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Pediatric Sleep Respiratory Disorders: Epidemiology, Risk Factors, Management, And Role of Family Medicine.

[1]Abdulaziz Khalid Albulaihed, [2]Reham Abdulrahman Qari, [3]Abdulaziz Mohammed Abdulaziz Alothman, [4]Ahmed Mohamed Darbashi, [5]Hessa Mohamed Dakkam, [6]Rakan Ibrahim M. Almuqbil, [7]Saad Abdulrahman S Almahbub, [8]Eman Alhar

[1] KSA, Ministry Of Health, Cluster One Riyadh
[2] KSA, Ministry Of Health
[3] KSA, Ministry Of Health, Al Yamamah Hospital Riyadh
[4] KSA, Ministry Of Health, Samtah General Hospital
[5] KSA, Ministry Of Health, Jazan Specialized Hospital
[6] KSA, Ministry Of Health, General Practitioner At Riyadh's Second Health Cluster Second.
[7] KSA, Ministry Of Health
[8] KSA, Ministry Of Health, School Health

Abstract:

Background: Sleep plays a vital role in the development and well-being of children. Sleep-disordered breathing (SDB), which includes obstructive sleep apnea (OSA), is a significant pediatric concern. Obstructive SDB can manifest primary snoring, upper airway resistance syndrome, obstructive hypoventilation, or OSA, with varying degrees of severity. These disorders affect children's physical growth, cognitive development, and overall health. SDB often remains undiagnosed, and its prevalence is complicated by varying diagnostic criteria and risk factors.

Aim: This review aims to examine the epidemiology, risk factors, and management of pediatric sleep respiratory disorders, particularly obstructive SDB, and the role of family medicine in addressing these issues.

Methods: A narrative review was conducted based on a computerized search of PubMed using the terms "sleep-disordered breathing" AND "children," focusing on studies from the past 20 years. Key studies were synthesized, focusing on the epidemiology, risk factors, and management of obstructive SDB in both the general pediatric population and children with complex health conditions.

Results: Obstructive SDB prevalence varies from 1.2% to 13%, with snoring affecting 7.45% of children. Common risk factors include adenotonsillar hypertrophy, allergic rhinitis, asthma, obesity, craniofacial abnormalities, and preterm birth. Two main phenotypes are identified: the "classic phenotype," which peaks between 2 and 8 years, and the "obese adult phenotype," prevalent in adolescents. Early identification and management are crucial, as untreated OSA can lead to cognitive, behavioral, and cardiovascular issues.

Conclusion: Pediatric sleep respiratory disorders are a prevalent and significant health issue. Obstructive SDB can range from mild snoring to severe OSA, and early intervention is critical for preventing long-term complications. Family physicians play an essential role in early detection, diagnosis, and management of these disorders, offering vital guidance for families.

Keywords: Pediatric, sleep-disordered breathing, obstructive sleep apnea, risk factors, management, family medicine.

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Introduction:

Sleep is not merely the absence of wakefulness; rather, it is an active neurophysiological process that serves a multitude of essential functions. The duration, quality, and architecture of sleep undergo significant changes throughout life, especially during the first five years, which profoundly affect brain development. Consequently, adequate sleep is critical for physiological growth, early memory consolidation, learning, cognitive development, and cardiovascular health [1]. Given the importance of sleep in the life of children—newborns, for example, sleep for up to 80% of the day [2]—and its persistence in early childhood, where toddlers and preschoolers spend half of their day sleeping, it is clear that insufficient sleep, poor sleep quality, or disturbances during infancy can negatively influence both immediate and future health outcomes for children and their families. Sleep-disordered breathing (SDB) encompasses a range of conditions, with obstructive SDB being the predominant category. Other, less common forms include central sleep apnea (CSA), central congenital hypoventilation syndrome (CCHS), prematurity apnea, apparent life-threatening events (ALTEs), and brief resolved unexplained events (BRUEs). According to the European Respiratory Society (ERS) statement [3], obstructive SDB refers to a syndrome characterized by dysfunction of the upper airway during sleep, typically manifested as snoring and/or increased respiratory effort due to elevated upper airway resistance and pharyngeal collapsibility. This syndrome includes a spectrum of disorders of varying severity, ranging from primary snoring to upper airway resistance syndrome (UARS), obstructive hypoventilation, and obstructive sleep apnea (OSA).

Obstructive SDB conditions include:

- **Primary Snoring**: Characterized by habitual snoring (occurring more than three nights per week) without the presence of apneas, hypopneas, frequent arousals, or gas exchange abnormalities.
- **Upper Airway Resistance Syndrome (UARS)**: Involves snoring, increased breathing, and frequent arousals, without clear obstructive events or gas exchange abnormalities.
- **Obstructive Hypoventilation**: Defined by snoring and elevated end-expiratory carbon dioxide partial pressure, in the absence of recognizable obstructive events.
- **Obstructive Sleep Apnea Syndrome (OSA)**: Characterized by recurrent partial or complete upper airway obstruction (hypopneas, obstructive or mixed apneas), disrupting normal oxygenation, ventilation, and sleep patterns.

