Pediatric Obstructive Sleep Apnea Syndrome

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ABSTRACT

Objective To review evidence-based knowledge of pediatric obstructive sleep apnea syndrome (OSAS).

Methods Review of published articles regarding pediatric OSAS; extraction of clinical symptoms, syndromes, polysomnographic findings and variables, and treatment options; and authors' recommendations.

Results Many complaints and syndromes are associated with pediatric OSAS. This diagnosis should be considered in patients who report the presence of such symptoms and syndromes. Orthodontic and craniofacial abnormalities related to pediatric OSAS are commonly ignored despite their impact on public health. One area of controversy involves the use of a Respiratory Disturbance Index (RDI) to define various pathologies, but apneas and hypopneas are not the only abnormalities obtained on polysomnograms, which can be diagnostic for sleep disordered breathing. Adenotonsillectomy is often considered the treatment of choice for pediatric OSAS. However, many clinicians may not discern which patient population is most appropriate for this type of intervention; the isolated finding of small tonsils is not sufficient to rule out the need for surgery. Nasal CPAP can be an effective treatment option, but it entails cooperation and training of both the child and the family. A valid but often overlooked alternative, orthodontic treatment may complement adenotonsillectomy.

Introduction

Understanding obstructive sleep apnea syndrome (OSAS) in children requires knowledge of the physiology of sleep and breathing. There is an immediate increase in upper airway resistance with sleep onset, with an initial "overshoot" in this resistance that decreases very quickly. Still, this resistance during established sleep is mildly higher than during wakefulness.[1] There is also a slight decrease in tidal volume with sleep. This decrease will be more pronounced with the occurrence of REM sleep. These mild decreases will be compensated by a very slight increase in breathing frequency to keep minute ventilation normal. Breathing frequency decreases during the first two years of life but stays the same thereafter; it has been calculated to be between a maximum of 16 to 18 breaths per minute in NREM sleep and 17 to 19 breaths per minute during REM sleep.[2-3]

The obesity epidemic, evident in the United States and industrialized countries, has complicated the investigation of obstructive sleep apnea and related syndromes. Fat distribution varies depending on genetic, gender, and hormonal patterns as well as the inherent relationship between these three factors. It is common for the fat to deposit in the abdominal region. Such abdominal obesity will lead to chest-bellows impairment, as seen in restrictive thoracic disorders. Although it may not lead to upper airway obstruction, abdominal obesity may worsen the poor gas exchange that may already exist because of OSAS. Sleep will always worsen the gas exchange in these subjects when they are in the supine position and when they achieve REM sleep. During REM sleep, the associated atonia eliminates contractions of the accessory respiratory muscles and the abdominal muscles, which engage in active expiration.[2-3] REM sleep is also associated with further flattening of the diaphragm's position.[2] These physiologic changes worsen gas exchange in subjects with abdominal obesity and may even lead to REM sleep-related hypoventilation with some degree of carbon dioxide (CO_2) retention. Upper airway impairment, per se, is not directly related to this CO_2 retention. It has, however, been hypothesized that abnormal gas exchange during sleep may impair the coordination of time-related contractions of both upper airway dilator muscles and inspiratory muscles.

OSAS was described in children in 1976.[4] Although children may present with OSAS, the literature had established, by 1982, that children had other abnormal respiratory effort patterns during sleep that were frequently associated with snoring and clinical symptoms.[5]

Epidemiology

There is no definitive population-based study evaluating the presence of OSAS in children. Previous studies were performed in different settings and implemented a variety of tools. Some considered regular nocturnal snoring as a marker of chronic obstructive breathing during sleep. The percentage of individuals younger than 18 years of age who have been reported with regular heavy snoring oscillated between 8 and 12%. Other studies polygraphically monitored subjects but were limited in terms of sample size and testing difficulties; initial studies estimated OSAS prevalence between 1 and 3%.[6-15] More recently, many specialists have quoted the OSAS prevalence between 5 and 6%. Although better monitoring techniques during polysomnography have shown that more abnormal breathing events are present,[16] the definitive data are still lacking.

Clinical symptoms

Abnormal narrowing in the nose, nasopharynx, oropharynx, or hypopharynx causes abnormal air exchange during sleep, which in turn leads to clinical symptoms. These symptoms will vary with age. Recognition of the problem is often only noted in older children, who are able to articulate complaints. Table 1 (A-D) indicates the parental complaints of children seen at sleep clinics over time.[17-25]

Abnormal breathing during sleep has been associated with specific clinical problems and findings. The clinical interview of a child suspected of having sleep disordered breathing (SDB) must lead to systematic questioning of the parents regarding their child's symptoms; the parents may not associate the occurrence of these symptoms with abnormal breathing during sleep. Table 2 outlines syndromes that have been shown to be related to SDB and are subsequently controlled after the appropriate treatment of the breathing disorder has been initiated.[20, 24, 27-51] Interestingly, some of the syndromes are related to the maxillomandibular development and are more connected to

orthodontic practice. Pediatricians do not traditionally consider orthodontic problems as part of a child's health issues, but in light of the related health cost and syndromic association, they should.

