Craniofacial Growth Modification for OSA Children

4

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4.1 Major Differences in Pediatric OSA

Pediatric OSA differs from adult OSA in both diagnosis and management. Pediatric OSA is more difficult to be correctly diagnosed than adult OSA due to less available PSG finding, less sensitive HST finding, less reliable questionnaire, limited information from lateral cephalometry, and uncommon application of CBCT to the growing patients. Different diagnostic criteria and subjective symptoms need to be contemplated.

4.1.1 Different Diagnostic Criteria

Overnight PSG is the gold standard for diagnosing OSA in children as in adults. However, AHI as specific criterion of OSA severity is provided differently from that

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S.-J. Kim, K. B. Kim (eds.), *Orthodontics in Obstructive Sleep Apnea Patients*,
https://doi.org/10.1007/978-3-030-24413-2_4

in adults (Table 4.1). Poor availability of pediatric sleep laboratories for overnight PSG has prompted a search for alternative diagnostic screening tools including a combination of history and physical examination findings.

4.1.2 Different Subjective Symptoms

Pediatric OSA can be undiagnosed, since the symptoms are different from adults. The main differences between children and adults are compared in Table 4.2 [1]. The chief complaint of OSA children is snoring or labored breathing during sleep, and mouth breathing is frequently observed during the examination time. The most common cause is known to be adenotonsillar hypertrophy, followed by nasal obstruction. Obesity contributes to the pediatric OSA less than the adult OSA. Since sleep fragmentation, witnessed apnea, arousal, and excessive daytime sleepiness, which are very common in adult OSA, are not usually detected, it is hard to recognize OSA in children. Cardiovascular and neurovascular comorbidities rarely occur, instead, irreversible disturbances such as psychosocial and behavioral problems, neurocognitive deficits, learning disorder, physical and facial growth disturbances make serious concerns. The behavioral problems represent various aspects across ages: attention deficit hyperactivity disorder (ADHD)-like behavior in the early childhood, in contrast, depression in adolescence [2].

As a simple screening instrument of pediatric OSA, BEARS (B, Bedtime; E, Excessive daytime sleepiness; A, night Awakenings; R, Regularity and duration of sleep; S, Snoring) has been used in primary care settings [3].

Table 4.1 Different objective criteria of OSA severity between children and adults

OSA severity	Children	Adults
Mild	$1 \leq$ AHI < 5	$5 \leq$ AHI < 15
Moderate	$5 \leq$ AHI < 10	$15 \leq$ AHI < 30
Severe	$10 \leq$ AHI	$30 \leq$ AHI

Table 4.2 Different subjective signs and symptoms of OSA between children and adults

Signs and symptoms	Children	Adults
Chief complaint	Snoring, difficulty breathing	Daytime sleepiness
Snoring	Continuous	Intermittent with pause
Mouth breathing	Common	Less common
Adenotonsillar hypertrophy	Common	Uncommon
Nasal obstruction	Common	Less common
Sleep architecture	Preserved	Fragmented
Clinical arousal	Uncommon	Common
Sequelae	Behavioral problems, Neurocognitive deficits, Growth disturbance	Cardiovascular, neurovasucular comorbidities
Obesity	Less common	Common

4.2 Early Intervention: Why?

Since the signs of OSA may be slow to show up in the PSG, OSA symptoms may be unrecognized for years, worsening the craniofacial growth. Dentists are responsible for detecting and interrupting oronasal dysfunction in the primary care to prevent abnormal craniofacial growth and progression to SDB. Although abnormal craniofacial growth may have different genetically or environmentally induced etiologies, craniofacial growth evaluation and prediction should be performed for the children with the oronasal dysfunction or SDB. Craniofacial growth modification treatment need to be actively provided aiming at restoration of nasal breathing during sleep as well as during wakefulness.

The objectives of early intervention for growth modification are as follows: (1) to prevent irreversible disorders like somatic growth disturbance, neurocognitive and learning disorders, psychosocial and behavioral deficits; (2) to interrupt progression to adult OSA that has more serious symptoms and comorbidities; and (3) to prevent or correct the craniofacial deformity to break the vicious cycle.

The ultimate goals of early growth modification treatment include UA development to secure nasal breathing, as well as unlocking or stimulating mandibular growth in skeletal Class II, and developing nasomaxillary complex in skeletal Class III patients (Fig. 4.1).

4.3 Early Intervention: How?

4.3.1 Phenotype-Based Patient Selection

4.3.1.1 Medical Phenotyping

The main phenotypic causes responsible for the pediatric OSA are anatomical obstruction, inflammation, and neuromotor dysfunction (Fig. 4.2). Neuromotor dysfunction is an intrinsic factor of UA, which is related to the modified pharyngeal muscle tone and reflex response during sleep. This phenotype may not respond well to the physical treatment, so CPAP is firstly recommended, although there are concerns that long-term use of the CPAP can lead to midfacial flattening [4].

