Sleep-disordered Breathing in Children

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Children with sleep-disordered breathing (SDB) can manifest a continuum from simple snoring and upper airway resistance syndrome to obstructive sleep apnea (OSA) with secondary growth impairment, neurocognitive deficits, and less often cardiovascular sequelae. Most children who present with SDB are four to eight years old with variable clinical symptoms at different ages. In general, infants often present with noisy breathing and disturbed nocturnal sleep, toddlers and preschool-aged children with snoring and mouth breathing, and school-aged children with behavioral and dental problems. The pathogenesis of SDB in children remains incompletely understood. Adenotonsillar hypertrophy is the leading cause of OSA. Other risk factors include allergic rhinitis, craniofacial anomalies, cleft palate following pharyngeal flap surgery, neuromuscular diseases, laryngomalacia, and obesity. Polysomnography (PSG) is the gold standard diagnostic tool. However, great variation exists in the interpretation of PSG and criteria for the definition of pediatric OSA, even though consensus statements have been used to standardize the scoring of summary indices for the disorders. Adenotonsillectomy is the cardinal treatment for pediatric SDB. Rapid maxillary expansion is a useful approach in upper jaw contraction. Distraction osteogenesis has become an acceptable procedure in the treatment of severe maxillomandibular deficiency. Continuous positive airway pressure has been successful in treating intractable or severe OSA in children with other underlying medical disorders and has modified the indications for tracheotomy in pediatric patients with craniofacial anomalies and OSA. Follow-up in children treated for OSA reveals that underlying structural or neuromuscular abnormalities can decrease the response to treatment and obesity may lead to recurrence of OSA later during adolescence. *(Chang Gung Med J 2009;32:247-57)*

Key words: sleep-disordered breathing, obstructive sleep apnea, adenotonsillectomy, rapid maxillary expansion, orthodontic procedure, continuous positive airway pressure

Sleep-disordered breathing (SDB), a disease spectrum, varies from partial upper airway obstruction (such as snoring and upper airway resistance syndrome [UARS]) to complete upper airway obstruction (obstructive sleep apnea [OSA]).(1) The spectrum in children can occur throughout childhood from infancy to adolescence. In 1976, Guilleminault et al.(2) first used polysomnography to describe

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obstructive sleep apnea syndrome in children as a combination of clinical symptoms and evidence of obstructive apneas. Since then, the criteria for OSA in children have been refined.[3-5]

Symptoms of pediatric SDB vary and specialty referral is often done according to symptoms noted by parents. For example, a child with snoring and tonsillar hypertrophy is most likely to be referred to an otolaryngologist, a child with growth impairment to a pediatrician, and a sleepy child to a neurologist. The clinical presentation of SDB in children is different than in adults. Children tend to have fewer nighttime symptoms since their obstructive spells are prone to be brief and periods of arousal less obvious. Likewise, they present with more subtle behavioral changes in the daytime and do not have the degree of daytime somnolence seen in adults.[6] Unlike adults, children with SDB tend to be of normal weight or thin and may fail to thrive. Boys and girls are equally affected in this age group.[7] Table 1 summarizes differences between children and adults with SDB. Although SDB is relatively common in children, and can result in significant impact on development, it is not well understood. This review article will concentrate on SDB in children rather than that seen in infants and discuss characteristics of pediatric SDB individually.

Table 1. Clinical Differences in Sleep-disordered Breathing between Children and Adults

Variables	Children	Adults
Sex distribution	Male: Female = 1:1	Male: Female = 8:1
Weight	Underweight	Commonly obese
Snoring	Continuous	Intermittent with pause
Mouth breathing	Common	Less common
Chief complaint	Snoring, difficult breathing	Daytime sleepiness
Enlarged tonsils/ adenoids	Common	Uncommon
Obstructive pattern	Mostly apneas	Mostly hypopneas
State with most obstruction	REM	REM or non-REM
Clinical arousal	Uncommon	Common
Sleep architecture	Preserved	Fragmented
Sequelae	Behavioral changes Neurocognitive deficits	Daytime sleepiness Cardiovascular disease
Primary treatment	Adenotonsillectomy	CPAP therapy

Abbreviations: SDB: sleep-disordered breathing; REM: rapid eye movement; CPAP: continuous positive airway pressure.