Clinical Evaluation and Diagnosis of Sleep Disordered Breathing

SDB in a child will be suspected based on the parental complaints. The presence of one of the syndromes listed in Table 2 should lead to a thorough interview of the behavior during sleep as well as sleep-related factors associated with SDB.[17-50]

The suspicion of SDB indicates the need for not only a general pediatric evaluation but for a thorough evaluation of the upper airway anatomy. Clinically, it involves a comprehensive examination of its successive segments. Starting with the nose, one should look for asymmetry of the nares, a large septal base, collapse of nasal valves during inspiration, a deviated septum or enlargement of inferior nasal turbinates (Figure1). Next, the oropharynx should be examined for the position of the uvula in relation to the tongue. The Mallampati scale may help evaluate this position.[52] The size of the tonsils should be compared to the size of the airway; application of a standardized scale is useful.[53] The presence of a high and narrow hard palate, overlapping incisors, cross-bite, and an important (> 2 mm) overjet (the horizontal distance between the upper and lower teeth) are indicative of a small jaw and/or abnormal maxillomandibular development. This clinical evaluation provides important details of the upper airway anatomy and identifies anatomical risk factors which can predispose one to developing abnormal breathing during sleep.

The results of this examination must be summarized, as the different anatomical narrowings have additive effects. The apparent sizes of tonsils and adenoids are not the only anatomical findings which determine whether or not sleep disordered breathing is present. Change in flow due to an abnormal nose, secondary development of turbulence and the increased collapsibility at specific vulnerable points in the upper airway are elements to consider.

There is a complex interaction between nasal breathing and maxillomandibular growth. Abnormal nose breathing in very young individuals leads to an increase in nasal resistance and

mouth breathing with secondary impairment of maxillomandibular growth[54-62] as shown experimentally in young rhesus monkeys.[63] The first four years of age are of particular importance, as 60% of the adult face is built during that period.[64] Otolaryngologic and orthodontic data have clearly demonstrated the impact of enlarged tonsils, adenoids, enlarged nasal turbinates, and upper airway allergies on orofacial growth in children.[21,55--70]

Other factors may be considered. Neck circumference and the presence of fatty infiltration should be noted, but no scale correlates neck circumference with age or pathology. The overall aspect of the face can be appreciated. The frontal aspect of the face can be subdivided into superior, middle, and inferior portions. They are approximately the same length in a normal child. The upper part of the bridge of the nose and the part just below the nare represent the middle third of the face. In individuals with a maxillomandibular risk factor for OSA, the lower third of the face may be longer than expected. The terms “long face” and “long face syndrome” have been used.[21, 26]

Objective confirmation of SDB

Testing during sleep is the only way to affirm the presence of SDB. There is controversy over the need for and type of test to be performed. Some of the measures employed for this testing include questionnaires and scales, home monitoring, and polysomnograms (PSGs).[71-74]

Questionnaires with specific emphasis on the common symptoms associated with SDB have been implemented. Although questionnaires may be helpful in directing the attention of parents to the diurnal and nocturnal symptoms of SDB, the sensitivity and specificity of questionnaires are not sufficient for affirming the presence of SDB.[23,75-77]

Home monitoring with or without videotaping has also been used. Ambulatory monitoring with recording of cardiac and respiratory variables has been suggested as the first diagnostic step in testing for SDB. These devices can detect the presence of oxygen saturation (SaO_2) drops, apneas, and hypopneas; affirm the diagnosis of SDB; and lead to treatment. Associated videotaping may confirm abnormal breathing behavior. This approach may recognize severe SDB but fails to identify

the presence of associated sleep disorders and partially obstructed breaths. A negative test does not rule out the diagnosis of SDB and must be followed by a PSG; however, a positive finding may lead to faster treatment.[78-80]

A PSG is the only test that may exclude the diagnosis of SDB. It must always include monitoring of sleep/wake states through electroencephalogram (EEG), electro-oculogram, chin and leg electromyography (EMG), ECG, body position, and appropriate monitoring of breathing. Nasal cannula-pressure transducer, oral thermistor, chest and abdominal belts, neck microphone, and pulse oximetry are recommended, but variable montages are used.

Respiratory efforts can be investigated by a variety of means during the PSG. Although infrequently used, the best approach involves measuring esophageal pressure (Pes) movements. A less reliable approach is to monitor intercostal/diaphragmatic EMG. A recently developed analysis of this signal appears promising but needs further testing in children.[81] CO_2 may be monitored using a nasal cannula with measurement of end-tidal CO_2. But the combination of two cannulas in the nose of a child may disturb sleep and negatively impact nasal breathing; thus, a transcutaneous CO_2 electrode will often be needed for this measurement.[16, 82-83]

SDB has consequences related to the repetitive changes induced by a decrease in size of the upper airway during sleep. As a compensatory first step, there will be an increase in breathing frequency (tachypnea) and an increase in respiratory efforts.[5,84-85] The selected response is related to both the decrease in size of the upper airway as well as the age of the subject. Following the classic "breathing frequency × tidal volume = minute ventilation," tachypnea is a more common finding in young children with small and relatively unstable chests; this population has mild to moderate breathing impairment during sleep.[5, 84] Despite better chest stability, this response will also be seen in older children. Tachypnea and an increase in inspiratory efforts have been seen in the same children in association with airflow limitation. The mechanisms behind a specific response and the relationship with sleep state are unknown.