Each of these clinical entities should be understood as part of a broad spectrum rather than a linear progression of increasing severity. However, it is important to note that approximately 2–3% of children with habitual snoring will progress to clinically significant OSA [4]. Therefore, habitual snoring should always be thoroughly investigated as a potential precursor to OSA [5]. Initially, obstructive SDB was predominantly described in the adult obese population. In 1976, Guilleminault [6] documented the first pediatric cases of OSA in children with adenotonsillar hypertrophy. Today, obstructive SDB remains a significant health concern among children. According to the American Academy of Pediatrics (AAP) guidelines from 2012 [7], the prevalence of OSA in the pediatric population is estimated to be between 1.2% and 5.7%. Similarly, the ERS 2016 statement [3], referencing a meta-analysis of published studies, reports that the prevalence of OSA in children ranges from 0.1% to 13%, with most studies showing a prevalence between 1% and 4%. The same meta-analysis also reports that the prevalence of habitual snoring among children is 7.45%, with some studies documenting a prevalence as high as 35% and others as low as 3% [8,9,10]. This wide variability in reported prevalence rates can likely be attributed to differences in the criteria used to define "habitual snoring," reliance on parental and self-reports, and cultural or individual differences in the perception of snoring. Moreover, the epidemiology of pediatric OSA and obstructive SDB in general is complex, with numerous methodological challenges arising from heterogeneity in diagnostic criteria, variations in the age of children studied, and differences in the presence of underlying medical and neurological conditions.

In this narrative review, we seek to synthesize key studies on the epidemiology and risk factors associated with respiratory sleep disorders, particularly obstructive SDB, within both the general pediatric

population and among children with more complex health conditions. By conducting a computerized search on PubMed using the terms "sleep-disordered breathing" AND "children" and limiting the search to studies from the past 20 years, we aim to provide an expansive overview of the current state of knowledge, while critically evaluating existing literature, acknowledging the lack of standardized methodologies and statistical analyses in many studies.

Obstructive SDB Phenotypes

Obstructive sleep-disordered breathing (SDB) affects children across all age groups, from neonates to adolescents, with a balanced gender distribution observed in preschool and older children [11]. The condition typically peaks between 2 and 8 years of age due to the relative increase in lymphatic tissue, which constitutes a primary risk factor for upper airway obstruction, often referred to as the "classic phenotype." However, the recent surge in obesity rates among pediatric populations has led to a second peak of SDB prevalence during adolescence. According to Dayyat et al. [4], an epidemiological shift has been observed, where children who would typically exhibit the adenotonsillar "classic phenotype" are increasingly displaying characteristics resembling those of adult SDB, known as the "obese adult phenotype." The European Respiratory Society (ERS) statement identifies two main pediatric populations affected by obstructive SDB: one comprising otherwise healthy children aged 2 to 18 years, which includes the "classic phenotype" and the "obese adult phenotype," and another group consisting of children under 2 years, who are more likely to suffer from complex conditions such as craniofacial abnormalities, neuromuscular disorders, and genetic syndromes, collectively known as the "congenital phenotype" [3,12].

Classic Phenotype

Adenotonsillar hypertrophy represents the most prevalent risk factor for obstructive SDB in otherwise healthy children. The growth of lymphoid tissue in the Waldeyer ring progresses from birth, reaching a significant increase between the ages of 3 and 6, which coincides with the peak incidence of obstructive sleep apnea (OSA). This physiological growth of lymphoid tissue can be further exacerbated by upper and lower airway inflammation, which may stem from conditions like allergic rhinitis (AR), asthma, recurrent infections, and exposure to environmental irritants such as tobacco smoke [4]. Children with AR are three times more likely to experience sleep disturbances [13], and chronic inflammation in the upper airways is a potential risk factor for adenotonsillar hypertrophy. A recent study involving 102 children with adenotonsillar hypertrophy found that 71% were sensitized to multiple allergens, either in their serum or adenotonsillar tissues, with 36% of those with specific IgE-negative serum displaying positive IgE in their adenotonsillar tissues [14]. Furthermore, AR itself has been identified as an independent risk factor for snoring, significantly increasing the likelihood of moderate or severe SDB [15]. Allergic congestion resulting in nasal resistance often leads to mouth breathing, which can cause impairments in maxillofacial development, malocclusion, relative tonsillar enlargement, and dysfunction of the genioglossus and geniohyoid muscles, further contributing to SDB. This can also result in a characteristic long face, a condition that predisposes children to SDB [16]. Additionally, certain inflammatory mediators associated with allergic reactions might disrupt sleep rhythms by directly influencing the central nervous system [15]. However, the ERS statement cautions that the evidence linking AR to OSA derives from studies of lower methodological quality [17].

Several studies have highlighted an elevated risk of SDB in children with asthma. This correlation is largely based on the similar increases in the prevalence of asthma and SDB, both of which share an inflammatory pathogenesis. Children with OSA show heightened expression of leukotrienes and leukotriene receptors in the adenotonsillar tissues, and inflammatory mediators associated with asthma, such as cysteinyl leukotrienes, appear to play a significant role in the development of adenotonsillar hypertrophy in these patients [18]. However, the causal relationship between asthma and SDB remains undetermined [19]. Similarly, viral respiratory infections can induce the production of leukotrienes and oxidative stress markers in pharyngeal lymphoid tissues, promoting adenotonsillar enlargement and exacerbating sleep apnea [20]. Other recognized risk factors for SDB involve anatomical features of the upper airways and craniofacial structure, such as a retrusive chin, steep mandibular plane, vertical

craniofacial growth direction, and class II malocclusion [9]. Interestingly, recent research has identified a relationship between sleep-related behaviors, such as bedtime resistance, fragmented sleep, daytime sleepiness, and sleep bruxism, with temporomandibular disorders and dental caries. Additionally, polymorphisms in serotonin and dopamine pathway genes may explain the association between sleep bruxism and OSA [21,22]. Certain craniofacial abnormalities also help explain the higher prevalence of obstructive SDB in black children, who are at four to six times greater risk compared to white children, and in Asian children, who often exhibit more severe forms of OSA than their white counterparts [9,23,24].