Diagnosis/definition

Polysomnography, as in adults, is the gold standard for diagnosis of OSA in children.[8] The standard parameters provided from polysomnography include sleep architecture, respiration, cardiac rhythm, muscle activity, gas exchange, and snoring. The most important index of polysomnography in defining the severity of OSA is the apnea/hypopnea index (AHI) which is defined as the number of apneas and hypopneas per hour of total sleep time. Apnea in children is defined as absence of airflow with continued chest wall and abdominal wall movement for a duration longer than 2 breaths,[9] whereas obstructive hypopnea is defined as a decrease in nasal flow between 30% and 80% from baseline with a corresponding decrease in oxygen saturation of 3% and /or arousal.[8] An AHI > 1 event/h in children is considered abnormal.[10] With the use of appropriate equipment and an experienced technician, polysomnography can be performed successfully in infants and children of all ages. There are several polysomnographic differences in OSA between children and adults.

1. Children with OSA frequently do not have cortical arousal associated with obstructive apnea and are less likely to have fragmented sleep than adults. Consequently, sleep architecture is preserved and daytime sleepiness is uncommon.[11,12]

2. In children, the majority of obstructive apneas occur during rapid eye movement (REM) sleep, particularly in later REM sleep.[13] As a result, OSA may be missed if the REM stage is decreased or absent on screening studies, e.g. nap studies.

3. Children may present with persistent obstructive hypoventilation, rather than cyclic obstructive apnea.[8,14] Clinically, these children manifest constant snoring and labored breathing instead of breathing pauses or gasps.

Epidemiology

The prevalence of OSA in children has been reported to be between 1% and 3%.[15,16] Although there is no integrated data, UARS is estimated to be more common than OSA since children with clinical

symptoms suggestive of OSA are more likely to have UARS than OSA.[14,17] Of note, 9% to 10% of children are habitual snorers. This condition may be transient or progress to UARS or OSA, and these children have the same risk of complications as children with OSA.[15,16]

Mechanism/etiology

The pathophysiology of SDB in children is similar to that seen in adults. During sleep, the ventilatory drive and upper airway muscle tone decrease. The inspiratory force collapses the pharyngeal airway that is already narrowed from other anatomic causes. The collapse of the pharyngeal airway leads to partial airway obstruction producing hypopnea, or total airway obstruction resulting in apnea. Apneic and hypopneic events are terminated by arousals, in which natural defense mechanisms, the pharyngeal dilators, are activated. The whole cycle may repeat itself when the child returns to a deeper sleep stage with decreased ventilatory drive and upper airway muscle tone. The etiology of OSA in children is complex. Several facts suggest that a combination of structural and neuromuscular abnormalities needs to be present for OSA to occur. The most common form of pharyngeal narrowing in children is caused by hypertrophy of the adenoids and tonsils and is associated with the fact that the facial bones grow more slowly than the lymphoid tissue during childhood.[18,19] Other factors predisposing to SDB in children include craniofacial anomalies,[20] neuromuscular diseases,[21] and obesity.[22] It's noteworthy that children with cleft palate may develop OSA following posterior pharyngeal flap surgery because of narrowing in the nasopharynx. A list of some of the diseases associated with OSA is shown in Table 2.

Table 2. Factors Predisposing to Sleep-disordered Breathing in Children

Adenotonsillar hypertrophy
Nasal obstruction*
Craniofacial disorders†
Cerebral palsy
Obesity
Laryngomalacia (infant)
Gastroesophageal reflux (infant)
Cleft palate following pharyngeal flap surgery

*: Common causes of nasal obstruction include allergic rhinitis, septal deviation, chronic sinusitis, nasopharyngeal stenosis; †: Common craniofacial disorders include Pierre Robin sequence, Crouzon syndrome, Apert syndrome, Treacher Collins syndrome, Down syndrome, and Prader-Willi syndrome, etc.

Clinical presentation

The three main nighttime symptoms of OSA in infants and children are snoring, apnea with noisy resumption of breathing, and difficulty in breathing with an inward movement of the upper chest during inspiration.[23]

Snoring occurs in almost all children with SDB and is the main reason many parents seek medical advice. However, only a proportion of snoring children have OSA (habitual snoring: OSA = 10%: 2% in the childhood population).[24] Besides, children with severe OSA may manifest without clear snoring because of prolonged breathing pauses. Consequently, snoring alone is an insensitive indicator of OSA and it is difficult to make a diagnosis of OSA based on a history of snoring alone.

