

Rapid Maxillary Expansion in Children with Obstructive Sleep Apnea Syndrome

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Objective: To evaluate the effect of rapid maxillary expansion on children with nasal breathing and obstructive sleep apnea syndrome.

Method: Recruitment of children with maxillary contraction, without of adenoid hypertrophy, with a body mass index < 24 kg/m², with obstructive sleep apnea syndrome demonstrated by polysomnography, and whose parents signed informed consent. Otolaryngologic and orthognathic-odontologic evaluation with clinical evaluation, anterior rhinometry and nasal fibroscopy, panoramic radiographs, anteroposterior and laterolateral telecephalometry were performed at entry and follow-up.

Intervention: Rapid maxillary expansion (ie, active phase of treatment) was performed for 10 to 20 days; maintenance of device (for consolidation) and orthodontic treatment on teeth lasted 6 to 12 months.

Results: 31 children (19 boys), mean age 8.7 years, participated in the

study. The mean apnea-hypopnea index was 12.2 events per hour. At the 4-month follow-up, the anterior rhinometry was normal, and all children had an apnea-hypopnea index < 1 event per hour. The mean cross-sectional expansion of the maxilla was 4.32 ± 0.7 mm. There was a mean increase of the pyriform opening of 1.3 ± 0.3 mm.

Conclusion: Rapid maxillary expansion may be a useful approach in dealing with abnormal breathing during sleep.

Key Words: Rapid maxillary expansion, obstructive sleep apnea syndrome, children, nasal opening, maxilla

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INTRODUCTION

THE ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA SYNDROME (OSAS), MAXILLOFACIAL MALFORMATIONS, AND MALOCCLUSIONS HAS ATTRACTED ATTENTION.¹ Many patients with OSAS show craniofacial abnormalities involving both the jaws as well as skeletal structures of the respiratory dynamic space.^{2,3} These aberrations may be apparent very early in life. Nasal septal deviation is known to reduce airflow and increase resistance to nasal breathing. Abnormal nasal resistance can be experimentally induced at birth in monkeys.³⁻⁵ When these conditions appear in the first year of life, they can cause a deformation of the upper jaw, affecting its cross-sectional development^{3,4,6-8} with a resulting reduced jaw size in experimental animals and in humans.

Nasal septal deviation results in the asymmetric distribution of intranasal space and affects the turbinates. The latter effect in turn causes a reduction of total airflow.^{3,9-11} The developmental impact of abnormal nasal resistance related to septal deviation early in life, with or without nasal turbinate hypertrophy, is abnormal maxillary development.

Rapid maxillary expansion (RME) treats upper-jaw constriction.¹¹⁻¹⁵ We questioned whether RME treatment for children could improve (1) nasal airflow by decreasing the abnormal nasal resistance and (2) OSAS.

MATERIALS AND METHODS

Subject Recruitment

All children were first seen at an orthodontic clinic. The study had been planned as a prospective investigation, and any child who was new to the clinic and had maxillary constriction, therefore being a candidate for RME, was first sent for otolaryngology (ENT) and sleep laboratory evaluations. Parents were told about the risk of the their children having OSAS with maxillary constriction, were provided with literature on the subject if requested, and underwent the clinical work-up with their child. To be included in the treatment study, the child had to respond to the inclusion and exclusion criteria, and the parents had to sign an informed consent approved by the university's internal review board.

Inclusion Criteria

To be considered for treatment, children had to meet the following criteria.

- Absence of adenotonsillar hypertrophy
- Body mass index¹⁶ < 24 kg/m²
- Presence of malocclusion characterized by upper jaw contraction such that RME would be an appropriate treatment based on orthodontic findings
- Presence of oral breathing, nocturnal snoring, and OSAS based on polysomnography
- Parental signatures on an informed consent approved by an ethics committee

Exclusion Criteria

Any child who was not compliant with the treatment and scheduled follow-up evaluations could be excluded from the study.