Facial dysmorphia within the craniofacial complex may also explain the observed correlation between apparent life-threatening events (ALTE) and OSA. A recent retrospective study involving 107 preschool-aged children, all with at least one ALTE in the first year of life, revealed a higher prevalence of snoring, apneas, restless sleep, habitual mouth breathing, and malocclusion in this group compared to a control group of children without any ALTE history. The authors suggested that the occurrence of ALTE may predict the future development of SDB [25]. Prematurity is another potential risk factor for pediatric OSA. Preterm children experience obstructive SDB at rates three to five times higher than their full-term peers [23]. A prospective multicenter cohort study found that 9.6% of 197 former preterm children (weighing between 500 and 1250 g) displayed OSA on polysomnography conducted at school age (5-12 years) [27]. Premature infants exhibit smaller nasopharyngeal volumes, and other distinctive anatomical features, such as a relatively large tongue, unique jaw shape, short neck, higher larynx, and softer laryngo-tracheal cartilage, predispose them to obstructive events. Additionally, prenatal and perinatal factors, such as chorioamnionitis and multiple gestations, further elevate the risk for OSA in former preterm children [27]. Comorbid conditions, including chronic respiratory issues and neurological injuries, may also influence respiratory control development in preterm infants. Further investigation is needed to better define the risk factors and timing of OSA presentation in preterm children, although it is strongly recommended that these children be routinely screened for OSA to prevent associated complications [27]. Finally, genetic predispositions and family history may contribute to a child's risk of developing SDB [4,22]. Notably, significant interactions have been observed between heritability and familial aggregation of OSA, especially among overweight individuals [29].

Obese Adult Phenotype

Upper airway narrowing in individuals with obesity may be attributed to fatty infiltration of the upper airways as well as fat deposition in the anterior neck region. Obesity also imposes an increased mechanical load on breathing, concurrently diminishing ventilatory drive and response. Studies have demonstrated that for each increase of 1 kg/m^2 in BMI above the average, the risk of obstructive sleep apnea (OSA) rises by 12% [4,30].

Numerous studies identify obesity as one of the strongest risk factors for obstructive sleep-disordered breathing (SDB) in both adults and children [31]; however, the available evidence is often limited by confounding factors such as age and ethnicity. The peak incidence of the obese phenotype in pediatric populations typically occurs during adolescence, a period when the risk of obstructive sleep apnea syndrome (OSAS) is reported to be 4-5 times greater compared to normal weight situations [32]. However, cases of OSA linked to obesity have also been observed in younger age groups. Although the precise contribution of obesity to upper airway obstruction remains to be fully quantified [33,34], the European Respiratory Society (ERS) statement posits that by the age of two years, obesity could independently increase the risk of obstructive SDB [3]. In a recent retrospective study involving 60 obese toddlers and preschool children (mean age, 4.4 ± 1.7 years), 22 out of 60 (36.6%) children were diagnosed with OSA, predominantly in the moderate to severe range. The proportion of habitual snorers compared to children with clinically significant OSA was substantially higher in the obese cohort (34.5% vs. 3%) [35], highlighting obesity's significant role in the pathophysiology of upper airway obstruction during sleep [4]. In older obese children, the prevalence of OSA varies widely, ranging from 13% to 59%. This variation is likely due to the heterogeneity of inclusion and diagnostic criteria for both obesity and polysomnography [33,36,37,38,39,40].

As observed in adults, several studies have documented a progressively higher risk of SDB with increasing BMI [30]; however, certain studies have failed to establish a direct association between OSA and estimates of adiposity. Verhulst et al., in their study of 27 overweight and 64 obese children and adolescents, found that 19% of obese subjects and 41% of overweight subjects had OSA [33]. Although further research is required to clarify this finding, one possible explanation is that OSA may lead to increased nocturnal energy expenditure due to the added respiratory effort required during sleep [33]. Obesity in children may also correlate with other types of SDB. In Verhulst et al.'s study, central sleep apnea (CSA) was found in 4% of obese and 17% of overweight children, often in association with significant desaturations [33]. Moreover, obesity is recognized as a risk factor for hypoventilation syndrome (OHS), a rare form of SDB characterized by impaired ventilatory responses, leading to persistent hypercapnia. While there is growing evidence supporting the presence of OHS in obese adults with OSA—where hypoventilation has been found in 5.9–17.8% of cases, depending on the definition used for OHS [41]—the data concerning pediatric cases is limited, with only a few reported instances [42].

SDB in Children Less than 2 Years Old:

In 2017, the European Respiratory Society (ERS) published a distinct statement concerning obstructive sleep-disordered breathing (SDB) in children under the age of two, a specialized group of patients whose susceptibility to upper airway obstruction is primarily linked to several comorbidities, including craniofacial anomalies, neuromuscular disorders, and genetic syndromes [12]. Nonetheless, although less common, even healthy infants under 2 years of age can be impacted by "early obstructive sleep apnea" (OSA) due to adenotonsillar hypertrophy. A 2003 study by Greenfeld et al. assessed 29 infants under 18 months of age who suffered from early OSA associated with adenotonsillar hypertrophy, revealing a high incidence of prematurity (24%), a male predominance, a strong link with failure to thrive, and significant post-surgical weight gain. A subset of these infants (17%) demonstrated an elevated risk of developmental delay prior to surgery, with most of these issues resolving post-treatment. However, one-quarter of the infants experienced a recurrence of symptoms following polysomnography (PSG), necessitating a second adenoidectomy due to insufficient adenoid tissue removal in the first operation, which was attributed to technical difficulties, small airway dimensions, and residual adenoid tissue left unidentified during the initial procedure [43].

Children with Complex Disorders:

Due to their underlying comorbid disorders, which often impact the central nervous system, neuromuscular tone, and craniofacial structures, children with complicated medical conditions are more likely to develop sleep-disordered breathing (SDB) [44]. Furthermore, over time, these children's susceptibility to SDB is further exacerbated by recurring pulmonary exacerbations brought on by respiratory infections, aspiration, or compromised cough reflexes. These children's anatomical and functional anomalies increase their risk of developing OSA, central sleep apnea (CSA), and hypoventilation, among other types of SDB. The variety of underlying clinical disorders, different ages at study time, small sample sizes, and varied diagnostic criteria among studies make it difficult to pinpoint the precise prevalence of SDB in children with complex illnesses. Like other complex instances, SDB's clinical presentation is frequently challenging to identify, and the associated morbidities are more severe and typically appear earlier, making the use of thorough screening techniques necessary.