The repetitive challenges resulting from a reduction of upper airway size have negative consequences on a child's well-being. However, the normative data for many of the studied variables are still unclear. The polygraphic normative data on sleep duration and sleep stages are available in children aged 7 years and older.[86] But the frequency of short arousals during sleep (i.e., EEG arousals lasting ≥ 3 seconds that can be reliably scored by 3 years of age[87]) is unknown for different age group. But abnormal breathing patterns during sleep have been identified (Table 3).[85]

Interpretation of the PSG

There are controversies concerning PSGs,[71,84] because many existing criteria are based on information obtained from small studies. Other recommendations were taken "by consensus," which means they were not necessarily based on data; still others were based on information collected with outdated technology. The specificity and sensitivity of many indices used have never been calculated. Only one study has looked at polygraphic respiratory patterns; their frequency of occurrence; their change in frequency with treatments; and the impact on the clinical outcome associated with polygraphic changes in pre-pubertal children.[84]

One of the most debated issues today is what type of respiratory event should be scored and tabulated. Another issue is determining when "pathology" is present.[23,76,84] Historically, the presence of OSA was easy to recognize with simple albeit relatively insensitive equipment (thermistors). Based on the variability of breathing frequency between birth and two years of age, an "apnea" was defined as "longer than 2 breaths." For many years, there was a consensus that OSA, a complete cessation of air exchange at nose and mouth, was associated with clinical symptoms. It was shown that removal of the obstructive apnea (OA) led to improvement of the symptoms. Initial criterion for an abnormal finding with polysomnography was "≥ 1 OA/hour of sleep."[88]

But pathology was also seen without complete absence of air exchange. To improve the scoring system, clinicians used the term "hypopnea," but there is no consensus of what a "hypopnea" is. Following adult criteria and utilizing thermistors with limited sensitivity, clinicians

suggested that a "hypopnea" should last "longer than 2 breaths." Also, the airflow signal from the combined nasal/oral thermistors should decrease by at least 50% of normal baseline breathing. Hypopneas should be terminated with either an EEG arousal or a drop of SaO_2 of at least 3%.[23, 76] Using these criteria, pathology was considered to be present if the OA index was ≥ 1 or if the AH index (AHI) was ≥ 5 events/hour.

Some children with very noisy breathing at night and enlarged tonsils and adenoids had a normal score at PSG, but had clinical symptoms[89,90] that led to adenotonsillectomy. Also, other sleep-disordered breathing syndromes without an associated abnormal AHI but with an elevated "respiratory disturbance index" were controlled with nasal CPAP or upper airway surgery.[85] Although an AHI ≥ 5 was considered pathological, there was the recognition that "apnea and hypopnea" as defined did not encompass all pathological breathing during sleep. Hence, an arousal index was calculated; thus, snoring sequences which were terminated with an EEG arousal were scored. The association of apnea-hypopnea and other measurements led to the usage of the term "respiratory disturbance index (RDI)." This term acknowledges that the defined PSG patterns did not encompass all abnormal breathing events.

The introduction of the nasal cannula/pressure transducer system[16,91] allowed a more accurate recognition of abnormal breathing during sleep, as this technique based on nasal flow is semi-quantitative. It allows better recognition of partially obstructed breaths. A "hypopnea" was defined when flow decreased by 30% of a normal breath. But many still require an "EEG arousal" and/or an SaO_2 drop, despite prior demonstration that clinical consequences can be obtained without a change in SaO_2. An RDI > 5 events was used based on prior habits.

A minority of sleep clinics monitored respiratory efforts using esophageal pressure (Pes). These groups showed that snoring without "hypopneas" was associated with abnormal efforts and an induction of EEG arousals. Based on Pes recordings,[83,85] specific patterns were recognized and defined, such as "Pes crescendos," "sustained respiratory effort," and "Pes reversals." Some evidence suggests that these patterns were frequently, but not always, seen with abnormal nasal

flow on the nasal cannula/pressure transducer recording. But a "flow limitation" between normal and a 30% decrease at the nasal cannula was usually seen with these patterns. The nasal flow limitation was described as a "flattening" of the nasal flow curve; several patterns of abnormal curves have been described. It may be easier to visually recognize a change of the Pes pressure than a "flattening of the nasal curve."[83-85]

