Original Research

Does rapid maxillary expansion improve nasal airway obstruction? A computer fluid dynamics study in patients with nasal mucosa hypertrophy and obstructed adenoids

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Declaration of interest

All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and none were reported.

Funding

Funding

This work was supported by KAKENHI from Japan Society for the Promotion of Science (No. 18K09860, 20K18766, 20K10230, 21K17189).

Highlights

- In normal children, nasal airway obstruction commonly improves following RME.
- In nasal mucosa hypertrophy children, RME improves nasal airway obstruction to some extent.
- RME is not effective in addressing obstruction among children with adenoids.

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Abstract

Introduction: Rapid maxillary expansion (RME) expands the maxillary dentition laterally and improves nasal airway obstruction. However, the incidence of nasal airway obstruction improvement following RME is approximately 60%. This study aimed to clarify the beneficial effects of RME on nasal airway obstruction in specific pathologic nasal airway diseases (nasal mucosa hypertrophy and obstructed adenoids) using computer fluid dynamics (CFD).

Methods: Sixty subjects (21 boys, mean age 9.1 years) were divided into three groups according to their nasal airway condition (control, nasal mucosa hypertrophy, and obstructed adenoids), and those requiring RME had cone- beam computed tomography (CBCT) images taken before and after RME. CBCT data were used to evaluate the nasal airway ventilation condition (pressure) using CFD and measure the cross-sectional area (CSA) of the nasal airway.

Results: The CSA of the nasal airway significantly increased after RME in all three groups. The pressures in the control and nasal mucosa groups significantly reduced after RME but did not change significantly in the adenoid group. The incidence of improvement in nasal airway obstruction in the control, nasal mucosa, and adenoid groups was 90%, 31.6%, and 23.1%, respectively.

Conclusions: The incidence of improvement in nasal airway obstruction after RME depends on the nasal airway condition (nasal mucosa hypertrophy and obstructed adenoids). In patients with non-pathologic nasal airway conditions, the obstruction may be sufficiently improved with RME. Furthermore, RME may be effective, to some extent, in treating nasal mucosa hypertrophy. However, RME was not effective in patients with nasal airway obstruction due to obstructed adenoids.

Key words: Rapid maxillary expansion, computer fluid dynamics, cone- beam computed tomography, nasal airway obstruction, nasal mucosa hypertrophy, obstructed adenoids

Introduction

Rapid maxillary expansion (RME) expands the maxillary dentition laterally and enlarges the nasal airway laterally; thus, an improvement in nasal airway obstruction is expected as a secondary effect.[1-3] Recently, the American Association of Orthodontists recommended that for obstructive sleep apnea (OSA) and orthodontics, the primary objective of RME is to normalize maxillary transverse deficiency and improve occlusion, whereas a secondary positive impact of increasing the upper airway volume and reducing nasal resistance may make it a plausible treatment modality in children with OSA.[4] Therefore, the improvement effect of RME on nasal airway obstruction is important. However, several studies estimate that the incidence of improvement of nasal airway obstruction following RME is approximately 60%.[1,3,5] In other words, not all cases show improvement in nasal airway obstruction following RME. Therefore, to improve nasal airway obstruction following RME, many studies[6-8] have investigated the use of expansion appliances[6,7] and an extended method.[8] However, it has been reported that the individual condition of nasal airway ventilation obstruction (anatomical, pathological, and physiologic) is important.[9] Therefore, when evaluating the improvement effects of RME on nasal airway obstruction, the study of the individual condition is necessary. However, the improvement effect of RME on nasal airway obstruction specifically due to nasal mucosa hypertrophy and obstructed adenoids remains uncertain. We aimed to analyze whether RME could help reduce nasal airway obstruction in pathologic nasal airway conditions. Therefore, this computer fluid dynamics (CFD) study aimed to determine the effects of RME on nasal airway obstruction in patients with nasal mucosa hypertrophy and obstructed adenoids.