In addition to snoring, the majority of children with SDB who are referred to otolaryngologists have mouth breathing and adenotonsillar hypertrophy. The relationship between mouth breathing and adenoidal hypertrophy is straightforward. A study revealed that mouth breathing is a significant predictor for suspecting OSA with a specificity and positive predict value of 100%, and warrants early polysomnography.[25] Mouth breathing has also been found to be a cause of abnormal facial development such as adenoid faces and dental malocclusion.[26,27]

The increased respiratory effort required in children with an obstructed upper airway can be manifested by suprasternal retraction, flaring of the costal margins, use of accessory respiratory muscles, and paradoxical inward motion of the rib cage (the downward movement of the diaphragm during inspiration causes the abdomen to move outward when markedly negative intrathoracic pressure causes inward motion of the rib cage).[16] Almost all children with SDB demonstrate increasing respiratory effort to some extent and the associated paradoxical motion usually frightens parents and leads them to seek therapy.

Restless sleep and persistent body movements are frequently observed in children with SDB. Odd

Table 3. Treatments of Sleep-disordered Breathing in Children

Non-surgical treatment	Surgical treatment
Treatment of nasal allergy	Adenotonsillectomy
Treatment of acute inflammation	Uvulopalatopharyngoplasty
Treatment of reflux	Nasal surgery
CPAP	Revision of posterior pharyngeal flap
Rapid maxillary expansion	Distraction osteogenesis
Weight reduction	Tracheotomy

Abbreviation: CPAP: continuous positive airway pressure.

sleep positions (kneeling or upright sitting) are not rare in childhood with SDB. These unusual behaviors are thought to be a compensatory mechanism to extend the neck and an improve airway obstruction during sleep.

Enuresis, particularly if secondary in etiology, is associated with SDB in children. It is thought that fragmentation of sleep architecture by apnea and arousal may affect normal secretion of anti-diuretic hormone, and contributes to enuresis. Nocturnal enuresis can be the principal clinical picture in some older children with SBD.[28] The manifestation can be reversed by adenotonsillectomy in most cases.[29]

Excessive daytime sleepiness, the most prominent clinical symptom of OSA in adults, is not a common complaint in pediatric SDB.[30] However, it is seen in some children with severe OSA and is more common in adolescents, particular if they are morbidly obese. In contrast, younger children often become hyperactive rather than sleepy.

Children with OSA can display daytime behavior disorders such as inattention, hyperactivity, aggressiveness, and social withdrawal.[2,31] There is good evidence that SDB leads to daytime disturbance closely mimicking attention-deficit/hyperactivity disorder (ADHD) in children.[32] Furthermore, snoring and other SDB-related symptoms are strong risk factors for the future emergence or exacerbation of hyperactive behavior in a four-year prospective cohort study.[33] In addition, learning problems can be at the forefront of the clinical picture in school-age children.[34] It is therefore recommended that a sleep study be performed to identify SDB in children diagnosed with ADHD.

Growth impairment is one of the main features in advanced pediatric OSA. Stunted growth in children with SDB could occur because of disturbance in growth hormone secretion from disruption in sleep architecture.[35] Hypertrophy of the tonsils can lead to difficulty in swallowing and interfere with adequate caloric intake, causing growth impairment. In addition, increased respiratory effort during sleep can drain the child's caloric resources which otherwise would be used in somatic growth.[16] Fortunately, the incidence is low nowadays, because OSA is diagnosed at earlier stages before failure to thrive becoming severe.

Physical examination and identification of the obstruction site

The site of obstruction in pediatric SDB is often noted on direct physical examination of the nose, oral cavity, and craniofacial morphology. A nasal speculum examination in children can show boggy changes with a pale inferior turbinate indicating allergic rhinitis (Fig. 1).

Oropharyngeal examination in children with SBD may demonstrate a crowded oropharynx with tonsillar hypertrophy, palate/tongue interposition, a high, narrow hard palate, macroglossia with tongue ridging, and lateral narrowing (impingement of the pharyngeal space by peritonsillar tissue), as shown in adults.[36] In addition, tonsillar size and tongue position are clinical predictors of OSA and can be used to predict outcomes of palatopharyngeal surgery for