Application of these Pes-related definitions showed that children who had no apneas/hypopneas, SaO_2 drop of 3% or more, or EEG arousals presented with clinical complaints and clinical sleep-related syndromes, primarily parasomnias.[32-33] Applying the above criteria, a clinical outcome study performed at the Stanford University Sleep Disorders Clinic focused on clinical complaints and the presence of clinical symptoms and signs. Complete treatment of the sleep-related upper airway problem with resolution of symptoms and signs was associated with less than one of the events included in the RDI.[84] Persistence of symptoms and signs was associated with the continued presence of an "event" that was not necessarily an "apnea" or a "hypopnea." Instead, the breathing event was noted to be either a "flow limitation with an increase in respiratory effort" or merely an increase in respiratory effort; a cut-off point for RDI at ≥ 1.5 events/hour of sleep was found.[84] But an RDI ≥ 1.5 events/hour is based on only one outcome study, even if several clinical studies have indicated the validity of such a cut-off point.[32,45,84]

Changes in autonomic nervous system (ANS) and breathing patterns during sleep

An increase in respiratory efforts is associated with changes in ANS settings. These changes will affect the cardiovascular system in an individual with a normal ANS.[49] One may want to evaluate these changes to recognize an abnormal pattern and determine if they may be detrimental. Two types of responses can be seen when an increase in respiratory effort occurs during sleep: "activation" or "arousal with cortical involvement."

"Activation" is a clinical neurophysiology term defined by Moruzzi[92] during the course of his study of the "ascending reticular formation;" it is related to the recruitment of sensory inputs

that will lead to a polysynaptic motor response after relay of sensory input in the brainstem and subcortical structures. The nucleus ambiguous receives information that simultaneously leads to efferent responses through the nucleus tractus solitarius (NTS). This relay leads to a simultaneous ANS stimulation, and an autonomic activation will lead to an increase in sympathetic tone.

An ANS response may be seen with brainstem reflexes leading to full reopening of the upper airway without EEG cortical arousal, or it may be seen as the consequence of an EEG cortical arousal. The presence of cortical arousals will be associated with clinical symptoms, e.g., complaints of excessive daytime somnolence, irritability, or unrefreshing sleep. The role of repetitive "activation" is unknown in children.

The determination of how much airway size change and the duration of the change needed to lead to cortical arousal are unknown. Sleep stages may play a role in the type of response seen, but no definitive information is available in prepubertal children.

The pulse transmit time (PTT), which measures the transit time of the pulse wave from approximately the aortic valve to the wrist, and the peripheral arterial tonometry (PAT), are two variables that were added to polysomnography to help recognize an "arousal".[93-96-] None of these devices can distinguish between a brainstem reflex and a cortical arousal response. The importance of the sympathetic response could be a relatively accurate indicator of cortical involvement, but the studies to validate such distinction have not yet been published. Based on a commercially designed algorithm involving both heart rate and finger plethysmography, the PAT does not really reflect the balance between the sympathetic and parasympathetic systems during sleep. The PTT also has limitations of interpretation. When used to identify cortical arousals related to SDB, both techniques have false positives and false negatives, which limit the accuracy of interpretation.[96] Monitoring of these different variables has, however, shown that repetitive snoring can be associated with activation and/or EEG arousal.

Changes in EEG sleep patterns with re-opening of upper airway

Historically, an EEG alpha or alpha and beta arousal lasting three seconds at the termination of an abnormal breathing event was requested to score an "event." But several studies have shown that limited upper airway occlusion may end with a burst of delta waves or a K complex.[97] The usage of a sleep scoring system, based on analysis of the "cyclic alternating pattern" (CAP), demonstrated the negative effect of these bursts.[98-100] The CAP scoring system is based on recurrent bursts of delta and K complexes with or without superimposed alpha waves within a time period of 60 seconds intertwined with low EEG amplitude. CAP is a normal phenomenon that occurs between wakefulness and slow wave sleep (SWS) or, at the end of night, between REM sleep and well-established, repetitive sleep-spindle sleep. It indicates a transition from one stable state to another stable state and is not seen in REM sleep. CAP is typically a transient period during which a greater instability of sleep may occur with greater chance to enter a light sleep or even to awaken. An abnormal CAP rate, defined in different age groups in children,[99-100] indicates an "instability of NREM sleep" as well as a difficulty in reaching a new stable state.[98] CAP is associated with autonomic activation and may lead to awakening and large sympathetic discharge. Chervin *et al* [101-102] have also reported a novel approach to evaluate EEG with abnormal breathing during sleep, based on an algorithm investigating the EEG changes seen with each abnormal inspiration associated with increased effort. The algorithm recognizes the changes in brain wave activity with increased inspiratory effort. When adenotonsillectomy is successful in relieving abnormal breathing during sleep, the abnormal EEG pattern disappears. Furthermore, the daytime symptoms, particularly sleepiness, abate. This analytic technique needs to be tested further.