Disclosure Statement

No significant financial interest/other relationship to disclose.

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Evaluation

All of the children received (1) clinical ENT evaluations, active anterior rhinomanometry, and nasal fibroscopy and (2) clinical and radiologic orthodontic evaluations.

Panoramic dental radiographs and posterior-anterior and lateral cephalometric radiographs were obtained at the initial examination to assess malocclusion. An intraoral radiographic series was also obtained.¹⁷⁻¹⁹ Posterior-anterior cephalometric radiographs were analyzed using E. Gianni's recommendations¹⁷ and Ricketts parameters.^{18,19}

A validated pediatric sleep questionnaire was used to evaluate daytime sleepiness and fatigue.^{20,21} Polysomnography was recorded using 19 channels. Sleep-wake states were based on electroencephalogram, electrooculogram, electromyogram, electrocardiogram, and body position. Respiration was monitored by nasal and oral flow, thoracic and abdominal movements, snoring noise and pulse oximetry.²² Polysomnograms were analyzed following the Rechtschaffen and Kales international criteria for sleep-wake scoring²³ and the American Academy of Sleep Medicine recommendation for the scoring of breathing events.²² Abnormal events were considered present if longer than 2 breaths in duration. Events were classified as apnea or hypopnea based on airflow and as obstructive, mixed, or central, based on thoracoabdominal movements and airflow.

These different evaluations were carried out at entry before any orthodontic therapy (T_0), after 4 to 6 weeks with the device (T_1), and 4 months after the end of the orthodontic treatment (T_2), which lasted 6 to 12 months depending on the child.

Tool

RME is an orthodontic procedure that uses a fixed appliance with an expansion screw anchored on selected teeth. The central expansion screw has a diameter of 1.5 mm and 4 arms for anchoring to the teeth. A heavy force (about 1 kg) should be applicable to the anchor teeth to act directly on the palatal suture, without any undesired tooth movement. This anchoring area thus results in a transpalatal force that exceeds the physiologic level that normally leads to orthodontic movement. Here, maxillary orthopedic movement is obtained by reopening the midpalatal suture. Osteoids development occurs at the borders of the palatal process, and a normal mineralized suture is built up again at the end of the expansion, usually after 3 or 4 months.^{10,11}

Because the goal is to increase the transversal diameter of the hard palate, dental arch tipping must absolutely be avoided.¹⁰⁻¹² The device must not be bulky but, instead, must be strong and well fit to the anchorage. The expansion screw must be as high as possible toward the palate. The effectiveness of the RME depends on the amount of force and on the duration of application.^{10,11}

Anchor Teeth

The anchor teeth are selected according to the phase of development of the teeth. Commonly, the first molars and permanent premolars are selected as anchor teeth in older children. In deciduous teeth, the second molars are selected, provided that they offer stability.

The Procedure Used in the Study

An activation of the device consisted of turning the central screw to apply force on the anchor teeth to separate the palatal suture. On the morning of Day One, 3 consecutive activations were performed at 10-minute intervals, and 3 activations were performed in the evening, again at 10-minute intervals.

From Day 2 onward, the parents performed 1 activation, morning and evening. Activation consisted of turning the central screw in a predetermined direction. At T_0 , before any movement, an intraoral occlusal radiograph was obtained, and a new one was performed 3 days after the beginning of activation (at $T_0 + 3$). This intraoral occlusal radiograph verified the movement and opening of the midpalatal suture and confirmed the appropriateness of the device placement, allowing the safe continuation of expansion.

Duration of the Expansion

The active expansion ranged from 10 to 20 days in duration based on the original narrowness of the maxilla and the predetermined individual needs and possibilities. The amount of expansion was usually aimed at 1 mm per day. Once the active phase was over, a fixed retention phase, with the device kept in place, lasted from 6 to 12 months.

Special Attention

Important oral hygiene information was given to the child and parents. Recommendations concerning the active phase of the activation were also provided.