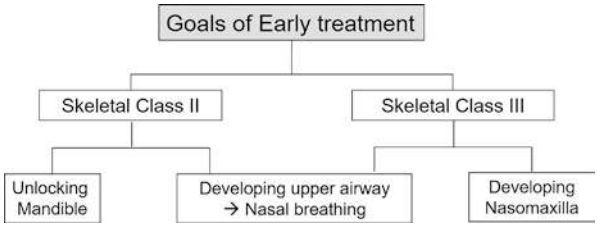


Fig. 4.1 Goals of early orthodontic intervention in terms of upper airway development in addition to skeletal development. Establishment of nasal breathing should be commonly included as well as unlocking the mandibular growth in skeletal Class II or developing nasomaxillary complex in skeletal Class III patients

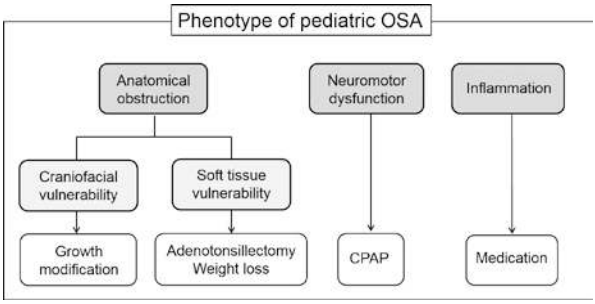


Fig. 4.2 Phenotype-based treatment approach for OSA children. Three major phenotypic causes of OSA children are anatomical obstruction, inflammation, and neuromotor dysfunction. Growth modification treatment can be effective in the OSA children with craniofacial anatomic vulnerability as a main phenotpyic cause

On the other hand, extrinsic factors of UA, including fat deposit, hypertrophic tissues associated with inflammation, and craniofacial deformity, can be controlled more favorably. Adenotonsillectomy is known to be the first-line treatment of pediatric OSA, since adenotonsillar hypertrophy has been regarded as the most frequent etiologic factor in children. According to current systematic review and meta-analysis [5], however, only 59.8% of children were cured of OSA (based on AHI <1) following surgery. Persistent mouth breathing or residual OSA may exist after surgery, and SDB symptoms tend to recur in early puberty, probably when other phenotypic cause is involved. Accordingly, phenotyping should be preceded. If the patient's main problem is nasal obstruction, nasal surgery, nasal expansion, or medication is required. If the patient is obese showing favorable craniofacial pattern and lymphoid tissues, weight loss with exercise is highly recommended, while craniofacial modification treatment should be actively applied to the patient with craniofacial skeletal risk factor. Combined approach should be considered to the patient with complicated phentoype.

4.3.1.2 Craniofacial Phenotyping

Once a patient is categorized into the group with craniofacial risk factors for the pathogenesis of OSA, the main target should be differentiated for the target-based treatment selection, which is called craniofacial phenotyping. Major facial traits, which should be attentively observed in the clinical examination, are retruded chin, depressed midface, and long and narrow midface. These can be confirmed by cephalometric and CBCT analyses.

4.3.2 Target-Based Treatment Selection (Fig. 4.3)

Children with craniofacial abnormalities have a high risk of residual OSA even after adenotonsillectomy [6]. Based on the craniofacial phenotyping, three major approaches for growth modification can be taken into consideration: unlocking or stimulating mandibular growth for skeletal Class II patient with retruded small chin; nasomaxillary

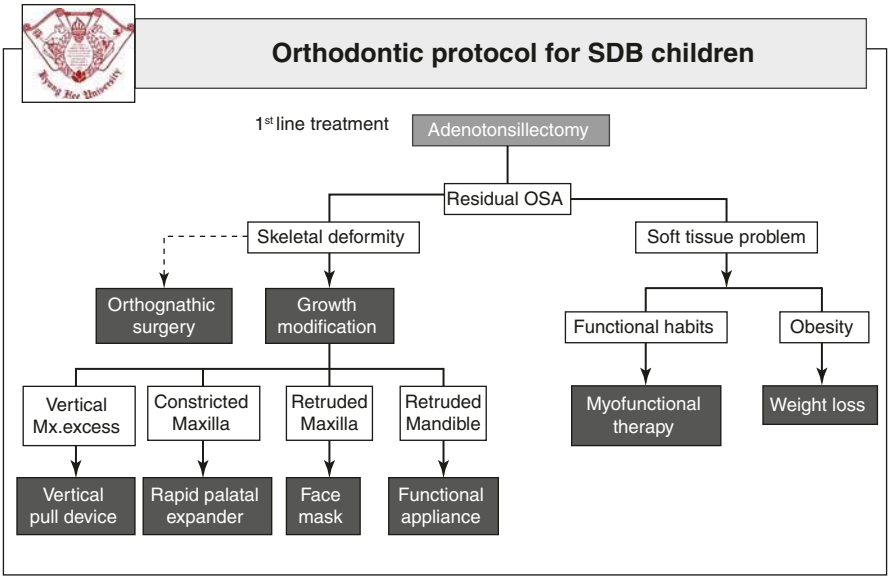


Fig. 4.3 A protocol of target-based treatment selection for SDB children (*Updated from Ahn et al. J Oral Maxillofac Surg 73:1827-1841, 2015*)

protraction for skeletal Class III patient with retruded midface; and nasomaxillary skeletal expansion for the patient with constriction of maxilla and nasal cavity. In addition, control of vertical maxillary excess should be carefully reflected at the same time. Also, myofunctional therapy needs to be combined with skeletal modification for ultimate functional improvement, particularly in patients having abnormal functional habits.