With a frequency of roughly 1 in 700 live births, Down syndrome (DS), also known as Trisomy 21, is the most common chromosomal condition in children [45,46]. Midfacial and mandibular hypoplasia, a shorter palate, macroglossia, a restricted nasopharyngeal lumen, and pharyngeal hypotonia are the main anatomical features of DS that lead to OSA and hypoventilation. DS children are also more susceptible to OSA and hypoventilation due to other reasons such obesity and adenotonsillar hypertrophy [47, 48]. According to several studies, the prevalence of OSA in children with DS ranges from 31% to 79%, which is significantly higher than in the general population [48,49,50,51,52,53,54,55]. But in this group, nocturnal hypoventilation and CSA are less prevalent. Sawatari et al. recently conducted a case-control research in which they used parental questionnaires and nighttime pulse oximetry to evaluate symptoms associated

with SDB in 51 children with DS and 63 healthy controls. According to their research, children with DS were far more likely to experience symptoms like snoring, apnea, midnight arousals, and daytime napping, and these symptoms tended to worsen as the kids got older. Furthermore, DS children had worse pulse oximetry measurements and were more likely to exhibit a specific sleep posture (53% of DS children vs. 9.5% of controls, $p < 0.0001$). Interestingly, children with DS were the only ones that slept in a sitting position [56]. Older age was linked to OSA, but there was no discernible sex difference, according to a recent meta-analysis of 24 studies that sought to uncover predicted risk factors for SDB in children with DS. However, because of the poor quality of the data, the author emphasized the difficulties in predicting SDB based just on clinical assessments including adenotonsillar hypertrophy, dental exams, body mass index (BMI), and sleep patterns [57]. Additionally, it was discovered that infants without snoring or observable apnea nevertheless had a significant prevalence of OSA [49,50,51]. Routine polysomnographic or polygraphic screening is advised for all children with DS by the age of four, or even earlier for symptomatic children, due to the high prevalence of SDB in DS and the challenge of detecting OSA solely through clinical examination [3,51].

About 1 in 10,000 to 30,000 neonates are affected by achondroplasia, the most prevalent skeletal abnormality [58,59]. A heterozygous mutation in the fibroblast growth factor receptor 3 gene (FGFR3), which is found on chromosome 4 (4p16.3), is the cause of this disorder [58]. Breathing problems are common in children with achondroplasia, especially when they sleep. OSA is the most common kind of SDB, and it is commonly associated by CSA and hypoventilation [3,12]. Midfacial hypoplasia, micrognathia, a depressed nasal bridge, relatively enlarged tonsils and adenoids, macroglossia, a high palate, decreased temporomandibular joint mobility, and hypotonia of airway muscles are the main causes of OSA and upper airway obstruction in this population. Severe CSA may also result from brainstem compression brought on by foramen magnum stenosis, which may necessitate surgical decompression in early childhood [59,60]. The reported frequency of OSA varies greatly, ranging from 19.1% to 87% [61,62,63,64,65,66,67], and there are few studies on the prevalence of SDB in children with achondroplastics. This variation is probably caused by variations in diagnostic criteria and patient heterogeneity (e.g., prior surgeries or oxygen supplementation). 43 children with achondroplasia (mean age 3.9 ± 3.5 years) participated in a recent retrospective study. Of these, 56% had aberrant PSG data, and more than a third had OSA despite previous or recurrent upper airway operations. There were no single CSA cases discovered [61]. In these cases, adenotonsillar hypertrophy was also found to be a complicating condition. 60% of achondroplasia patients had moderate to severe OSA at baseline (mean age 4.8 ± 4.1 years), according to Zaffanello et al. The severity of OSA significantly improved after adenotonsillectomy (mean apnea-hypopnea index [AHI] at baseline = 9.5 ± 9.6 events/hour; mean AHI post-surgery = 2.1 ± 1.1) [67]. For early SDB screening in these children, especially during the first few months of life or when signs of SDB are noticeable, polysomnography in conjunction with magnetic resonance imaging (MRI) is advised [68].
With an estimated prevalence of 1 in 10,000 to 25,000 live births, Prader-Willi syndrome (PWS) is a congenital hereditary condition that can be brought on by either maternal disomy or a partial deletion of the paternal chromosome 15 [69]. Due to coexisting risk factors, such as craniofacial anomalies including micrognathia and a tiny naso-and/or oropharynx, muscle hypotonia that causes airway collapsibility, and obesity brought on by hypothalamic dysfunction and hyperphagia, SDB is frequently seen in children with PWS [70]. With a very varied frequency ranging from 35% to 92.9%, OSA is the most common kind of SDB in children with PWS [70,71,72,73,74,75].

In contrast to healthy controls, this population also commonly exhibits CSA, sleep-related hypoventilation, excessive daytime drowsiness, and altered sleep architecture [3,12]. Other contributing factors to CSA in PWS newborns include brainstem immaturity, hypothalamic dysfunction, and aberrant chemosensitivity to CO2 and O2 [76]. Cohen et al. (2014) reviewed polysomnographic data from 44 children with PWS, including 21 older children with a median age of 5.1 years and 23 babies with a median age of 1.0 years. They discovered that 57% of the children had sleep apnea. While OSA without CSA was less common in newborns (17%) compared to older children (48%, $p = 0.032$), CSA with considerable oxygen desaturation was more common in infants (43%), compared to older children (5%, $p = 0.003$) [70,77]. Pavone et al. reported OSA in 53% of children and 41% of adults with PWS in a large cohort of 88 PWS patients (median age 5.1 years). They found no relationships between polygraphic results and

anthropometric data (age, BMI, BMI z-score). There were no variations in the pattern of SDB across the lifespan, which implies that hypotonia and/or facial dysmorphic traits may be more important causes of SDB in PWS than fat [73].