Statistical Analysis

Wilcoxon signed rank test was used to compare before and after treatment parameters. The statistical software package SPSS version 10 (SPSS, Inc. Chicago, Ill) was used for the analysis.

RESULTS

One hundred successively seen children with maxillary constriction were first sent to ENT and sleep laboratory evaluations. Thirty-one children met the criteria for inclusion: they had no adenotonsillar hypertrophy. In 22 children, this was due to prior adenotonsillectomy performed earlier in life, usually for recurrent tonsillitis or otitis, without investigation of presence or absence of OSAS at the time of surgery; in 9 children, no adenotonsillar hypertrophy was seen. These 31 children (19 boys) had a mean age of 8.68 years (range: 6-12 years).

The other 69 children were subdivided in 2 groups. Twenty-six presented with OSAS and adenotonsillar hypertrophy and, per protocol definition, were excluded from the study. Forty-three snored intermittently without demonstration of a pathologic apnea-hypopnea index (AHI) on polysomnography. All parents of the eligible children signed the informed consent. There were no dropouts and no exclusions. All children completed the treatment.

AT T₀

Orthodontic Evaluation

Extraoral Examination

All of the children presented a flattening of the middle third part of the face and were mouth breathers during wakefulness and sleep. The 31 children were distributed in the 3 skeletal classes that are based on skeletal sagittal relationship (Class I, *n* = 9; class II, *n* = 14; class III, *n* = 8). This distribution in all 3 classes was expected, as maxillary constriction is an abnormality of the transversal diameter.

Intraoral Examination

All of the children demonstrated a contraction of the upper jaw resulting from a high and narrow palatal arch. It was associated with malocclusion characterized by unilateral or bilateral cross-bite. Cephalometric radiographs confirmed the constriction of the upper jaw.

The ENT evaluation indicated the absence of the adenoids, as shown by nasopharyngoscopy and absence of tonsils. Twenty-two children previously had adenotonsillectomy or tonsillectomy, and 9 children had normal appearing.

Anterior rhinoscopy indicated nasal septal deviation with hypertrophy of the inferior nasal turbinates in 23 patients and isolated septal deviation in 8 patients.

Active anterior rhinoscopy indicated pathologic nasal resistance > 1.8 Pascal per cubic centimeter of water, with bilateral nasal breathing difficulties in 26 patients and unilateral nasal obstruction in 5 patients.

Prick allergy testing was negative for major perennial and seasonal allergies for all of the children.

All of the children reported daytime tiredness and were known snorers. Polysomnography demonstrated the presence of obstructive breathing during sleep. The mean AHI, defined as the number of events per hour of sleep, was 12.2 events per hour, with a range of 5.7 to 21.1 events per hour. Three subgroups were created based on AHI. Group A consisted of 7 children with an AHI > 5 and < 10 events per hour. Group B had 20 children with an

AHI = 10 and < 15 events per hour (mean = 12.4). Group C had 4 children with an AHI = 15 events per hour (mean = 18.3). All apneas and hypopneas were mixed and obstructive.

AT T₁

ENT Evaluation

Twenty-one of the 26 children with bilateral resistance and all of the children (*n* = 5) with unilateral resistance at T₀ had normal nasal resistance at T₁ (Wilcoxon *z* = -4.86, *P* = .0001).

Polysomnography

Twenty-nine of the 31 children had an AHI < 5. The other 2 children, both in Group C, had an AHI of 6.3 and 8.1 events per hour, respectively, from an initial AHI of 19.6 and 21.1 (Wilcoxon *z* = -4.0, *P* = .0001). (see table 1)

AT T₂

The ENT and polysomnographic evaluations demonstrated further improvement with significant difference not only from T₀, but also from T₁. Compared to T₁, anterior rhinometry demonstrated a further decrease in nasal resistance in all of the children (Wilcoxon *z* = -5.39, *P* = .0001), with complete absence of any pathologic reading (Figures 1 and 2).