Methods

This study was approved by the institutional review board of XXX university (180073 (657) Epi-ver. 8). Owing to the retrospective nature of the study, the need for obtaining informed consent

was waived.

Eligible subjects were selected retrospectively from the archives of a large private orthodontic office in Himeji, Japan, among those who underwent serial cone-beam computed tomography (CBCT) imaging before (T1) and after (T2) RME between October 2012 and September 2021. The ages of the subjects ranged from 7 to 12 years. The inclusion criteria were: 1) maxillary constriction and a need for maxillary expansion, requiring approximately 5 mm of maxillary expansion as part of their orthodontic treatment (no passive retention appliance was used before full orthodontic treatment); 2) no previous orthodontic treatment; and 3) no craniofacial or growth abnormalities. The exclusion criteria were as follows: 1) nasal mucosa hypertrophy combined with adenoid hypertrophy, 2) history of adenoidectomy or tonsillectomy, and 3) presence of systemic disease.

Thus, the patients were selected from a total of 542 patients. They were divided into three groups according to their nasal airway condition (Figure 1): 1) control group (20 subjects; six boys, mean age: 9.4 years, minimum age: 7.9 years, maximum age: 12.6 years); 2) mucosa group (20 subjects; eight boys, mean age: 9.1 years, minimum age: 7.1 years, maximum age: 10.9 years); and 3) adenoids group (20 subjects; seven boys, mean age: 9.0 years, minimum age: 7.8 years, maximum age: 10.5 years). The three groups were approximately matched in terms of sex, age, and dentition.

1) Control subjects were those without nasal mucosa hypertrophy or obstructed adenoids (Figure 1A). Absence of nasal mucosa hypertrophy was defined as the posterior nasal airway at the maxillary first molar on coronal section imaging showing no marked hypertrophy of the turbinate mucosa, whereas hypertrophy was defined as one or both turbinates being enlarged or fused.[10] On CBCT imaging, the absence of adenoids was defined as an obstruction of no more than 25%[11] in the space in the midsagittal plane between the posterior outline of the soft palate and the closest point on the adenoid tissue.

2) Subjects with nasal mucosa hypertrophy[12] without obstructed adenoids were classified as nasal mucosa subjects (Figure 1B). Nasal mucosa hypertrophy was defined as the posterior nasal airway at the maxillary first molar on coronal section imaging showing no marked hypertrophy of the turbinate mucosa, whereas hypertrophy was defined as one or both turbinates being enlarged or fused.[10]

3) Adenoids subjects were those with adenoid hypertrophy without apparent nasal mucosa hypertrophy (Figure 1C). Using the CFD study approach, previous studies have shown that 75% of adenoid obstructions had nasopharyngeal airway obstructions.[11] Adenoidal obstruction accounted for between 25% and 75%, corresponding to grades II and III; the nasopharyngeal airway did not show airway obstruction. Thus, obstructed adenoid was defined as more than 75% obstruction of the space in the midsagittal plane from the posterior outline of the soft palate to the closest point on the adenoid tissue on CBCT.

The participants were seated in a chair with the Frankfort horizontal plane parallel to the floor and scanned consistently during all CBCT scans (Alphard 3030; Asahi Roentgen, Kyoto, Japan).[13] CBCT was indicated for the patients in this study due to several reasons. CBCT scanning minimized radiation exposure. CBCT was performed before RME to examine the three-dimensional maxillofacial form and nasal and pharyngeal airways, paranasal sinus condition, as well as tooth problems. After RME, but before moving to phase II of the orthodontic treatment, we again examined the three-dimensional maxillofacial form, airways, and tooth conditions, especially tooth root resorption[14] and buccal alveolar bone reduction[15] due to RME. CBCT was set to a maximum of 80 kV, a maximum of 2 mA, and an exposure time of 17 s. Data were sent directly to a personal computer and stored in digital imaging and communications formats for medicine.