Genetic Risk Factors of SDB

Both genetic[64,103 -111] and environmental risk factors have been identified in the development of SDB; they are associated to variable degrees. Oral mucosa thickness has been identified as an ethnic risk factor in African-Americans, and skull base length has been noted to be an ethnic risk

factor in Far East Asians. [-107-108] African-American and Far East Asian populations have been shown to have significantly higher risk than Caucasians when age, sex and BMI were considered.[64,103-111] The familial trait of dolichocephaly (or narrow face) has also been implicated as a risk factor, independent of ethnicity.[51,110] Familial cases of SDB are seen in all ethnic groups. Genetic investigations are performed although there is currently no clear indication for a specific gene location responsible for increased risk. The strongest current indicators have been related to facial morphotype.[64] Clearly, there is an increased risk of SDB in a family in which a member is affected.[103-105,109-111] Pediatricians should, therefore, systematically question other family members about sleep-related problems when there is a positive history of SDB (Figure 2).

Treatment

There is an overall consensus that children with SDB should be evaluated by an otolaryngologist for surgical treatment. It is also clear that the well-described but extremely complex interaction between nasal breathing and facial growth is important, even if rarely investigated.[51]

Treatment for short-term outcomes indicates that adenotonsillectomy with or without radiofrequency (RF) treatment of nasal inferior turbinates is the first approach to consider.[85,112-113] Independent of the size of tonsils or adenoids, adenotonsillectomy will provide more airway space. Two points must be emphasized. First, outcome investigation has shown that isolated tonsillectomy or adenoidectomy is not as effective as adenotonsillecomy.[84,114] Also, RF of the nasal turbinates should be performed at the same time as the child is under general anesthesia if enlarged turbinates are present. Performance of adenotonsillectomy without performance of nasal turbinate treatment may negatively impact the outcome.[84] Outcomes of adenotonsillectomy have been reviewed,[112] but no review addresses the reasons for failure. A recent study examined the short-term outcomes to understand why results were incomplete.[114] Surgeons often utilize techniques that are not aimed at maximally opening the airway; they may fail to treat the nose simultaneously with

adenotonsillectomy; and others simply do not recognize the craniofacial changes that contributed to the sleep disordered breathing.

Only two studies have looked at the long-term outcome of regular adenotonsillectomy performed in prepubertal children.[115-116] Evaluating outcome to a minimum of 10 years later, both studies indicated that there was failure to control the problem due to the presence of hypopneas and apneas at the long-term follow-up recordings. Demonstration of absence of apnea-hypopneas within 6 weeks to 3 months after surgery was requested in one of the two studies.[115] The long-term outcome in that study linked the recurrence of abnormal breathing during sleep to both the absence of dealing with a narrow maxilla and/or mandible at the time of the initial surgery as well as the later occurrence of tongue/mucosal enlargement at the time of puberty, when 90% of oro-facial adult growth had already occurred.

Association of adenotonsillectomy with orthodontic treatment has been done.[117] Rapid maxillary distraction (RMD) is an orthodontic technique that is based on bone formation process. A distractor anchored on two molars on both sides applies daily pressure, pushing apart the two half of the maxilla; bone grows from the borders of the cartilage.[117-118] This technique pushes the soft tissues laterally, decreases the height of the soft palate and enlarges the nasal orifices.[117] RMD may be associated with distraction of the mandible, but as no mid-cartilage is present, there is very limited widening. This fact may limit the degree of maxillary widening with RMD (Figures 1, 3 and 4). Slow maxillary distraction is based on similar principles and optimizes the degree of widening at the different growth periods that occur in prepubertal children. Both rapid and slow maxillary distractions are performed between 5 and 11 years of age. Distraction results in widening of both the palate and the nose; thus, this procedure remedies nasal occlusion related to a deviated septum, for which little can be done before 14-16 years of age. But even in association with adenotonsillectomy, orthodontics may not control all SDB. Abnormal mandibular or maxillomandibular anteroposterior development is a bigger challenge. Nasal CPAP will be the recommended treatment until further orthognathic surgery[119] can be performed.

Home nasal CPAP has been used in infants, pre-pubertal children, and pubertal children. The first report of its usefulness in children in 1986 was a prospective study that followed 5 children, age 3 to 11 years, for 10 months.[120] Similar findings were reported in several large retrospective studies.[110,121-126] These studies primarily involved children older than 12 months of age. Infants 8 to 18 weeks of age were followed from the onset of treatment through the first 12 months of age in a study in 1995;[123] this was replicated in 1999.[125]

The difficulty in application of nasal CPAP relates to training of the family and child as well as finding the appropriate nasal interface. Children often need to be trained to tolerate the facial interface; behavioral modification techniques and daytime training may help with this training. CPAP is very useful when the SDB is related to major craniofacial deformities or other illnesses. If the upper airway problem is complicated by neuromuscular disease, nasal bilevel may be used.

Regular follow-up should be performed within the first three months to evaluate mask fitting.[123] Due to rapid craniofacial growth of young children, CPAP pressure should be evaluated every 6 months. An annual craniofacial specialist visit should occur to affirm that headgear and mask do not worsen a maxillary growth deficiency.[127] Clinicians should encourage the use of humidification, aggressively treat allergies and/or rhinitis and check nasal patency. In light of children's favorable response to surgery with or without orthodontic and anti-allergic treatment, nasal CPAP should only be a second consideration.