In spite of concerns about the residual growth, orthognathic surgery or distraction osteogenesis is another option to fix cases like severe skeletal Class II hyperdivergent pattern especially with syndromic deformity or medically refractory OSA (Fig. 4.3) [7, 8]

4.3.3 Timely Target Application of Appliances

4.3.3.1 Functional Appliance to Unlock or Stimulate Mandibular Growth

Recent systematic review [9] summarized that functional appliances had positive effect to open the oropharyngeal airway by forward growth of the mandible and subsequent adaptive advancement of tongue base, hyoid, and soft palate, through the action of genioglossus and palatoglossus muscles (Fig. 4.4). On the other hand, little improvement of nasopharynx and hypopharynx was observed. Most of the studies found no significant restraint in the maxillary growth, which might cause negative effects on nasopharyngeal airway [10]. Interestingly, oropharyngeal enlargement in three dimension (mediolaterally greater than anteroposteriorly)

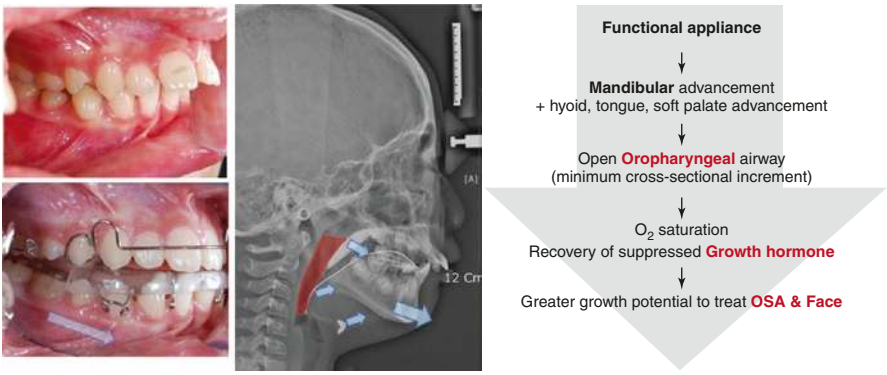


Fig. 4.4 Mechanism of functional appliance to open the upper airway with mandibular advancement. Along with the advancement of tongue base, hyoid, and soft palate, oropharyngeal airway including minimum cross-sectional (MCS) area can be enlarged permanently in case of true mandibular forward growth. Increment of MCS may recover the suppressed growth hormone by increasing oxygen saturation in the blood, contributing to the treatment of OSA with better face

includes the minimum cross-sectional increment [11], leading to the recovery of suppressed growth hormone with increasing oxygen saturation level. Increased growth hormone may release the suppressed physical and mandibular growth, improving face and airway better (Fig. 4.4). There was no significant difference in this effect among various types of functional appliances.

Four clinical implications underlie this mechanism of action. First, the positive effect of functional appliance can be expected only in the patients with good growth potential (before pubertal peak), good growth pattern (parallel or counterclockwise growth pattern), and good compliance (Fig. 4.5). Not all patients respond to the appliance. Second, there is no scientific evidence to affirm that functional appliance is effective to treat the OSA even with successful growth response [12]. Further research focusing on the respiratory functional improvement is anticipated. Third, even though true mandibular growth cannot be favorably expected with functional appliance in an OSA child, the appliance is worthy of releasing daytime symptoms by holding the mandible forward to open the airway during sleep. Lastly, in spite of controversy on the effect of functional appliance stimulating the mandibular growth, unlocking the mandibular growth itself should be the target of early intervention.

4.3.3.2 Facemask for Nasomaxillary Protraction

Maxillary protraction has the potential to improve respiratory efficiency in children with maxillary deficiency. Recent systematic reviews [13] demonstrated that skeletal modification effects induced by facemask include stimulation of forward maxillary growth with direct counterclockwise rotation of the palatal plane, and indirect clockwise rotation of the mandible. These orthopedic effects lead to the enlargement of nasopharyngeal airway by forward and downward displacement of PNS,

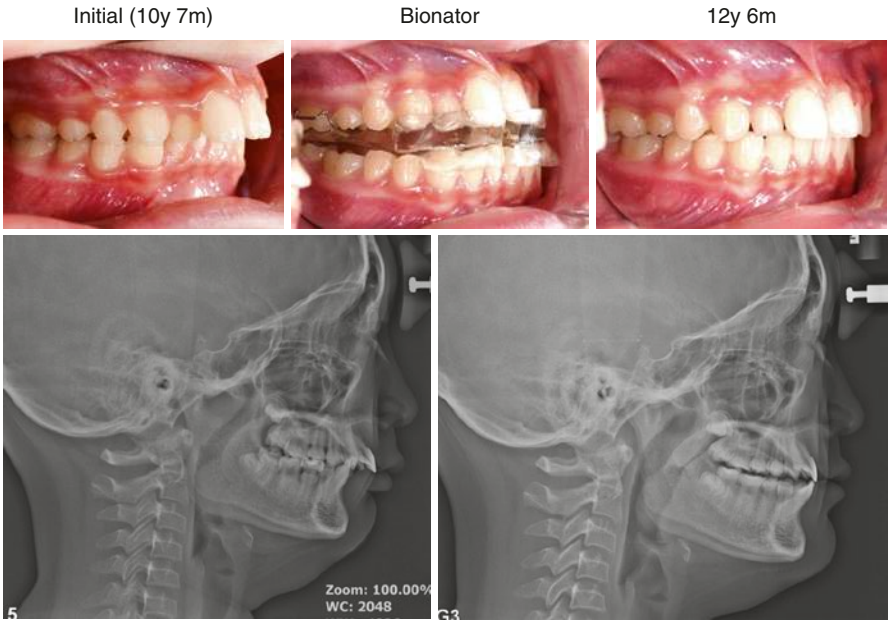


Fig. 4.5 Comparison of lateral cephalographic images and intraoral photographs between initial and 2-year after treatment using bionator in a skeletal Class II OSA patient. Oropharyngeal enlargement is noticed with mandibular advancement in spite of big tonsils and submental fat pads