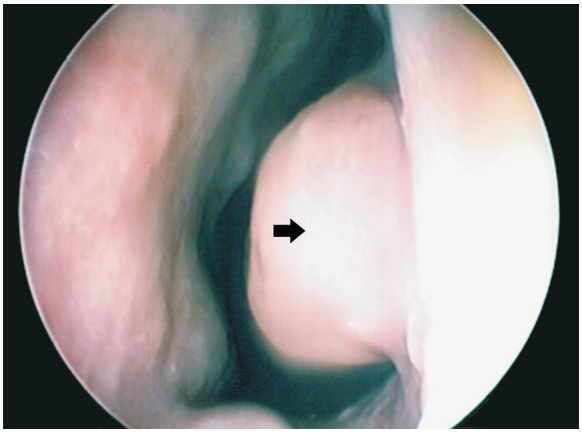

Fig. 1 Nasal speculum examination in a child with allergic rhinitis. Hypertrophic changes with a pale inferior turbinate (arrow) indicates allergic rhinitis.

OSA in adults.[37,38]

Fiberoptic nasopharyngoscopy is helpful in identifying adenoidal hypertrophy and the extent of obstruction by the adenoids in the nasopharynx (Fig. 2). In a study by Brooks et al.,[39] adenoid size was related to the duration but not the number of episodes of obstructive apnea in children. It is also necessary to perform a nasopharyngoscopy to examine whether or not there is regrowth of the adenoids in children with recurrent SDB after adenotonsillectomy. In addition, fiberoptic endoscopy is particularly useful in determining laryngomalacia in pediatric laryngeal OSA (Fig. 3).

Image studies to evaluate the lumen of the upper airway include plain radiography of the lateral neck (Fig. 4), cephalometry, and computed tomography.[40] Radiological evaluation of the adenoids and tonsils in children with OSA has failed to show a correlation between adenotonsillar size or upper airway space and the degree of OSA.[41,42] However, image studies can be helpful in demonstrating abnormal structural relationships and planning the surgical approach, which are not always necessary in routine use. Children with severe OSA should be assessed for pulmonary hypertension by chest radiography and electrocardiography.

Treatment/outcome

Treatment for children with SDB can be surgical

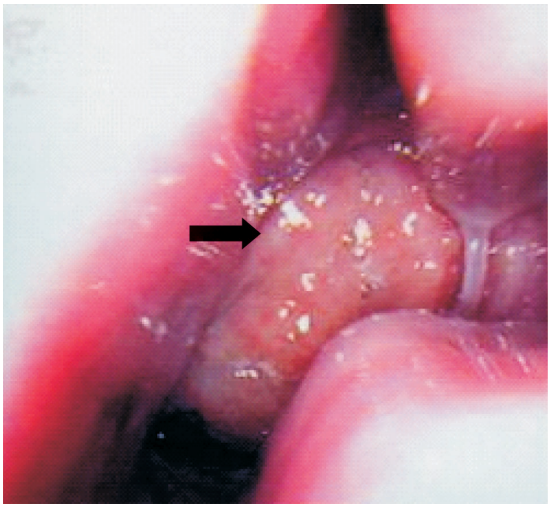

Fig. 2 Nasopharyngoscopy in a child with adenoidal hypertrophy. Hypertrophic adenoid tissue (arrow) obstructs the nasopharyngeal and posterior nasal airway and may cause sleep-disordered breathing.

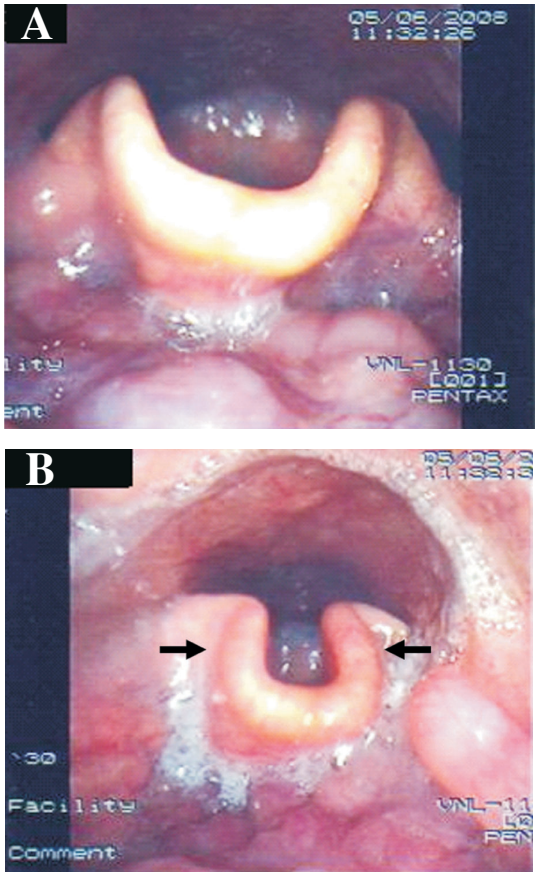

Fig. 3 Fiberoptic laryngoscopy in an infant with laryngomalacia. During expiration, the gross appearance of the epiglottis is normal (A). During inspiration, the flaccid epiglottis moves inward into the larynx and obstructs the airway (arrows).