Polysomnography indicated an AHI of less than 1 event per hour in all cases (T₁ vs T₂, Wilcoxon *z* = -2.0, *P* = .046).

The anatomic changes on the upper jaw and nasal septum were based mostly on the posterior-anterior cephalometric evaluations (Figures 1 and 2). Lateral cephalograms and intraoral occlusal radiographs were also obtained to confirm the opening of the midpalatal suture.

The expansion of the maxilla and mandible after RME had a mean cross-sectional increase (jL-jR distance)^{16,17} of 4.32 ± 0.7 mm. The study of the upper molar distance showed an average increase of 3.89 ± 0.3 mm, which associated with poor vestibular tipping due to rapid expansion. Consequent to midpalatal opening, a hallmark of the procedure, an interincisive space was always present, with a mean opening of 2.97 ± 0.2 mm. This interincisive opening, always of concern to parents, gradually disappeared due to transseptal fiber movement in all cases. Orthodontic therapy can speed up this movement but is usually unnecessary.

Finally, RME had an impact on the nasal cavities, with a mean increase of the nasal pyriform opening of 1.3 ± 0.3 mm (Figure 3). None of the children presented any problem with RME. They

Table 1—Polysomnographic Data for 31 Subjects

Polysomnographic Parameter	T ₀	T ₁	T ₂
Obstructive apnea-hypopnea index	12.18 ± 2.6	9.8 ± 2.7	0.4 ± 1.1
Range	5.7-21.1	0-8.1	0-2.1
Nadir SpO ₂ , %	78.5 ± 8.2	89.6 ± 5.9	95.3 ± 1.7
Duration of longest obstructive apnea, sec	35.2 ± 18.6	28.3 ± 14.1	12.6 ± 7.4
Duration of desaturation (SpO ₂ < 92%), % total sleep time	19.7 ± 3.5	6.6 ± 1.9	1.3 ± 1.1
Sleep efficiency, %	87.1 ± 8.8	88.6 ± 6.4	89.2 ± 7.7

T₀ refers to time before any orthodontic therapy; T₁, after 4 to 6 weeks with the device; T₂, 4 months after the end of the orthodontic treatment. All data are displayed as mean ± SD, unless otherwise indicated.



Figure 1—Face and profile of a child before treatment and profile of same child after treatment.

tolerated both the activation procedure and the stabilization phase well.

DISCUSSION

RME is a well-known procedure that has been used by different groups for many years in children with orthodontic problems.^{10-15,24-26} It is aimed at skeletal expansion of the upper jaw. The technique consists of the application of orthopedic force to the midpalatal suture. This anatomic area comprises mainly compact bone laterally and fibrous tissue with fibroblasts, collagen fibers, and blood vessels centrally.^{9,27-30} RME results in maxillary widening by distraction osteogenesis, which was defined more

than 100 years ago by Gavriel Ilizarov³¹ as “mechanical induction of new bone between two bony surfaces that are gradually distracted.” Histologic investigation demonstrates that the application of strong forces, through an orthodontic device anchored to the teeth, rearranges the central zone, with the collagen fibers arranging in the direction of the distraction, and a progressive ossification occurs. A child can withstand up to 1 mm of expansion daily, but the speed of expansion varies. The bone distraction at suture level causes an actual widening of the maxilla with increasing of the cross-section as well as the volumetric space of the nasal cavity. Radiographs of the region clearly indicate that RME moves nasal and palatal bones. The total expansion effect consists of a downward and forward movement of the maxillary complex with a resulting increase in the nasal canal with an improvement in nasal airflow.

Since 1984, Timms^{13,14,25} has published several articles documenting the subjective and then objective improvement of nasal resistance in 10- to 20-year-olds using rhinometry.^{13,25} This author even noted an improvement of nocturnal enuresis with RME but never made the connection with OSAS. Kural et al²⁴ also reported improvement of nasal resistance in 10 prospectively studied children, 8 to 13 years old, treated with RME.