A volume- rendering software (INTAGE Volume Editor® Cybernet, Tokyo, Japan) was used to manually create 3D nasal airway (from the external nares to the choanae) images and evaluate

the intermaxillary molar width and nasal airway width (Figure 2A, B).[16] A 3D coordinate system and 3D image were constructed with a medical image analyzing system (Imagnosis VE®; Imagnosis, Kobe, Japan). Cross-sectional areas (CSAs) of the nasal airways were measured at the anterior and posterior regions of the nasal airway (Figures 2C, D, E).[16] The anterior CSA was defined as lying in the frontal plane through the anterior nasal spine (CSAa); the posterior CSA was defined as lying in the frontal plane through the maxillary molar palatal root apex (CSAp).

The nasal area was measured in the posterior region (Figure 2E)[17] and the nasal-mucosal ratio (NMR) was calculated (CSAp/nasal area). Since the shape of the nasal airway is complex, it is difficult to evaluate the degree of hyperplasia of the nasal mucosa quantitatively. Therefore, we calculated the ratio of the nasal airway cross-section to the cross-section of a nasal region as a quantitative evaluation method in this study; we considered this to be a value indicating the quantity of nasal mucosa as the nasal mucosa of the soft tissue is thought to account for most of the components, and a low rate is found in nasal mucosa hypertrophy.

For the measurement of nasal septum deviation, two landmarks were identified on the frontal view: (1) the junction of the perpendicular plate with the cribriform plate of the ethmoid bone, and (2) the junction of the vomer bone with the palatine bone (Figure 2F).[10] Nasal septum deviation was measured as the maximum difference between the actual septum and the hypothetical straight septum in coronal sections at the level of maximal septum deviation; we defined the presence or absence of nasal septum deviation as ≥ 2 mm and < 2 mm, respectively, based on a previous conventional study.[16]

The presence or absence of maxillary sinus mucosa hypertrophy was defined as the degree of thickening of the sinus mucosa by ≥ 2 mm and < 2 mm, respectively (Figure 2G).[18]

The 3D nasal airway model was then converted to a smoothed model via meshmorphing software (DEP Mesh Works/Morpher®; IDAJ, Kobe, Japan) without losing the subject-specific pattern of the airway shape. The models were exported to CFD software (Phoenics®; CHAM Japan,

Tokyo, Japan) in stereolithographic format. This software can simulate and evaluate various CFDs under a given set of conditions. The flow was assumed to consist of a Newtonian, homogeneous, and incompressible fluid.[19] Elliptic-staggered equations and the continuity equation were used in the study.[20] The CFD of the airways was analyzed under the following conditions: (1) volume of air flowing at a velocity of 200 mL/s in accordance with subjects' growth stage; (2) non-slippery wall surface; and (3) simulations repeated 1000 times to calculate the mean values. Convergence was evaluated by monitoring the magnitude of the absolute residual sources of mass and momentum, normalized to respective inlet fluxes. The iteration was continued until all residuals fell below 0.2%. The simulation estimated the maximum pressure and velocity of the nasal airway.[3]

According to Ohm's law, nasal airway resistance was calculated from air mass flow and the pressure difference between the external nares and choanae using postnasal rhinomanometry.[21] Airflow pressure and velocity were measured using the maximum value of the nasal airway. We used the nasal airway model's standardized gray level in CBCT (corresponding to Hounsfield Unit in CT) value to ensure that the resistance value of the nasal airway model obtained via CFD matched the nasal resistance value for rhinomanometry.[22]

Definition of nasal obstruction

A previous study[23] reported the nasal disease airway resistance in elementary school children to be 0.5 Pa/mL/s. Hence, we defined nasal airway obstruction as 0.5 Pa/mL/s, which corresponds to a resistance level equivalent to 100 Pa according to our flow quantity settings (200 mL/s). We concluded that nasal obstruction occurs when the negative pressure exceeds 100 Pa. Moreover, complete obstruction was assumed (3D obstruction) when the continuity of the bilateral nasal meatus of the 3D nasal airway model was broken.[3]