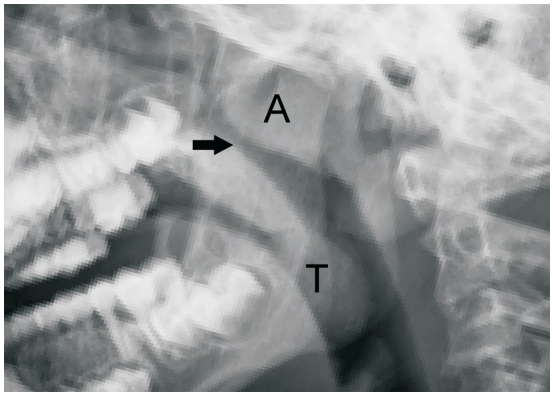

Fig. 4 Plain radiograph of the lateral neck in a child with sleep-disordered breathing (SDB). Adenoidal (A) and tonsillar (T) hypertrophy are the most frequent findings in children with SDB. The hypertophic adenoid can narrow the nasopharyngeal airway (arrow).

or nonsurgical, and the choice depends on the underlying etiology. Commonly the first step in the treatment of pediatric SDB is adenotonsillectomy.

Adenotonsillectomy

1. Rationale. Adenoid and tonsillar hypertrophy occurs most often between the ages of 2 and 6 years. During this period, the pharyngeal lymphoid tissue grows faster than the facial bones and is the largest in relation to the underlying airway size, which are frequently leading to pharyngeal obstruction during sleep.[19] Spontaneous resolution of OSA secondary to adenotonsillar hypertrophy within an 1-year observation period was reported to be only 9%.[43] Adenotonsil-lectomy significantly improves obstructive symptoms in 80% of cases.[44] Hence, adenotonsillectomy is the most common treatment for SDB in children.

2. Age restrictions. The age limit for a adenotonsillectomy may be related to fear about more frequent upper airway infections and postoperative complications. In a cohort study, children receiving adenotonsillectomy were compared with age-matched subjects. The results showed no significant differences in the number and duration of upper respiratory infections between the two groups in a 20-year follow up.[45] To what extent age influences the morbidity and mortality of adenotonsillectomy is debatable. Another report revealed that children under 3 years old who had an adenotonsillectomy had more postoperative complications.[46] It is our policy to implement routine adenotonsillectomy for SDB in children older than 3 years. However, adenotonsillectomy can be performed in severe airway obstruction without any age restriction.

3. Surgical technique. The procedure for an adenotonsillectomy includes removal of the palatal tonsils and adenoids by traditional or power instruments (e.g. laser, harmonic, radiofrequency, microdebrider, coblation).[47-52] Power instruments have been reported to reduce the duration of the operation, postoperative hemorrhage and pain.[47-52] However there is no robust evidence to support their advantages and cost needed to be taken into account. Whether the tonsillar wound needs to be sutured is not certain. Guilleminault et al.[53] suggested suturing the tonsillar wound reduces the collapsibility of the pharynx. At Chang Gung Memorial Hospital, we routinely suture the tonsillar fossa wound (palatoglossus muscle, palatopharyngeus muscle, pharyngeal constriction muscle) to maximally enlarge the retropalatal airspace and decrease the collapsibility of the lateral pharyngeal wall and soft palate. It is arguable whether a tonsillectomy should be performed in children with SDB who have isolated adenoid hypertrophy. Hulcrantz et al.[54] deemed that tonsils may look relatively small but rotate medially and superiorly, obstructing the retropalatal airspace. For this reason, it would be prudent to remove the tonsils in cases of documented OSA to maximize the pharyngeal airspace.