Short stature, increased fat mass, and decreased muscle strength are the results of early growth hormone (GH) deficiency, which affects more than half of newborns and children with PWS. Growth and body composition have been demonstrated to be improved by GH treatment when started before the age of two [78]. However, because of the documented instances of unexpected death in children with PWS receiving GH treatment, GH therapy necessitates careful monitoring despite its advantages [79]. Elevated GH-induced IGF-1 levels have been associated with a possible increased risk of OSA during GH medication, perhaps inciting tonsillar and adenoid tissue hyperplasia [80]. While a large multicentric research with 112 PWS children in Australia reported no significant rise in the AHI after starting GH medication, other studies have found no significant effect on the frequency of obstructive episodes. However, 3% of children, especially those under three, who had no or mild OSA at baseline went on to develop moderate or severe OSA after treatment. There were no discernible changes in CSA. Current guidelines advise polygraphic screening before and 3–6 months after starting GH medication in children with PWS, albeit these results might be the result of a limited sample size [78].

Chiari Malformations and Sleep Disordered Breathing (SDB)

From modest cerebellar tonsillar ectopia to total herniation of the hindbrain through the foramen magnum, Chiari malformations (CMs) comprise a broad range of clinical diseases divided into four subgroups, each with differing degrees of abnormalities [81]. Sleep disordered breathing (SDB) in patients with CMs can be caused by weakening of the pharyngeal muscles and impairments of the lower cranial nerves, which can lead to obstructive sleep apnea (OSA), or more frequently, by a disruption in the central respiratory drive, which can lead to central sleep apnea (CSA). Furthermore, stridor or apneic episodes may be triggered by the mechanical effects of cerebellar herniation on the medullary respiratory centers [3,12]. With documented incidence rates ranging from 24% to 70% among affected persons, SDB is particularly common in people with Chiari malformation type 1 (CM1) [81,82,83,84]. Surgery, such as foramen magnum decompression, may be required to treat CM1, especially when brainstem compression is present. The best time to do surgery is complicated and depends on a number of variables, such as the patient's clinical history, MRI results, sleep study findings, and symptom presentation [85]. Amin et al. conducted a retrospective study on 68 pediatric patients with CM1 (mean age, 7.33 ± 4.01) and discovered that 49% of the cohort had SDB, especially OSA. They also found a significant correlation between tonsillar herniation as determined by brain imaging and the polysomnography (PSG) obstructive apnea–hypopnea index [83]. The degree of pegged tonsillar structure, dorsal cerebrospinal fluid attenuation, presence of syrinx, and posterior angulation of the dens were among the MRI variants that were more prominent in patients with SDB than in those with normal sleep study outcomes, according to Khatwa et al. (2013), who used PSG and brain MRI to evaluate 22 children with CM1 (median age, 10 years; range, 1–18 years) [82]. In a more recent study, Voutsas et al. examined how decompression surgery affected the outcomes of SDB in 15 children with CM1 and discovered that PSG significantly decreased both obstructive and central episodes after surgery. Nevertheless, 46.7% of patients needed continuous positive airway pressure (CPAP) therapy following surgery because they still met the criteria for SDB [86]. Additionally, CM1 is linked to a number of complicated illnesses or comorbidities. A significant prevalence of SDB, ranging from 50% to 80%, and a notably low incidence of CSA (9%), were described in a recent retrospective analysis comprising 57 children with CM1 (45 with solitary CM1 or concomitant disorders, 5 with CM1 and craniosynostosis, and 7 with CM1 and polymalformative syndrome). The significance of combining MRI results, sleep study findings, and symptom assessment in the treatment of CM1 patients was emphasized by this study [87]. Even after neurosurgical intervention, polysomnography or polygraphy is advised for monitoring SDB in children with CM1, especially if there are neurological symptoms or characteristic MRI abnormalities. This is despite the lack of established recommendations. According to certain research, every child with a CM1 diagnosis should have an SDB screening [81]

Craniofacial Abnormalities and SDB

Craniofacial abnormalities are recognized as risk factors for obstructive SDB, with midface deficiency (such as in Apert syndrome and Crouzon syndrome) and mandibular hypoplasia (e.g., Pierre Robin sequence, PRS) being the most prevalent features in this population [12]. PRS, a congenital condition characterized by micrognathia, glossoptosis, and airway obstruction, can occur in isolation or as part of other syndromic or chromosomal abnormalities. OSA is commonly observed in children with PRS, where airway obstruction is primarily caused by glossoptosis, which leads to narrowing of the oropharynx and/or hypopharynx [88,89,90,91,92]. Van Lieshout et al. reported that the prevalence of OSA in infants with PRS ranges from 46% to 100%, irrespective of symptomatic presentation. The considerable variability in OSA prevalence may be influenced by the type and severity of craniofacial abnormalities, as well as the age of the children at the time of evaluation. During the first two years of life, natural growth of the mandible and upper airway results in gradual improvement in airway obstruction [93]. Moreover, the necessity of continuous positive airway pressure (CPAP) to maintain airway patency may lead to an underestimation of the true prevalence of OSA [90]. Polysomnography and nocturnal oximetry are critical in guiding treatment decisions for patients with PRS [12].