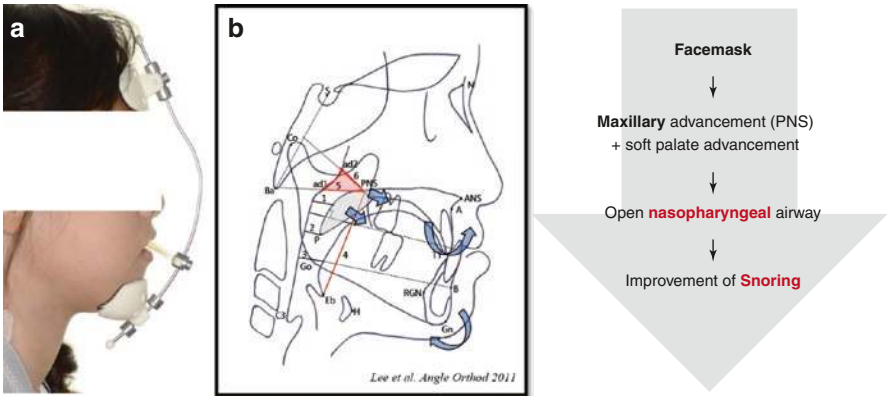


Fig. 4.6 Mechanism of face mask to open the upper airway with maxillary protraction. Stimulation of forward and downward maxillary growth can open the nasopharyngeal airway by pulling soft palate forward along with the displacement of posterior maxilla. In spite of clockwise mandibular rotation with maxillary protraction, oropharyngeal and hypopharyngeal airway spaces showed no significant decrease

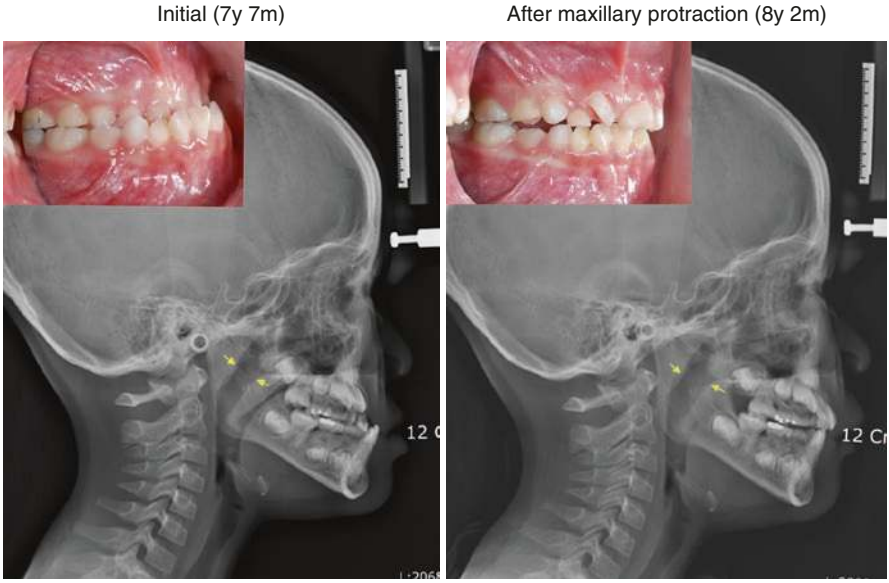


Fig. 4.7 Comparison of lateral cephalographic images before and after maxillary protraction using face mask. Yellow arrows indicate the nasopharyngeal space increase with improved maxillomandibular relationship

advancing the soft palate concurrently to increase velopharyngeal space (Fig. 4.6). Although the growth of adenoid tissues can be progressed until the age of 10–12, stimulation of suppressed maxillary growth by active treatment no later than 9–10 years old, when circum-maxillary sutures are still responsive, may ensure nasopharyngeal airway enlargement likely in a child showing normal growth (Fig. 4.7). On the other hand, oropharyngeal airway space showed no significant change after maxillary protraction as a result of combined effects between the increased tongue space by maxillary advancement and the possible backward tongue position due to the clockwise mandibular rotation.

4.3.3.3 Rapid Palatal Expander for Nasomaxillary Expansion

Nasal airflow is a critical factor for nasal breathing and pharyngeal collapsibility. Intranasal surgery or nasopharyngeal surgery can be considered to improve nasal airflow when there are hypertrophic or inflammatory tissues. However, constricted nasal cavity with narrow nasal floor and maxillary constriction requires structural expansion. Rapid maxillary expansion (RPE) is widely applied to the patients with nasal obstruction aiming at increasing narrow nasal cavity volume in sagittal and vertical as well as transverse dimensions, which is attributable to forward and downward displacement of nasomaxillary complex as well as maxillary basal expansion. Increased airflow will decrease pharyngeal collapsibility by lowering its critical closing pressure. Simultaneously, expanded oral cavity allows forward and upward repositioning of the tongue to open the oropharyngeal airway indirectly (Fig. 4.8).