4. Results. The efficacy of pediatric adenotonsillectomy for snoring is around 91% based on -parent-reported questionnaires.[54,55] The cure rates of OSA in children after adenotonsillectomy as determined by an AHI < 5 events/hr postoperatively vary between 78.4% and 100%.[56,57] Quality of life based on the OSA-18 quality-of-life survey improved significantly following adenotonsillectomy in children with SBD in one study.[58] Moreover, subjective evaluation using the Child Behavior Checklist and objective examination using the Test of Variables of Attention showed that the behavior of these children improved following adenotonsillectomy.[59-61]

5. Failure and recurrence. Children with underlying airway abnormalities such as craniofacial anomalies, neuromuscular deficits and pathological obesity are more likely to fail after initial treatment with adenotonsillectomy than otherwise normal children.[62] In toddlers and preschool-aged children, SDB may recur because of adenoid regrowth. Postoperatively, residual adenoid can continue to grow to reach its growth peak.[63] Persistent inflammation such as allergic rhinitis may exacerbate the process of adenoid hypertrophy. By contrast, obesity, particularly the body mass index slope, after adenotonsillectomy is the most important factor causing recurrent SDB in adolescents.[64] In addition, children with bite abnormalities are prone to recurrence in adolescence several years after adenotonsillectomy since the craniofacial bones reach their full manifestation during puberty.[65] Guilleminault et al.[65] reported that 13% of children who had been successfully treated with adenotonsillectomy developed recurrence of OSA as adolescents. It is therefore necessary to monitor these patients regularly even if they have a good response to adenotonsillectomy initially. It is noteworthy that some children develop persis-

tent instead of transient velopharyngeal incompetence after adenotonsillectomy. This could be largely attributed to neurological disease or a submucosal cleft, and demonstrates the necessity for careful assessment before planning treatment. Pharyngeal augmentation or pharyngoplasty is indicated in those who fail to respond to conservative therapy.[66]

Uvulopalatopharyngoplasty (UPPP)

UPPP has been successfully used to strengthen hypotonic pharyngeal musculature in children in whom abnormal upper airway neuromuscular tone contributes to OSA, such as those with cerebral palsy or Down syndrome.[67,68] It has also been reported to be helpful in treating an otherwise normal child with OSA.[69] Currently, we consider to use UPPP for cases of pediatric OSA with obesity or redundant oropharyngeal soft tissue. Nevertheless, the muscular structure of the soft palate must be preserved and the procedure done as conservatively as possible in children to prevent troublesome velopharyngeal insufficiency.

Children with cleft palate who have received pharyngeal flap surgery are prone to develop SDB because of stenosis in the nasopharynx. Flap revision with relief of the nasopharyngeal synechiae is often necessary regardless the technique is difficult.

Tracheotomy

Tracheotomy is the ultimate treatment of OSA. Bypassing the pharyngeal obstruction can relieve obstructive apnea but not central apnea. However, a tracheostomy in children is associated with many side effects including impediments in speech and learning, chronic tracheitis, and interference with social activity. Fortunately, the introduction of continuous positive airway pressure (CPAP) and other treatment modalities has decreased the requirement for tracheostomy in pediatric OSA. At present, tracheotomy is rarely used in otherwise normal children who fail adenotonsillectomy, but may be needed in children with neuromuscular disorders such as cerebral palsy or severe craniofacial anomalies.

Rapid maxillary expansion (RME)

RME is an orthodontic procedure that uses a fixed appliance with an expansion screw anchored on selected teeth. It is aimed at skeletal expansion of the upper jaw by the application of orthopedic force to the midpalatal suture resulting in maxillary widening.[70] Intervention with RME includes an active expansion phase (1 mm/day) for 10 to 20 days based on the original narrowness of the maxilla, and a fixed retention phase for consolidation with the device kept in place for 6 to 12 months.[71] Children with OSA who have maxillary contraction, no adenotonsillar hypertrophy, and a body mass index < 24 kg/m^2 are considered to have the most favorable response to RME.[70] A significant reduction of AHI from 12.2 to 0.4 events/hr was found in a recent report using the aforementioned criteria.[71] The improvement in OSA by RME may stem not only from augmentation of the maxillary complex, but also from modifying the resting posture of the tongue.[72]

Distraction osteogenesis

Distraction osteogenesis, which involves slowly moving the mandible or midface in the direction desired using a distraction device, has become an accepted procedure in the treatment of OSA in children with severe maxillomandibular deficiency.[73,74] The efficiency of distraction osteogenesis for OSA in syndromic children is not clear because the majority of reports did not demonstrate polysomnographic data before and after the operation. However, distraction osteogenesis can effectively avoid or end a tracheotomy, which is generally the major treatment objective in this patient group.[75]