RME and associated orthodontic movements can also indirectly improve the oropharyngeal space by modifying the resting posture of the tongue.^{3,11,32}

Guilleminault et al,³³ in a recent review of 400 children with OSAS treated by ENT surgeons, found that about 20% had residual problems after undergoing ENT operations. These incidents were associated with abnormal craniofacial features and untreated nasal resistance. Guilleminault et al³⁴ and more recently

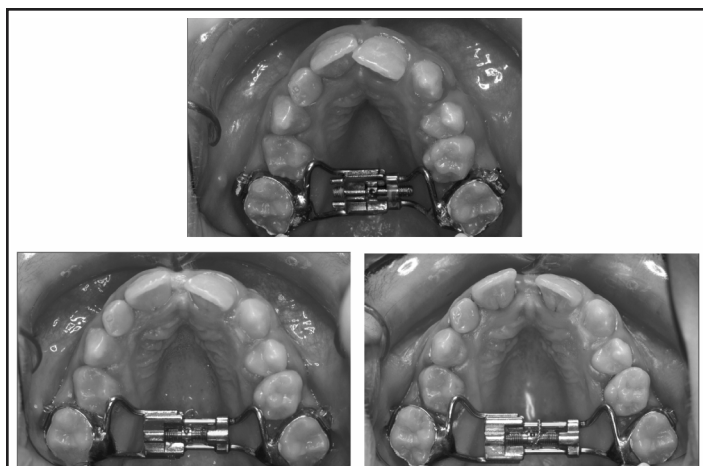


Figure 2—Occlusal sequence of treatment with rapid maxillary expansion, from crowding in the upper central incisors (upper left) to a wide space (lower image). Note how the palatal vault has changed.