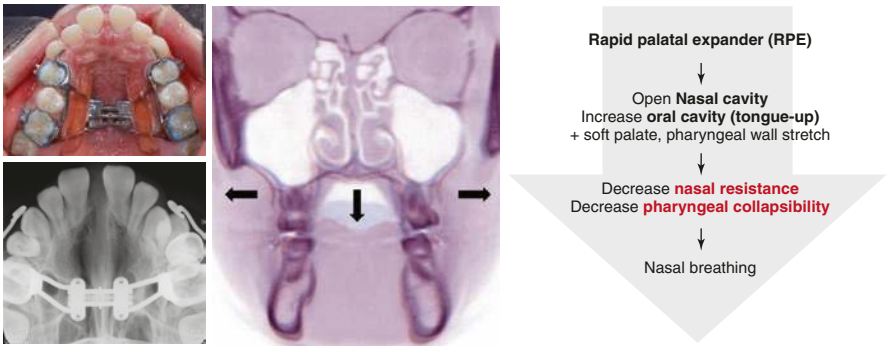


Fig. 4.8 Mechanism of rapid palatal expansion to open the upper airway. Through the midpalatal sutural expansion, nasal cavity and nasal floor are enlarged to increase nasal airflow, and palatal vault and oral cavity are expanded to move the tongue forward and upward. In addition, nasomaxillary complex is displaced forward and downward, inducing three-dimensional skeletal expansion. As a result, decreased nasal resistance and indirectly decreased pharyngeal collapsibility will treat the OSA patients with nasal obstruction

The success of nasomaxillary skeletal expansion depends on the stage of midpalatal sutural obliteration. Therefore, removable expander can be effectively applied to separate the suture in the preschool ages, while fixed type rigid expander should be used in the school ages before 15–16 years old [14]. Microimplants or adjunctive surgery is required to overcome the strong tissue resistance to sutural opening from postadolescence to adults. Nonetheless, microimplant-assisted rapid palatal expander (MARPE) is more favorable for both skeletal and airway expansion even in the growing children than RPE: greater maxillary base expansion and more parallel sutural separation from anterior to posterior palate with less dentoalveolar effect are expected in MARPE.

4.3.3.4 Headgear for interruption of Vertical Maxillary Excess

Traditionally, headgear treatment is intended to restrict the forward growth of the maxilla; thus, some clinicians are concerned about negative impact on the airway. Despite controversies, there was a study reporting the nasopharyngeal enlargement without oropharyngeal and hypopharyngeal changes after cervical pull headgear treatment [15]. Combined activator-headgear therapy represented the potential to increase oropharyngeal airway dimensions in skeletal class II patient with vertical maxillary excess and mandibular deficiency [16]. Given that the aggravating impact of vertical maxillary in excess results in clockwise mandibular rotation with narrowing oropharyngeal airway, inhibition of vertical maxillary excess using vertical pull headgear may be superior to interrupting increased facial divergency and increasing oropharyngeal patency (Fig. 4.9).

Not only posterior vertical maxillary excess with anterior openbite, but also severe gummy smile indicating anterior vertical maxillary excess needs to be carefully managed. Simultaneously with vertical maxillary inhibition, compensatory extrusion of lower

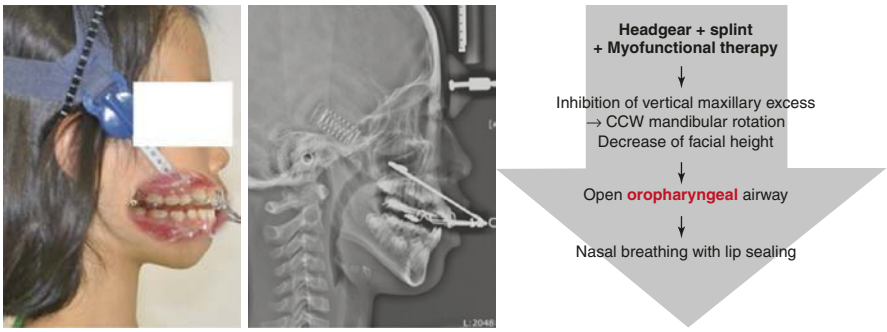


Fig. 4.9 Mechanism of splint-supported vertical pull headgear to open the upper airway

posterior teeth should be prevented by lingual arch or more active lower posterior intrusion using microimplants. Myofunctional therapy should be actively combined for the successful outcome.

4.3.3.5 Limitation: Rule Out the Poor Responders

Early growth modification treatment does not work successfully in every patient. Differential diagnosis should be based on the growth potential, growth pattern, growth stage, and patient's attitude. Here comes the orthodontist's role to determine whether to and when to intervene in individual patient.

- It is not easy to predict mandibular response to stimulate its growth, since it is controlled by genetics more than environments. At least, the concept of "unlocking the mandibular growth and airway development" should be a determinant of early intervention.
- Hyperdivergent vertical growth pattern with diverged mandibular shape can be a sign of poor mandibular growth and poor treatment response. Note that most of early interventions tend to increase vertical dimension with the correction of sagittal malocclusion. This may have an adverse influence on the oropharyngeal airway development.
- Due to the lack of untreated controls and prospective long-term data in the literatures, and due to high interindividual variability, controversy on both skeletal and airway effects still exists.