Orthognathic surgery entails shifting bones and disrupting the bone growth structures.[119] Such an approach is normally postponed until 10-13 years of age. Two surgical techniques used in SDB patients include mandibular distraction osteogenesis and maxillomandibular advancement.

Mandibular distraction osteogenesis is very similar to RMD, but it is applied to the mandible when a maxillary and a mandibular widening are needed and when the slow mandibular orthodontic distraction cannot achieve the needed result.[119] A vertical transection of the maxilla is performed between the 2 central incisors, and a distractor is used as in RMD. Twelve to 14 mm of widening can easily be obtained in three weeks. Orthodontic treatment is similar to the one described with

RMD. At that age, both procedures can be simultaneously performed to provide an anterior displacement of the tongue and enlargement of the retro-lingual airway space.[119]

Maxillomandibular advancement is a very successful procedure. Nevertheless, it is a major surgery that should be performed after there has been appropriate orthodontic treatment. Surgeons who perform this procedure must have a good understanding of upper airway mechanics as well as dental problems. It may be performed at any time during childhood, but it is often post-pone till 11-12 years of age.

A controversial issue is how early to perform adenotonsillectomy. Most will agree that adenotonsillectomy is often performed by 24 months of age. But OSA has been noted as early as three weeks of age, and cases of heavy snoring and clinical symptoms in children aged 6 to 24 months are actually common. Adenotonsillectomy has been performed as early as 6 months of age.[128]

Several advances have been made in sleep medicine. Apneas and hypopneas are not the only indicators of abnormal breathing during sleep. But in this rapidly evolving field, it has been challenging to establish new scoring criteria, despite the availability of new technologies. However, the clinical findings and the polysomnographic results should be used to determine the diagnosis and guide treatment recommendations.

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Table 1: Complaints reported by parents regarding their children

A. Infants (3-12 months)	B. Toddlers (1-3 years old)	C. Preschool children	D. School children
Disturbed nocturnal sleep with repetitive crying	Noisy breathing or snoring	Regular, heavy snoring	Regular, heavy snoring
Poorly established day/night cycle	Agitated sleep or disrupted nocturnal sleep	Mouth breathing	Agitated sleep
Noisy breathing or snoring	Crying spells or sleep terrors	Drooling during sleep	Abnormal sleeping positions
Sweating at night	"Grouchy" and/or aggressive daytime behavior	Agitated sleep	Insomnia
Poor suck	Daytime fatigue	Nocturnal awakenings	Delayed sleep phase syndrome
Absence of normal growth pattern or failure to thrive	Nocturnal sweating	Confusional arousals	Confusional arousal
Observation of apneic events	Mouth breathing	Sleepwalking	Sleepwalking
Report of ALTE	Poor eater or failure to thrive	Sleep terrors	Sleeptalking
Presence of repetitive ear aches or URI	Repetitive URI	Nocturnal sweating	Persistence of bed-wetting
	Witnessed apneic episodes	Abnormal sleep positions	Nocturnal sweating
		Persistence of bed-wetting	Hard to wake up in the morning
		Abnormal daytime behavior	Mouth breathing
		a. aggressiveness	Drooling
		b. hyperactivity	Morning headache
		c. inattention	Daytime fatigue
		d. daytime fatigue	Daytime sleepiness with regular napping
		Hard to wake up in the morning	Abnormal daytime behaviors
		Morning headache	a. Pattern of attention deficit hyperactivity disorder
		Increased need for napping compared to peers	b. Aggressiveness
		Poor eating	c. Abnormal shyness: withdrawn and depressive presentation
		Growth problems	Learning difficulties
		Frequent URI	Abnormal growth patterns
			Delayed puberty
			Repetitive URI
			Dental problems appreciated by dentist
			a. Cross-bite
			b. Malocclusion (Class II or III)
			c. Small jaw with overcrowding of teeth

Abbreviations: ALTE, Apparent Life-Threatening Event; OSAS, obstructive sleep apnea syndrome; URI, upper respiratory tract infection.