To ensure reliability, all measurements were repeated by the same evaluator (RSI) after 1

week. Subsequently, two authors (TI and RSI) confirmed the accuracy of these measurements. If additional measurements were needed, the same evaluator (RSI) performed the measurements again; the Dahlberg formula[24] was used to calculate the measurement error. The measurement error of the images obtained in this study showed that the intermaxillary molar width, nasal airway width, nasal area, nasal CSA at the anterior nasal spine, nasal CSA at the maxillary first molar, nasal septum deviation, maximum nasal airway pressure, and maximum nasal airway velocity were 0.055 mm, 0.045 mm, 1.20 mm^2, 0.340 mm^2, 0.524 mm^2, 0.023 mm, 1.823 Pa°, and 0.423 m/s, respectively. According to all repeated analyses, the method error was considered to be negligible.

Statistical analysis

ANOVA and the Kruskal–Wallis tests were used to detect significant differences in measurement values among the groups, and post-hoc testing with Bonferroni correction was used. The significance of treatment changes (T1 and T2) was assessed via paired *t*-test and Wilcoxon rank- sum test.

Fisher's exact test clarified the incidence of nasal airway obstruction and the improvement in the incidence of nasal airway obstruction following RME in the three study groups. In addition, it also determined the incidence of nasal septum deviation and maxillary sinus mucosa hypertrophy following RME in the three groups, the presence or absence of nasal obstruction, and whether nasal airway obstruction improved after RME.

Spearman correlation coefficients (rs) were calculated to evaluate the relationships among the CFD values, CSA, and NMR at each stage and across all stages. For all tests, $P < 0.05$ was considered statistically significant.

In accordance with our hypothesis that RME improves nasal airway ventilation conditions, we performed a sample size calculation based on the difference in treatment changes of nasal airway

ventilation conditions following RME.[16] To calculate the β error, a power analysis using G*power 3.1.9.7 was performed (1−β error = 0.80, α = 0.05, two- tailed test); an adequate sample size was determined to be 18 subjects.

Results

The nasal airway width, maxillary molar width, and nasal area were significantly enlarged following RME in each of the three groups. However, there were no significant differences among the three groups at T1 (9.1±1.1 years), T2 (10.9±1.2 years), and T1–T2 (1.8±1.0 years) (Table 1).

The CSAa of the three groups was significantly higher following RME. However, there were no significant differences among the groups at T1 and T2 (Table 1). In addition, the CSAp of the three groups was significantly increased following RME. Treatment changes in the CSAp were not different among the groups. However, the CSAp of the mucosa group was significantly smaller than that of the control and adenoids groups at T1 and T2.

The NMR did not significantly change in the control or adenoids groups following RME (NMR, around 30%; adenoids, 27%; Table I). However, the NMR in the mucosa group improved significantly from 17% to 22% following RME. The NMR in the mucosa group was significantly smaller than that in the control and adenoids groups at T1 and T2. However, treatment change values in the mucosa group were significantly greater than those in the control and adenoids groups.

Nasal septum deviation in the mucosa group was significantly larger than that in the control and adenoids groups at T1 and T2 (Table I). Nasal septum deviation and maxillary sinus mucosa hypertrophy were significantly different among the three groups (Table IV). However, the distribution of patients with nasal obstruction was not significantly different from that of those with nasal septum deviation and maxillary sinus hypertrophy (Table V). Furthermore, the effect of RME on nasal obstruction improvement was not significantly different between maxillary sinus mucosa

hypertrophy and nasal septum deviation (Table VI). Since the ostia was broken and did not continue between the nasal and paranasal airways, we considered that the 3D nasal airway model of patients with no ostia would not show the paranasal airway. No ostia were observed in eight patients (five in the mucosa and three in the adenoid group, and only one adenoid patient showed improved nasal airway obstruction by RME).