Neuromuscular Disorders and SDB

Duchenne muscular dystrophy (DMD) and spinal muscular atrophy (SMA) are examples of neuromuscular disorders (NMDs) that are linked to respiratory insufficiency, impaired airway clearance, and progressive muscle weakness. Alveolar hypoventilation and irregular gas exchange are caused by respiratory muscle dysfunction. These symptoms first appear during rapid eye movement (REM) sleep and then spread into non-REM sleep stages as the condition worsens, eventually resulting in diurnal respiratory failure. Diaphragmatic or pseudo-central apnea may also be caused by reduced lung volumes while you sleep [94]. In addition to respiratory muscle weakness, other conditions such kyphoscoliosis, obesity (typically as a result of decreased mobility and corticosteroid usage), bulbar involvement, and macroglossia can make upper airway obstruction during sleep worse [95]. Furthermore, central apnea is associated with specific NMDs [96]. Depending on the particular form of NMD and the diagnostic standards applied, the prevalence of SDB in NMD patients varies greatly.

A mutation in the dystrophin gene causes DMD, an X-linked recessive myopathy that affects roughly 1 in 3,600 to 6,000 male babies born. The respiratory and cardiac muscles are among the striated muscles that gradually weaken and deteriorate. Chronic respiratory failure, SDB, recurrent pneumonia, and impaired cough are caused by respiratory involvement [97]. Even in individuals on steroid medication, pulmonary function usually deteriorates early in DMD, with a restrictive spirometric pattern that gets worse during adolescence, especially when scoliosis makes it worse [98]. Both ambulatory and early non-ambulatory children are affected by sleep disorders, especially OSA, which is more prevalent in DMD patients [80]. According to a retrospective analysis of the polysomnographic data of 110 boys with DMD (mean age: 11.5 years), who were receiving steroid treatment, 63.6% of them had OSA, 33.6% had CSA, and 17% had hypoventilation. Age, body mass index (BMI), and BMI z-score all showed favorable correlations with the obstructive index during REM sleep stages [100]. The initial indications of respiratory deterioration are often nocturnal hypoventilation (NH), which tends to deteriorate in the second decade of life [101]. A "red flag" suggesting an elevated risk of nocturnal hypercapnia and a requirement for starting nocturnal non-invasive ventilation is a forced vital capacity (FVC) of less than 50% of expected [102].

As the relationship between spirometry and NH is not well established [102,103], a recent retrospective cohort study of 134 children with DMD revealed that over half of those with FVC < 50% had some level of CO_2 retention as they slept. Of these, 20% had borderline hypoventilation and 30% developed NH. While 28% of children with NH had FVC > 50%, the majority of children with FVC > 50% had normal nighttime gas exchange. According to these results, NH can be detected with an FVC threshold of less than 50% with 73% sensitivity and 86% specificity [104].
Bilateral proximal muscle atrophy and weakening are hallmarks of SMA, another genetic neuromuscular illness brought on by mutations in the survival motor neuron 1 gene. There are three main phenotypes of

the disease (type I–III), and they differ in terms of the severity of the illness and the age at which it first appears [105]. With early diaphragmatic function preserved, respiratory involvement is typified by increasing intercostal muscle weakening [106]. OSA, which is frequently accompanied by NH or a mixed form of sleep apnea, can occur in patients with SMA [80]. According to a 2020 study by Chacko et al., all 31 children with SMA who were not receiving Nusinersen showed signs of SDB, with REM-stage occurrences predominating. Six of the children had SMA I, sixteen had SMA II, and nine had SMA III. It is noteworthy that no child isolated from OSA. Additionally, the study demonstrated that a higher usage of non-invasive ventilation (NIV) improved gas exchange, decreased the apnea-hypopnea index (AHI), and dramatically improved REM-stage sleep [107]. Together with nocturnal gas exchange monitoring, polysomnography continues to be a crucial diagnostic technique for detecting and tracking SDB in children with NMDs because of the low sensitivity of sleep complaints, which are frequently overlooked by these patients and their families [108].

Mucopolysaccharidoses (MPS) and SDB

Defects in the breakdown of glycosaminoglycans (GAGs) cause a class of hereditary lysosomal storage disorders known as mucopolysaccharidoses (MPS), which cause mucopolysaccharides to build up in different tissues [109]. Polysomnography and nasal flow monitoring in afflicted persons have shown that OSA is highly prevalent in these circumstances [110]. Airway narrowing, tonsillar and adenoidal enlargement, macroglossia, and occasionally skeletal abnormalities including short neck and thoraco-lumbar kyphosis are the primary causes of upper airway obstruction (OSA) in MPS patients [111,112]. Hyperactivity, afternoon drowsiness, and snoring are additional typical symptoms. Moderate to severe OSA is frequently prevalent in MPS patients, including those with types I, II, and VI, according to a number of studies; prevalence rates range from 58% to 100% [113,114]. For these patients, prompt CPAP therapy, tonsillectomy, and adenoidectomy are therefore essential, particularly in conjunction with thorough neurosurgical intervention and genetic counseling. In conclusion, there are many different anatomical and genetic defects that contribute to the pathophysiology of SDB in both adults and children. Preventing negative consequences like growth retardation, daytime weariness, and cognitive impairment requires early detection and action. The gold standard for identifying SDB and determining its severity is polysomnography.

Role of Family Medicine and Management Techniques for Pediatric Sleep-Related Respiratory Disorders

Pediatric sleep-related respiratory disorders (SRRDs), including obstructive sleep apnea (OSA), central sleep apnea, hypoventilation syndromes, and other conditions affecting breathing during sleep, represent a significant challenge in the field of pediatric medicine. Family medicine practitioners play a crucial role in the early detection, diagnosis, and management of these disorders, which can have long-term consequences on a child's physical and cognitive development if left untreated. Management techniques involve a multi-disciplinary approach, including medical treatment, behavioral strategies, and coordination with specialists in sleep medicine, pulmonology, and otolaryngology (ENT), among others.

