral head movements (from one side to another), hitting the head, and rocking movements with the body. It should be differentiated from rhythmic movements occurring during daytime, as seen in children on the autism spectrum or with cognitive delay. The child generally does not remember the episode upon awakening, and these movements are only diagnosed as a disorder when there are significant associated consequences such as accidents.[29]

Treatment of sleep disorders

Table 3 summarizes therapeutic strategies according to the diagnosis of sleep disorder. For doses and side effects of medication, the literature should be consulted.

Management of sleep disorders varies according to etiology and sometimes according to the degree of clinical impairment. Some diagnoses, such as benign myoclonus and rhythmic movements when falling asleep, do not need specific treatment, since they tend to resolve with growth and development.

For bruxism, no treatment has proven effective at this time, and in most cases, it can be considered an oral parafunction that needs to be monitored.[43] Possible associated comorbidities should be eliminated, such as allergies, sleep apnea, and stress factors, and other strategies should be abandoned in selected cases.

Management of OSA should consider not only the index resulting from PSG, but also the degree of daytime symptoms and morbidities.[14] Recommendations for tonsillectomy should be individualized and should be considered for first-line treatment in children with moderate to severe OSA associated with adenotonsillar hypertrophy and who do not have contraindications to surgery (very small adenoids and amygdala, morbid obesity associated with small adenoids and amygdala, coagulopathies refractory to treatment, and the presence of submucosal cleft palate). Even among children without evident adenotonsillar hypertrophy upon physical examination, who present with OSA, the procedure can be considered, since the lymphoid tissue can occupy significantly more of the upper airways than can be observed. Furthermore, even though failure rates are higher than that in eutrophic children, obesity should not by itself contraindicate the procedure, considering that some degree of clinical improvement is generally seen.[16]

Medication is always used to manage narcolepsy, with a view to reducing daytime symptoms.

Table 3. Managing sleep disorders in childhood

Diagnosis	Management
Insomnia[7]	Sleep hygiene
	Behavior therapy
	Antihistamines
	Clonidine
	Melatonin
	L-5-hydroxytryptophan
	Zolpidem (adolescence)
Sleep-Related Breathing Disorders	
Obstructive sleep apnea syndrome[38]	Weight loss when necessary
	Nasal steroids
	Antileukotrienes
	Adenoidectomy/amygdalectomy
	Orthodontic treatments (such as maxillary expansion)
	Positive airway pressure
Centrally Originating Hypersomnias	
Narcolepsy[16,39]	Scheduled naps during the day
	Modafinil (daytime sleepiness)
	Sodium oxybate (cataplexy, daytime sleepiness, and sleep disorders)
	Methylphenidate and amphetamines (daytime sleepiness)
	Tricyclic antidepressants, selective serotonin uptake inhibitors, and venlafaxine (cataplexy)
Circadian Rhythm Sleep and Wakefulness Disorders	
Delayed sleep phase[7]	Sleep hygiene
	Melatonin
	Zolpidem
Parasomnias[25]	
Confusional arousal	Reassure parents
	Scheduled wakening
	Benzodiazepines*
Night terrors	Reassure parents
	Security measures in bedroom/ home
	Scheduled wakening
Sleepwalking	Reassure parents
	Security measures in bedroom/ home
	Scheduled wakening
	Benzodiazepines*
Nightmares	Reassure parents
	Selective serotonin reuptake inhibitors (off-label)

Primary nocturnal enuresis[36]	Restrict water intake 2 h prior to bedtime
	Try to empty the bladder just before bedtime
	Positive reinforcement
	Alarm therapy
	Imipramine
	Oxybutynin
	Desmopressin
Sleep-Related Movement Disorders	
Restless leg syndrome[32]	Higiene do sono
	Reposição de ferro, quando necessário
Periodic limb movement disorder[32]	Sleep hygiene
	Iron supplementation when necessary
Bruxism[33]	Long-term monitoring
	Behavioral therapy
	Sleep hygiene
	Temporary occlusal appliance
Benign sleep myoclonus of childhood[34]	Reassure parents
Rhythmic movements when falling asleep[21]	Reassure parents
	Measures to prevent accidents according to the severity of the symptoms

*should not be used as first choice.

For the treatment of primary nocturnal enuresis, behavioral measures (such as not drinking water 2–3 h before bedtime and urinating before bedtime) are recommended alongside positive reinforcement with posters or calendars prepared by the child and parents together, presenting rewards for nights when enuresis does not occur. One device regarded as first-line management is an alarm that wakens the child when a small amount of urine emissions is detected, allowing them to avoid unintended loss of large volumes of urine.[46]

Common denominators in the management of sleep disorders are behavioral strategies and sleep hygiene routines. They may be sufficient in a series of diagnoses, and even when other types of approaches are recommended, they may act as adjuvants. Pediatricians should be familiar with these techniques to orient and supervise families. Sleep hygiene measures should include regular bedtime according to age, and restriction of stimulants such as soft drinks, teas, and chocolate, especially at night. Positive routines, which consist of quiet and pleasant activities before bedtime (reading and listening to calm music), should be widely adopted.[47]

The sleep environment should be the child's own bed without the need for parental intervention to avoid sleep onset associations. In children with insomnia associated with inappropriate associations, the techniques of extinction ("crying it out") and minimal checking with systematic extinction can be put into practice.[7] Gradual extinction consists of ignoring nighttime demands (crying and calling parents) for increasingly longer periods of time, starting with short periods of ≤ 1 min, according to parent tolerance and judgment. Minimal checking with systematic extinction consists of applying the extinction technique, but permits periodic checks on the child (every 5–10 min) and quick comforting when necessary.[47] For both techniques, it is important to ensure the safety of the environment at the moment when the child is put down to sleep.[7]

For children who tend to wake during the night, scheduled waking may be used. This consists of waking the child around 15 min before the time when they awaken spontaneously and spacing the episodes gradually.[47] Scheduled waking can also be used in cases of nocturnal enuresis, and the children awoken before the time enuresis generally occurs.[32]

Electronic media should also be avoided at least 1 h before sleep, and sleep should begin in bed (and not in other places such as the living room couch and the child later transferred to bed), the temperature should be appropriate, and the room should be dark enough to permit sleep.[7]

Reshaping of children's sleep in the age range where a period of daytime sleep is still expected allows nighttime sleep to not be impaired by daytime napping. This consists of organizing naps to occur 4 h prior to nighttime sleep among children who take two naps per day and 6 h prior in those who take one.[47]

In the management of delayed sleep phase syndrome, one strategy is to delay bedtime to ensure that the child or adolescent falls asleep quickly when he or she lays down. When the habit of falling asleep quickly is established, bedtime can be moved earlier by 15–30 min each night until suitable time is achieved.[47]

CONCLUSION

Sleep disorders are heterogeneous with regard to their causal factors, clinical presentation, and morbidity, which makes knowledge of physiological aspects of sleep and of the most prevalent disorders essential for appropriate orientation and management of the family.

Even though nocturnal PSG is considered the gold standard in the diagnosis of sleep disorders, the pediatrician plays a fundamental role in the diagnostic process, which includes clinical suspicion and referral for specialist assessment, when necessary. For this, a patient history that includes questions on the routine and details of the family environment related to the child's sleep, in addition to a detailed clinical examination, must be a part of the physician's routine anamnesis.

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