4.4 Cases

Case 1: A Snoring Child with Locked Small Mandible

A 10-year-old girl was visited orthodontic clinic with the chief complaint of upper incisor protrusion. She had no subjective nasal obstruction and no history of ENT treatment, but had snoring and structural mouth breathing due to the severely proclined upper incisors and trapped lip (Fig. 4.10). She showed a convex profile with retruded chin and short lower facial height. Dental Class II molar and canine



Fig. 4.10 Initial facial and intraoral photographs. Convex profile with retruded chin and short lower facial height was observed and severely proclined upper incisors with trapped lip were noticeable. Maxillary arch constriction and anterior deep overbite was locking the mandibular growth

relationship with large overjet and deep overbite, and constricted maxillary dental arch was observed. The lateral cephalometry indicated skeletal Class II malocclusion with retruded small mandible and hypodivergent vertical pattern with favorable mandibular shape for the pubertal growth (Fig. 4.11). Hypertrophic adenoid was seen in relation to the narrow nasopharyngeal and velopharyngeal airway. Her skeletal age was SMI 5–6, implying the imminent pubertal growth spurt (Fig. 4.11).

In terms of the growth prediction and growth stage, her mandible was evaluated to have favorable growth potential to improve the face and the airway. The important thing here is that any environmental factors locking the mandibular growth should be checked and removed before the growth peak. The most frequent locking factors that we have to always assure are maxillary arch constriction and anterior deep overbite. Since she had both of these factors, immediate intervention to allow the catch-up growth of the mandible by expansion of maxillary arch and intrusion of anterior teeth was required. Although this case seems to have sagittal mandibular deficiency, transverse and vertical control of maxilla needs to be firstly considered rather than stimulating the mandibular growth only using functional appliance.

As a result of timely orthopedic treatment passing through the pubertal growth peak, Class I molar and canine relationship with proper overjet and overbite could be accomplished with facial improvement (Fig. 4.12). The mandible underwent catch-up growth to attain skeletal and dental Class I relationship with increased pharyngeal dimension (Fig. 4.13). Spontaneous atrophy of adenoid tissue was observed along with the correction of structural mouth breathing.

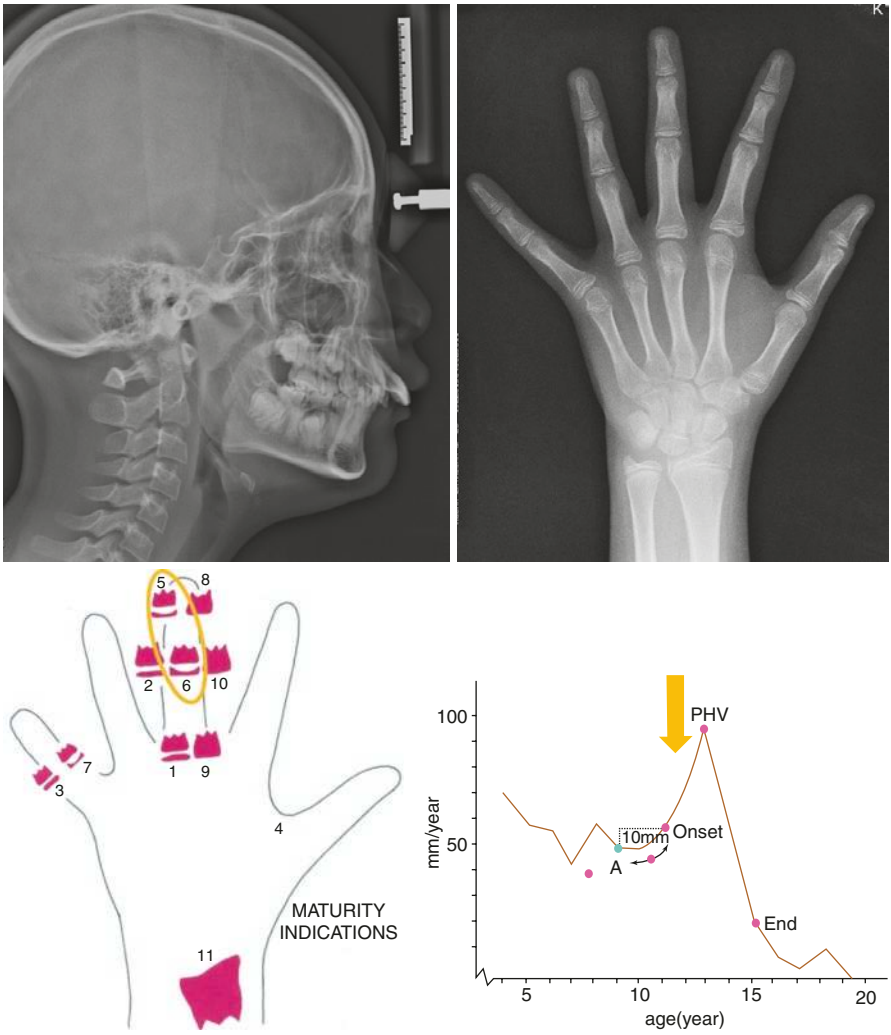


Fig. 4.11 Lateral cephalogram (left) and hand wrist X-ray (middle). SMI chart (right) indicated the growth stage of SMI 5–6, which means imminent pubertal growth peak

Case 2: An OSA Child with Gummy Smile and Nasal Obstruction

A 10-year-old girl was referred from ENT department for the evaluation of facial deformation. She was complaining about gummy smile and retruded chin and suffering from nasal obstruction with allergic rhinitis and subsequent mouth breathing.