Maxillary sinus mucosa hypertrophy was observed before RME in 14 out of 20 patients (10%) and nine of 20 patients (45%) after RME in the mucosa group; in the adenoid group, it was observed before RME in three out of 20 patients (15%) and two of 20 patients (10%) after RME. However, in the control group, it was not observed before and after RME, and there was no significant difference between nasal airway obstruction and maxillary sinus mucosa hypertrophy before and after RME.

The pressure and velocity of the control and mucosa groups were significantly reduced following RME; however, those of the adenoids group did not change (Table 1). The pressure and velocity in the mucosa group were significantly greater than those in the control and adenoids groups at T1 and T2. However, the pressure and velocity were not significantly different among the groups.

Regarding the incidence of nasal obstruction at T1, 10 of the 20 subjects in the control group had an obstruction detected by 3D reconstruction or computational fluid dynamics (50%; Table 2); following RME, 9 of the 10 subjects had improvement in their nasal airway obstruction at T2 (improvement rate: 90%). In contrast, 19 (95%) of the 20 mucosa group subjects had nasal airway obstruction at T1, and the incidence of nasal obstruction improved following RME at T2 in six of the 19 subjects (improvement rate: 31.6%). In the adenoids group, 13 (65%) of the 20 subjects had nasal airway obstruction at T1, and three of these 13 subjects had improved nasal airway obstruction following RME at T2 (improvement rate: 23.1%). The improvement rates were significantly different between the groups.

There were no significant associations between CSAa and the pressure and velocity at each

stage, indicating the nasal airway ventilation condition (Table 3). However, a moderately significant negative correlation was identified between CSAp and the nasal airway ventilation condition at each stage (Table 3), and the nasal airway ventilation condition showed a significant negative correlation with NMR. There were significant negative associations between pressure and CSAa and CSAp at all stages (Figure 3).

Discussion

The present study showed that nasal airway ventilation conditions were affected by the specific clinical condition (nasal mucosa hypertrophy or obstructed adenoids) of the nasal airway. Furthermore, the improvement in nasal airway ventilatory conditions following RME was dependent on the underlying clinical condition (nasal mucosa hypertrophy or obstructed adenoids) of the nasal airway. In the absence of nasal mucosa hypertrophy and obstructed adenoids, the improvement in nasal airway obstruction following RME was high. Conversely, it was found that the improvement effect was low when nasal mucosa hypertrophy or obstructed adenoids were present.

Nasal airway cross-sectional area

From previous studies, it was decided that the ventilation conditions of the airway are greatly influenced by airway form.[11,25,26] Regarding the nasal airway, Garcia et al.[27] reported that the cross-sectional area of the nasal airway of the nasal valve, which is close to the CSAa evaluated in the present study, is the smallest area in normal adults; they suggested that the CSA of this part greatly influences the nasal airway ventilation conditions. However, in the present study (Table 1), the three groups had different nasal airway ventilation conditions (pressure and velocity) but no difference in CSAa; there were differences in CSAp. The anterior nasal airway, located in the proximal portion of the nasal cavity, is covered by epithelium and has no erectile tissue, whereas the posterior nasal

airway is covered by mucosa. The posterior nasal airway is considered susceptible to nasal mucosa hypertrophy and obstructed adenoids. In the presence of nasal airway obstruction, these results suggest that the posterior nasal airway influences nasal airway ventilation conditions (Figure 3).[28]

In a study of nasal airway CSA and nasal airway maximum pressures in 11- and 13-year-olds, the cross-sections o the posterior nasal airway in healthy children ranged from 260–280 mm^2, and the maximum pressure value ranged from 40–80 Pa (corresponding to 0.2–0.4 Pa/mL/s).[16] Regarding the nasal airway cross-section in children with cleft lip and palate, the CSA of 207 mm^2 was associated with a pressure of 291 Pa (corresponding to 1.46 Pa/mL/s), and the area of 270 mm^2 associated with expansion following RME was associated with a pressure of 49 Pa (corresponding to 0.25 Pa/mL/s).[16]

Furthermore, Holsbeke et al.[29] reported that the CSA of the posterior nasal airway of normal 6-year-old children was 317 mm^2, whereas it was 171 mm^2 in children with OSA and upper airway ventilatory obstruction (there may not be nasal airway obstruction in all cases).