Role of Family Medicine in Pediatric SRRDs

1. **Early Detection and Diagnosis:** Family physicians are often the first healthcare providers to identify signs and symptoms of pediatric sleep-disordered breathing. Symptoms may include loud snoring, mouth breathing, excessive daytime sleepiness, restless sleep, and behavioral issues such as irritability or hyperactivity. These symptoms can be subtle, especially in younger children, which can make early detection challenging. A thorough patient history is essential, including inquiries into parental observations during sleep, developmental milestones, and the presence of any risk factors such as obesity, craniofacial anomalies, or a family history of sleep apnea. The physical exam in family practice may reveal signs such as enlarged tonsils or adenoids, nasal obstruction, or malocclusion, all of which may point to OSA. Family physicians also play a vital role in recognizing other underlying conditions, such as neuromuscular disorders, that could contribute to sleep-related respiratory disturbances.

2. **Referral for Diagnostic Testing:** If a pediatric SRRD is suspected, family physicians will typically refer the child to specialists for further evaluation. Polysomnography (PSG) is the gold standard for diagnosing OSA and other sleep-related respiratory issues, as it allows for the monitoring of various physiological parameters during sleep, including airflow, oxygen saturation, heart rate, and brain activity. Home sleep apnea testing (HSAT) may also be an option in certain cases, though PSG remains the preferred diagnostic tool for complex cases. Family physicians work closely with sleep medicine specialists and pulmonologists to interpret test results and develop a tailored treatment plan.

Management Techniques for Pediatric SRRDs

Management of pediatric sleep-related respiratory disorders requires a comprehensive, multidisciplinary approach that includes medical, behavioral, and environmental interventions. Family medicine plays an integral role in coordinating care, monitoring progress, and providing ongoing support to families.

1. **Medical and Surgical Treatment:**
    - **Continuous Positive Airway Pressure (CPAP) Therapy:** For children diagnosed with moderate to severe OSA, CPAP therapy is the most common medical intervention. CPAP works by delivering a constant flow of air through a mask to keep the airways open during sleep. While CPAP can be effective, its success often depends on the child's adherence to the treatment. Family physicians are essential in educating families about the importance of CPAP therapy, addressing concerns about comfort, and monitoring progress over time.
    - **Adenotonsillectomy:** For many children with obstructive sleep apnea, particularly those with enlarged tonsils and adenoids, surgical removal (adenotonsillectomy) can be an effective treatment. Family physicians work with pediatric ENT specialists to determine the need for surgery and help families understand the procedure, risks, and post-operative care requirements.
    - **Weight Management:** Obesity is a significant risk factor for sleep apnea in children, and addressing obesity through dietary changes, physical activity, and behavioral strategies is an essential aspect of management. Family physicians often collaborate with dietitians and pediatricians to create individualized weight management plans.
2. **Behavioral and Environmental Interventions:**
    - **Sleep Hygiene:** Teaching good sleep habits is an essential part of managing pediatric SRRDs. Family physicians can provide guidance on creating a consistent bedtime routine, reducing screen time before bed, and ensuring a sleep-friendly environment (e.g., dark, quiet room, and a comfortable mattress). Establishing regular sleep patterns can help improve sleep quality and reduce the severity of symptoms associated with sleep apnea.
    - **Position Therapy:** In some cases, children may experience positional obstructive sleep apnea (e.g., apneas that occur when the child sleeps on their back). Position therapy involves teaching parents to encourage the child to sleep in a position that reduces airway obstruction, such as on their side. While position therapy alone is not a cure for OSA, it can be a helpful adjunct in milder cases.
3. **Collaborative Management with Specialists:** Given the complex nature of pediatric sleep-disordered breathing, family medicine practitioners work closely with specialists such as pediatric pulmonologists, sleep medicine experts, ENTs, and even pediatric neurologists, depending on the underlying cause of the disorder. For children with neuromuscular disorders, for instance, collaboration with a pediatric neurologist and respiratory therapist may be necessary to manage central sleep apnea or hypoventilation syndromes. Children with craniofacial abnormalities may require surgical intervention, and family medicine providers can coordinate care between the surgical team and other specialists.

4. **Monitoring and Follow-Up Care:** Ongoing follow-up is crucial for pediatric patients with sleep-related breathing disorders. Family physicians are essential in monitoring the child's progress, managing any complications or side effects of treatment, and adjusting the treatment plan as necessary. Regular follow-up visits may include re-assessment of the child's sleep patterns, evaluation of weight status, and re-evaluation of the effectiveness of CPAP therapy or surgical interventions. Family physicians can also provide emotional and psychological support to families, helping them navigate the challenges associated with managing a chronic condition.
5. **Parental Education and Support:** Educating parents is a key component of managing pediatric sleep-related respiratory disorders. Family physicians should ensure that parents are aware of the symptoms of SDB, the importance of treatment adherence, and strategies for managing the condition at home. Families should also be informed about the potential long-term effects of untreated SDB, such as cognitive impairments, behavioral issues, and cardiovascular problems. Providing support and guidance throughout the treatment process can help reduce anxiety and ensure better outcomes.

Family medicine plays an essential role in the early detection, diagnosis, and management of pediatric sleep-related respiratory disorders. By identifying symptoms, coordinating diagnostic testing, and implementing medical, behavioral, and environmental interventions, family physicians contribute significantly to improving the health and well-being of affected children. Given the potential impact of SDB on a child's development, a multidisciplinary approach to treatment is vital, and family medicine providers serve as a central point of contact for families navigating this complex condition. Through ongoing follow-up, patient education, and support, family medicine ensures that children with sleep-related respiratory disorders receive comprehensive, holistic care that addresses both the physical and emotional aspects of their health.