Table 2: Syndromes related to abnormal breathing during sleep

Chronic snoring
Daytime fatigue
Daytime sleepiness
Sleep maintenance insomnia
Sleep phase delay
Confusional arousal
Sleep talking
Sleep terror
Sleepwalking
Enuresis (primary or secondary)
Morning headache
Nocturnal migraine
Periodic limb movement
Learning or memory problem
Attention deficit hyperactivity disorder
Abnormal social contact (psychologically withdrawn)
Depressive affect
Hypotension with orthostasis
“Fainting” (rare)
Hypertension (rare)
Cor pulmonale (rare)
Nocturnal asthma or nocturnal wheezing
Cross-bite
Pathological overjet
Overcrowding of teeth
Impacted wisdom teeth

Table 3: Abnormal breathing patterns

Term	Definition
Apnea	Absence of airflow at nose and mouth for longer than 2 breaths, independent of desaturation or change in EEG. Subdivision in central, mixed or obstructive based on airflow and Pes recording.
Hypopnea	Reduction by at least 50% in nasal flow signal amplitude for a minimum of 2 breaths. Scored independently from SaO2 drop or EEG arousal. Often but not always associated with snoring.
Abnormal respiratory effort	Reduction in nasal flow of less than 50% with flattening of nasal cannula signal (flow limitation)[7] and decrease in the mouth signal (thermistor). Often seen with snoring and increased effort shown on Pes signal defined as:
Pes Crescendo[8]	Sequence of 4 or more breathes that show increasingly negative peak end inspiratory pressure. May be seen with flow limitation on nasal cannula.
Continuous sustained effort[9]	Repetitive, abnormally negative peak end inspiratory pressures, ending at same negative inspiratory pressure without a crescendo pattern. Associated with discrete flow limitation on nasal cannula/pressure transducer signal, with "flattening" of the breath signal curve for at least 4 successive breaths.
Pes Reversal[8]	Termination of abnormal increase in respiratory effort with abrupt switch to a less negative peak end inspiratory pressure.
Respiratory Event Related Arousals (RERAs)	Patterns of progressively negative pressure terminated by both a sudden change in pressure to a less negative level as well as an arousal event lasting 10 seconds or more.[129]
Tachypnea	Increase in respiratory rate, above that seen during quiet unobstructed breathing, by minimum of 3 breaths/minute in NREM sleep, or 4 breaths/minute in REM sleep, for 30 seconds or more. No changes in oxygen saturation, Pes or EEG were required.[11]

Table 4: Nasal CPAP in children

Author Name	Type of Study/# of subj	PSG	Conclusions
Guilleminault et al. 1986	+Feasibility study 5 children in hospital +Prospective home study (10 months) 5 children	PSG done before, during titration, and during follow up (F/U)	Feasibility with parent training +4/5 infants daily usage of CPAP at 10 months F/U
Waters et al. 1995	Retrospective review 80 children 12 days / 15.5 years	PSG for diagnosis and titration	86% completed of training of there terminate training 12.5% drop out
Marcus et al. 1995	Retrospective study 94 children (3-12 months of age) applied post T&A in 76% 1st treatment in 23 children	PSG for diagnosis and titration	1 drop out
Guilleminault et al. 1995	Prospective study 8-18 weeks of age at entry-systematic follow up for 12 months. Family screened at entry for understanding of treatment	PSG for diagnosis, treatment, and each F/U retitration	Need to readjust equipment and pressure on regular basis due to fast craniofacial growth in infancy
Rains 1995	Prospective study 4 children (3-12 years) training of parents	PSG for diagnosis, and titration	Follow up for 3 months Effective treatment No drop out for 3 months/ 1 after
McNamara et al. 1999	Prospective study 24 infants (6 weeks to 51 weeks) for 12 months	PSG for diagnosis, titration, regular follow-up	Family training and support Continuous usage in 18 children Effective treatment
Downey et al. 2000	Retrospective study 18 children < 2 years of age		12 children successfully treated

Figure 1: A seven-year old child illustrates many anatomical abnormalities, including asymmetry of the nares, an enlarged septal base, large medial crus, deviation of the septum to the right, and a narrow and high-arched palate. A rapid maxillary distractor has been placed in order to widen the maxillary cavity, decrease the height of the soft palate, and enlarge the bony aspects of the nose