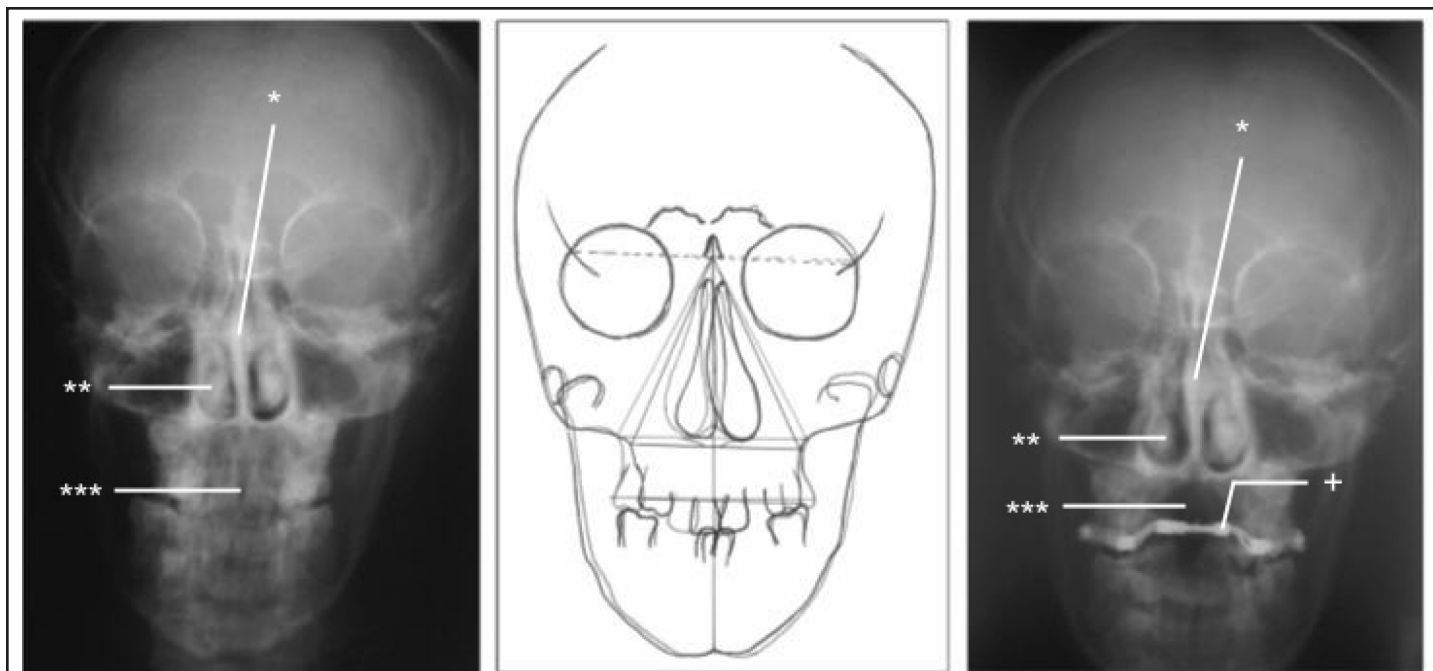


Figure 3—Posteroanterior cephalograms and cephalometric tracings before and after rapid maxillary expansion. The left image presents the anatomic structures before any distraction. As can be seen, the maxillary arch is narrow, the midpalatal suture cannot be seen, and the image of the inferior turbinate on the left is very close to the septum (*). On the right of the figure, the results at the end of the distraction can be seen: Note the anatomic changes—the skeletal expansion, caused by the maneuver, has opened the maxillary arch. The wide-open distractor (+) can be seen, and the midpalatal structure (***) is open. The inferior turbinate (**) on the left side is further apart from the septum compared to before treatment, indicating the changes that also occur in the nasal cavity. The drawing in between the 2 radiographs superimposes the 2 images to emphasize the pyriform opening and the widening of the nasal cavity with lateral and external displacement of the inferior nasal turbinates. The anatomic change occurs not only in the maxillary arch, but also in the nasal cavity.

Tasker et al³⁵ have shown that years after adenotonsillectomy performed in childhood, sleep-disordered breathing may be found and may mark the beginning of OSAS in adulthood. In Guilleminault et al's patients,³⁴ untreated craniofacial problems were at the basis of the long-term abnormal breathing during sleep.

As previously shown, RME does not always improve nasal airway and nasal resistance. Warren et al,²⁶ who performed a prospective study on 16 children aged 10 to 14 years, demonstrated improvement in only 45% of cases. One must remember that abnormal nasal resistance will have an impact not only on the maxilla, but also on the mandible. Despite a change in tongue position with RME, the gain may not be sufficient. The width of the mandible should be considered when RME is performed, as upper and lower teeth must be in apposition. Combined treatment on the maxilla and mandible, as recently presented by Guilleminault and Li,³⁶ may be necessary. However, RME may be a useful approach in dealing with abnormal breathing during sleep. It may be very helpful for patients with septal deviation, a problem that is often congenital and perhaps genetically determined. Septal surgery is not indicated in children, but abnormal nasal resistance can lead to maxillary deficiency early in life, and both problems may be addressed with RME.

Finally despite prior work that has been published on RME,^{13-15,25} this was the first prospective study on children with OSAS. We were cautious in our approach and selected only children without enlarged adenoids and tonsils. Our results indicate that RME is a very valid treatment of OSAS in children without enlarged tonsils and adenoids. It was, in our hands, without complication. Parents need to supervise their children's treatment and help maintain good dental hygiene during the distraction and as long as the distractor is in place.

We did not enlist children with enlarged adenoids and tonsils in this first study, and we cannot indicate if RME should be the first treatment approach even in children with adenotonsillar enlargement. This first study, however, allowed us to calculate the power needed for our on-going second prospective randomized study with RME and adenotonsillectomy as the 2 randomly selected first treatment approaches.

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