Conclusion:

Pediatric sleep respiratory disorders, particularly obstructive sleep-disordered breathing (SDB), represent a major health challenge for children across all age groups. The prevalence of these disorders has been reported to range widely, from 1.2% to 13%, with habitual snoring affecting a significant proportion of the pediatric population. The condition typically peaks between the ages of 2 and 8 years due to the growth of lymphoid tissue, which often causes upper airway obstruction. However, with the rise in childhood obesity, a second peak in SDB prevalence is observed in adolescence, reflecting a shift toward the "obese adult phenotype." Risk factors for pediatric obstructive SDB include adenotonsillar hypertrophy, allergic rhinitis, asthma, craniofacial abnormalities, and prematurity. Children with these conditions are at higher risk for developing more severe forms of SDB, such as obstructive sleep apnea (OSA). Studies also indicate that children with asthma or allergic rhinitis are at increased risk for SDB, as chronic upper airway inflammation contributes to the enlargement of lymphoid tissue, exacerbating airway obstruction. Obstructive SDB in children can lead to a wide range of complications if left untreated, including cognitive, behavioral, and cardiovascular issues. The long-term effects of untreated OSA in childhood can significantly impact learning and developmental milestones, leading to school performance issues, increased daytime sleepiness, and behavioral disorders. Furthermore, untreated OSA has been linked to hypertension, cardiovascular disease, and metabolic disorders later in life. Early diagnosis and management are essential to mitigate these risks. Family medicine practitioners play a critical role in the early identification and management of pediatric SDB. By recognizing early signs such as snoring, mouth breathing, and sleep disturbances, family physicians can guide parents toward appropriate diagnostic testing, such as polysomnography, to assess the severity of the condition. Treatment options for pediatric SDB may include lifestyle modifications, such as weight management for obese children, the use of continuous positive airway pressure (CPAP) for more severe cases, or surgical interventions like adenotonsillectomy for children with adenotonsillar hypertrophy. In conclusion, pediatric sleep respiratory disorders are complex conditions that require early recognition and management to prevent long-term complications. Family physicians are integral in providing early detection and intervention, ensuring better health outcomes for affected children. Understanding the epidemiology, risk factors, and available management strategies for

pediatric SDB can help to address the growing prevalence of these disorders and improve overall pediatric health.

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اضطرابات التنفس أثناء النوم لدى الأطفال: الوبائيات، عوامل الخطر، العلاج، ودور طب الأسرة

الملخص:

الخلفية :يلعب النوم دورًا حيويًا في تطور ورفاهية الأطفال. تُعد اضطرابات التنفس أثناء النوم(SDB) ، التي تشمل انقطاع النفس النومي الانسدادي(OSA) ، مصدر قلق كبير في طب الأطفال. يمكن أن يظهر اضطراب التنفس أثناء النوم الانسدادي على شكل شخير أساسي، متلازمة مقاومة مجرى الهواء العلوي، انخفاض التهوية

الانسدادي، أو انقطاع النفس النومي الانسدادي، مع درجات متفاوتة من الشدة. تؤثر هذه الاضطرابات على النمو الجسدي للأطفال، وتطورهم الإدراكي، وصحتهم العامة. غالبًا ما تبقى اضطرابات التنفس أثناء النوم غير مكتشفة، وتزداد نسب انتشارها تعقيدًا بسبب تنوع معايير التشخيص وعوامل الخطر.

الهدف :يهدف هذا الاستعراض إلى دراسة الوبائيات، وعوامل الخطر، والعلاج لاضطرابات التنفس أثناء النوم لدى الأطفال، لا سيما انقطاع النفس النومي الانسدادي، ودور طب الأسرة في معالجة هذه القضايا.

الطرق :تم إجراء استعراض سردي استنادًا إلى بحث حاسوبي في PubMed باستخدام المصطلحات "اضطرابات التنفس أثناء النوم" وَ "الأطفال"، مع التركيز على الدراسات التي تم نشرها خلال العشرين عامًا الماضية. تم تلخيص الدراسات الرئيسية مع التركيز على الوبائيات، وعوامل الخطر، والعلاج لانقطاع النفس النومي الانسدادي في كل من الأطفال الأصحاء والأطفال ذوي الحالات الصحية المعقدة.

النتائج :تتراوح نسبة انتشار انقطاع النفس النومي الانسدادي من 1.2% إلى 13%، ويؤثر الشخير على 7.45% من الأطفال. تشمل عوامل الخطر الشائعة تضخم اللوزتين واللحمية، التهاب الأنف التحسسي، الربو، السمنة، التشوهات الوجهية القحفية، والولادة المبكرة. تم تحديد نوعين رئيسيين: "النمط الكلاسيكي" الذي يزداد بين عمر 2 إلى 8 سنوات، و "النمط السمين البالغ" الذي ينتشر بين المراهقين. يُعد التشخيص المبكر والعلاج أمرًا حيويًا، حيث يمكن أن يؤدي انقطاع النفس النومي الانسدادي غير المعالج إلى مشاكل إدراكية، وسلوكية، وقلبية، وعائية.

الخلاصة :تُعد اضطرابات التنفس أثناء النوم لدى الأطفال قضية صحية شائعة ومهمة. يمكن أن يتراوح انقطاع النفس النومي الانسدادي من الشخير البسيط إلى انقطاع النفس النومي الانسدادي الشديد، ويعد التدخل المبكر أمرًا بالغ الأهمية للوقاية من المضاعفات طويلة الأمد. يلعب أطباء الأسرة دورًا أساسيًا في الكشف المبكر، والتشخيص، وإدارة هذه الاضطرابات، مقدّمين إرشادات حيوية للعائلات.

الكلمات المفتاحية :الأطفال، اضطرابات التنفس أثناء النوم، انقطاع النفس النومي الانسدادي، عوامل الخطر، العلاج، طب الأسرة.