Continuous positive airway pressure (CPAP)

Nasal CPAP, continuous blowing of air into the larynx to develop an air splint to prevent airway collapse, is feasible in infants and children with OSA.[76,77] Nasal CPAP has been successful in treating severe OSA in children with other underlying medical disorders who can not benefit from current surgical techniques or who need temporal treatment until definitive surgical therapy is done.[78] Moreover, the introduction of CPAP has already modified the indications for tracheotomy in infants and children with OSA due to craniofacial anomalies.[79] Nevertheless, the facemask and pressure level are needed to change as growth proceeds.[76,77] Furthermore, the full cooperation of parents is indispensable to good compliance in pediatric OSA patients.

Pharmacologic treatment

Adenotonsillar hypertrophy usually results from

repeated infection or inflammation. Acute inflammation of pharyngeal lymphoid tissue can exacerbate OSA-related symptoms. Pharmacologic treatment should be tried before surgical intervention as alleviation of apnea may occur. Broad spectrum antibiotics have been shown to decrease tonsil size significantly in the short term. However, in one study only 15% of patients avoided surgery in long-term follow-up.[80] Intranasal steroids may be tried if adenoid hypertrophy is the predominant cause of mild OSA in children. Decreases of 29% in adenoid size and 82% in the symptom score were noted in one study.[81] Systemic steroids may be tried in nonacute adenotonsillar hypertrophy, as a short-term decrease in tonsil size has been noted, but long-term improvement and avoidance of surgery are usually not seen.[82] It is now generally accepted that pharmacologic agents have no significant and persistent effect in the treatment of OSA in children. However, in children with mild SDB symptoms related to inflammation, a trial of pharmacologic treatment may be helpful. Additionally, persistent control of allergic rhinitis is important in children with SDB who receive an adenotonsillectomy to decrease adenoidal regrowth.

Conclusions

SDB in children is not the same as in adults. Essentially, the symptoms, assessment, diagnosis, treatment, and sequelae are different. Clinical presentations in children with SDB are insensitive in predicting OSA. Polysomnography remains the gold standard for diagnosing OSA in children. The treatment of SDB in children needs to be tailor-made according to the etiology. Adenotonsillectomy can improve most OSA-related symptoms in children with adenotonsillar hypertrophy and may be helpful in pediatric behavior and learning. Recurrence of OSA after adenotonsillectomy due to adenoidal regrowth or obesity is not uncommon and long term follow-up is needed.

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小孩睡眠呼吸障礙

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有睡眠呼吸障礙的小孩能以單純打鼾、上呼吸道阻力症候群或阻塞性睡眠呼吸中止症來表現，並導致生長遲緩、神經認知缺損，但較少出現心血管後遺症。有睡眠呼吸障礙的小孩其臨床表現呈多樣性，但多數出現在 4 至 8 歲。一般而言，睡眠呼吸障礙在嬰幼兒多出現吵雜呼吸聲及夜間睡不安寧，學齡前小孩常出現打鼾及張嘴呼吸，至於學齡兒童則在行爲異常及牙齒咬合問題較常被注意到。小孩睡眠呼吸障礙的致病機轉尚未被完全了解，扁桃腺樣體肥大是主要原因，其他的危險因素包括過敏性鼻炎，顱顏異常，腭裂經後咽皮瓣手術，神經肌肉疾病，軟喉症及肥胖。多項睡眠生理檢查是診斷的標準方法，然而在診斷的定義上仍有歧見。治療小兒睡眠呼吸障礙最主要的方法爲扁桃腺樣體切除術。以牙套快速擴張上頷骨可以治療因上頷骨狹窄；成骨牽引術則可用於嚴重上下頷缺陷之兒童；連續正壓呼吸器能治療困難醫治的或重度的睡眠呼吸中止症小孩，此療法亦逐漸取代氣管切開術來治療顱顏異常併阻塞性睡眠呼吸中止症。經追蹤接受治療之阻塞性睡眠呼吸中止症兒童後顯示，合併有顱顏結構異常或神經肌肉功能不全者，其療效較差，而青春期的肥胖則會導致兒童阻塞性睡眠呼吸中止症的復發。(長庚醫誌 2009;32:247-57)

關鍵詞：睡眠呼吸障礙，阻塞性睡眠呼吸中止症，扁桃腺樣體切除術，牙套快速擴張上頷骨，成骨牽引術，連續正壓呼吸器

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