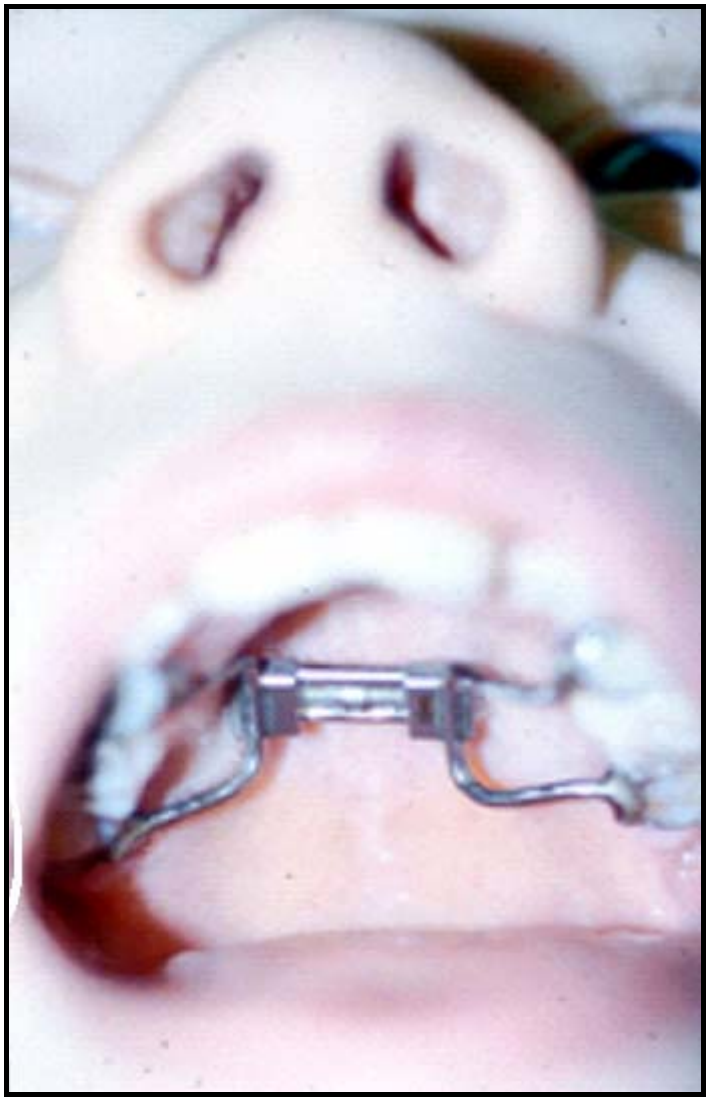

Figure 2: Influences on oro-facial growth

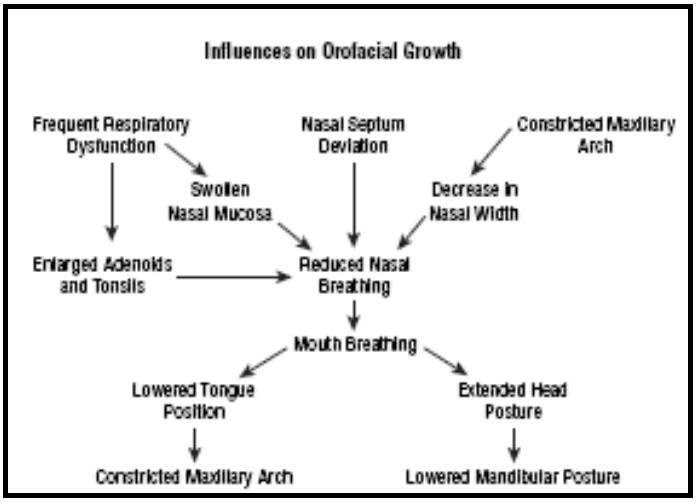

Figure 3. The significant impact of rapid maxillary distraction. A, Frontal cephalometric study demonstrates a narrow maxillary arch before distraction. B, Drawing superimposing the image in A and the postdistraction image (shown in C) to show the widening of the maxillary and nasal cavities.The patient's right inferior turbinate is closely approximated to the septum. C, The frontal cephalometric demonstrates that the maxillary arch has been opened since the distractor has been placed. The nasal cavity has also been altered because the patient's right inferior turbinate is now farther from the septum than it was before placement of the distractor.

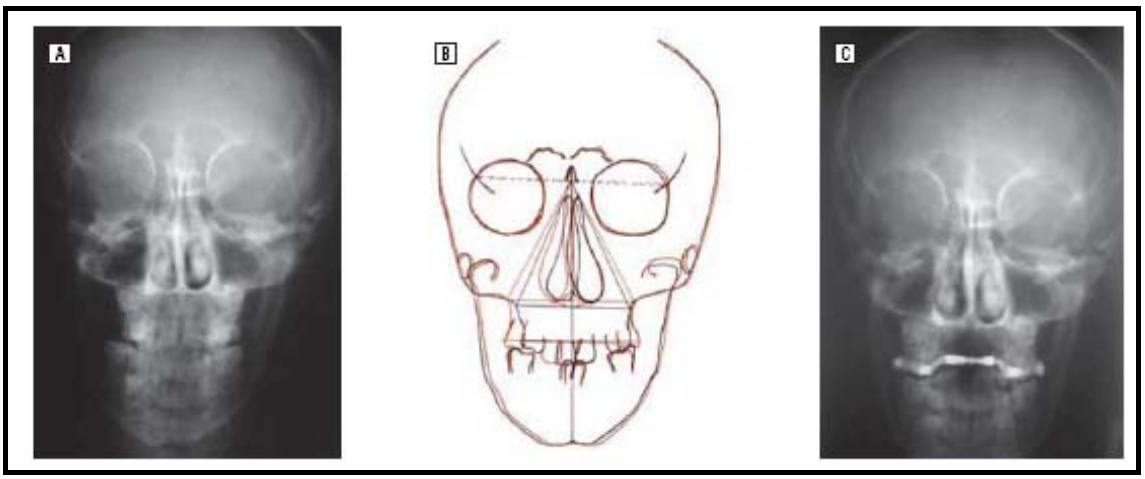

Figure 4: Rapid maxillary distraction demonstrates progressive improvement in the crowding of this child's teeth; one can see the progressive widening indicated by the space in between the two frontal incisors.