In the clinical examination (Fig. 4.14), she showed convex profile with retruded chin, and long face with excessive lower facial height. She had narrow and deviated nasal bridge with narrow nares. Severe gummy smile in spite of anterior openbite was present in the mouth. Maxillary arch was constricted with deep narrow palatal



Fig. 4.12 Comparison of facial and intraoral photographs between initial (above) and at the end of treatment (below)

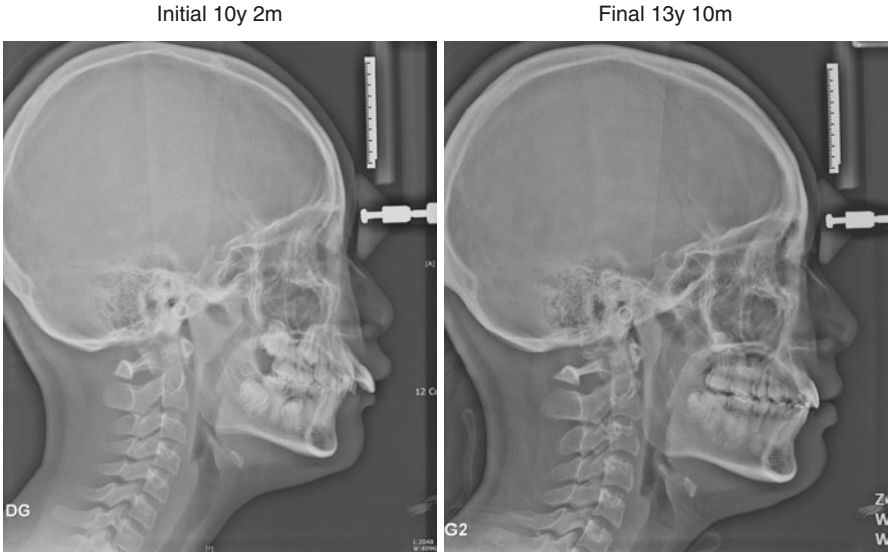


Fig. 4.13 Comparison of lateral cephalograms between initial (left) and at the end of treatment (right) representing favorable mandibular catch-up growth to attain skeletal and dental Class I relationship with increased pharyngeal dimension

vault. As a result of home sleep test (Apnealink Plus™), AHI was 2 indicating mild OSA in relation to the nasal obstruction.

She was diagnosed as OSA accompanied by craniofacial risk factors like skeletal Class II with retrognathic mandible, hyperdivergent vertical pattern with anterior vertical maxillary excess, and transverse discrepancy. Treatment objectives for the first phase growth modification treatment included maxillary skeletal expansion and



Fig. 4.14 Initial facial and intraoral photographs. Convex profile with retruded chin and excessive lower facial height was observed and excessive gum exposure during smile was noticeable. Maxillary arch constriction and anterior openbite was related to nasal obstruction-oriented mouth breathing



Fig. 4.15 Intraoral photographs before (left) and after (right) MARPE and occlusal topographic image showing parallel midpalatal sutural split

inhibition of vertical maxillary excess to redirect the mandibular growth and ultimately to open the upper airway (particularly nasal cavity).

Maxillary skeletal expansion was performed using microimplant-assisted rapid palatal expander (MARPE) in order to maximize skeletal effect involving nasal cavity and to minimize mandibular clockwise rotation as a response to the maxillary expansion (Figs. 4.15 and 4.16). Subsequently, high-pull headgear was applied with incisor intrusion arch to attain the flattening of upper occlusal plane and forward mandibular autorotation, as seen in the cephalometric superimposition (Fig. 4.17). As a result of

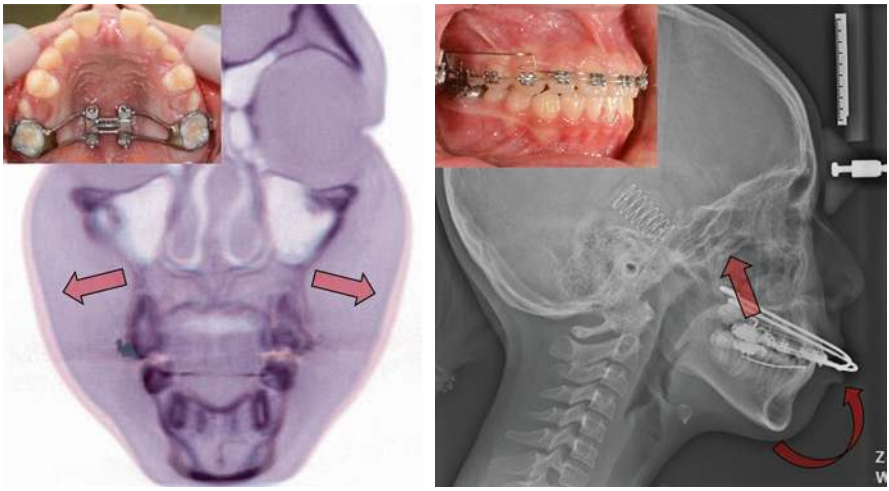


Fig. 4.16 Application of MARPE for the transverse expansion as noted on the CBCT superimposition (left) and high-pull headgear with incisor intrusion arch for the vertical control to apply the directional force system (right) as confirmed by the lateral cephalogram

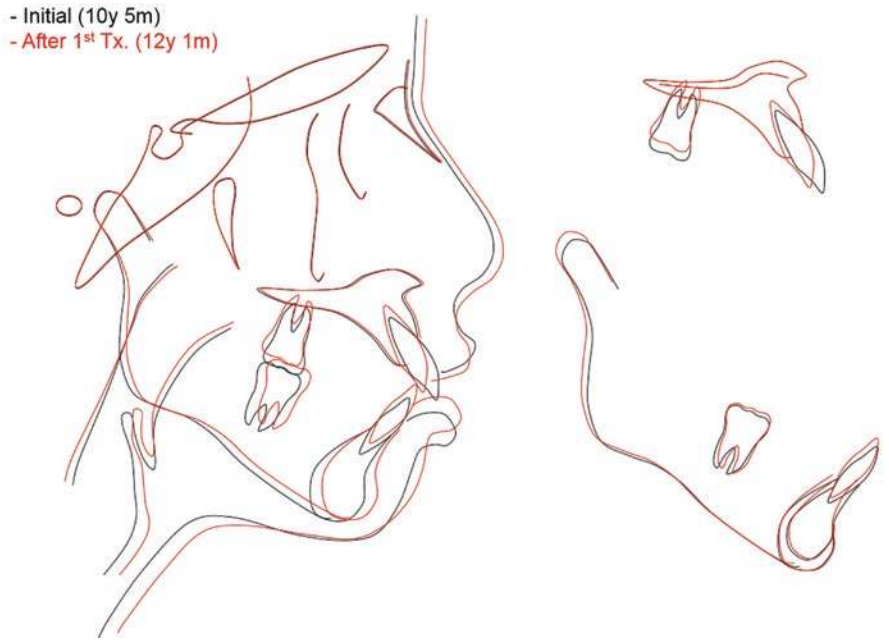


Fig. 4.17 Superimposition of lateral cephalograms between initial (black) and at the end of treatment (red). With successful vertical control, forward mandibular growth can be induced to develop pharyngeal airway space

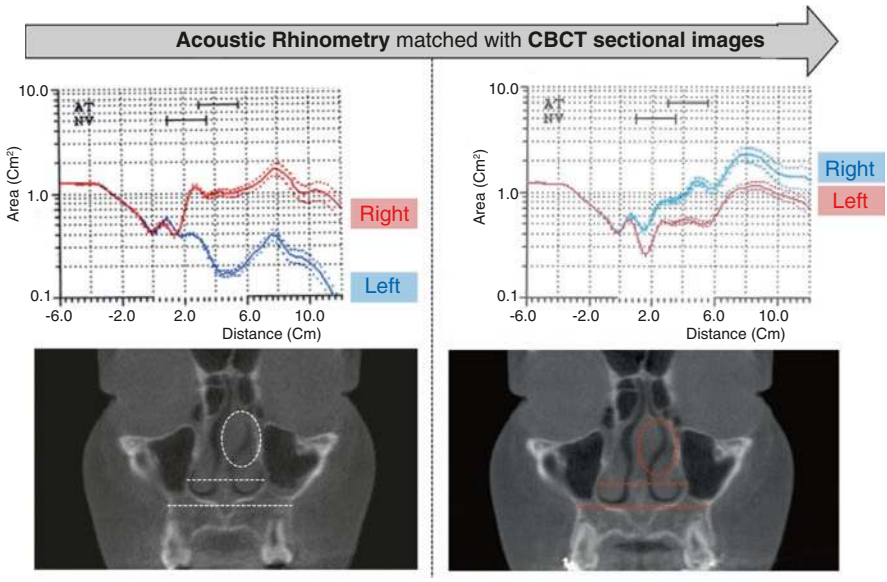


Fig. 4.18 Acoustic rhinometric finding matched with CBCT sectional images in the nasal cavity area. Intranasal area was increased especially in the left side where serious nasal obstruction existed as a result of maxillary expansion

Table 4.3 Comparison of home-sleep test between initial and at the end of growth modification treatment. AHI and ODI dropped to 0, and the lowest oxygen saturation increased up to 94% with decreased flow limitation

Apnea link plus™	Initial	After first treatment
AHI	2	0
$LSaO_2$ (%)	92	94
ODI	1.2	0
Flow limitation, Sn	29	17

acoustic rhinometry matched with the CBCT sectional images in the nasal cavity area (Fig. 4.18), the intranasal area was remarkably increased especially in the left side where almost complete obstruction existed as seen in the CBCT section, following the nasal cavity expansion with MARPE. Functional evaluation using a home-sleep test (ApneaLink Plus™) found the release of AHI with increased oxygen saturation after treatment (Table 4.3). She could have esthetic improvement with the correction of gummy smile and favorable facial profile in addition to respiratory improvement through the timely target intervention (Fig. 4.19).



Fig. 4.19 Comparison of facial photographs between before (above) and after treatment (below). Gummy smile was remarkably corrected, and chin profile was improved by the aid of enhanced nasal breathing

Clinical Pearls of Airway-Friendly Craniofacial Growth Modification

- Timely Target approach: Based on the differential growth theory, intervention timing and appliance type should be decided according to the bony target and respiratory obstruction area.
- Earlier intervention for nasal breathing: Early nasal expansion with MFT is important to prevent or interrupt the progression of SDB and facial deformation.
- Bone-anchored appliances for greater airway expansion: Greater amount of skeletal modification and more favorable skeletal effects on the airway patency with microimplants or miniplates may extend the range of orthodontic and orthopedic application for SDB growing patients.
- Interdisciplinary approach: We need to refer the patient with non-craniofacial or complicated phenotype to pediatric sleep specialists for intimate collaboration.

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