Due to the complicated cross-sectional form of the posterior region of the nasal airway, a strong association between the cross-sectional area and nasal airway resistance[25] of the nasal valve (rs = 0.816) was not found in the current study in terms of CSAp and nasal airway pressure (corresponding to nasal airway resistance) (rs = -0.569).

From these reports[29] and the results of the present study, we concluded that the threshold of nasal airway obstruction (more than 100 Pa, corresponding to 0.5 Pa/mL/s) [30] of CSAp in children was approximately 250 mm^2 (Figure 3B).

Treatment change and nasal mucosa rate

NMR (CSAp/nasal area, i.e., the nasal-mucosal ratio) was used to evaluate nasal mucosa hypertrophy in each group (Figure 4). The NMR of the normal group did not change and

was a relatively high value (approximately 30%). Since they did not have nasal mucosa hypertrophy and the NMR values were already high, there was little scope for change. The NMR of the adenoids group was maintained at around 27% following RME. However, the NMR of the nasal mucosa group significantly increased following RME, from 17.4% to 22.0%. Nasal mucosa hypertrophy decreased, which was associated with a reduction in velocity from 34.8 m/s to 17.6 m/s, and mechanical stimulation of the nasal mucosa by intense airflow at the nasal airway ventilation may have relieved mucosal inflammation and hyperplasia of the nasal mucosa. However, the NMR remained at 22.0%. An improvement in upper airway obstruction following oral myofunctional therapy (MFT) has been recently reported.[31] Therefore, other than otolaryngology treatment, MFT may also be effective.

Furthermore, in the adenoids group, the nasal airway cross-section was significantly expanded in terms of both CSAa and CSAp. However, we hypothesized that the improvement in nasal airway ventilation following RME was absent because the NMR and CFD did not show significant improvements.[32-34] In terms of adenoids and nasal airway relationships, obstructed adenoids have been linked to nasal mucosa hyperplasia in a previous study, and adenoidectomy has been linked to improved nasal airway ventilation.[35] Therefore, nasal airway obstruction may be caused by obstructed adenoids. In the case of conventional examinations[36,37] for the degree of nasal airway ventilation, nasal airway ventilation may be affected by obstructed adenoids for an anatomical reason. Therefore, evaluating the ventilation conditions for only the nasal airway was difficult. However, in the present study, we were able to evaluate only the nasal airway in the case of obstructed adenoids as it was derived from the CFD evaluation of a 3D nasal airway model except for the obstructed adenoids. Improvement of nasal airway obstruction was not observed following RME in the presence of obstructed adenoids; this may be because nasal breathing took place in a non-physiological situation.[32] Furthermore, because the nasopharynx becomes constricted when we perform nasal breathing in the presence of obstructed adenoids, very fast airflow occurs when air

passes the nasopharyngeal airway (Figure 5). Therefore, we performed nasal mucosa mechanical stimulation,[12] and the possibility that the nasal mucosa was thickened by those effects was considered. Thus, when adenoids were detected, it was hard to obtain an improvement in nasal airway ventilation conditions following RME, and otolaryngology treatment for the adenoids was considered necessary.[32]

Differences in the improvement of nasal airway obstruction following RME

The sizes of the nasal airways before the expansion and improvement in nasal airway obstruction following RME were different (Figure 6); thus, the improvement rates of each group in this study were different.

Regarding the nasal airway CSA (before 11.1 years and after 13.4 years), in a previous study,[16] the anterior and posterior regions of the nasal airway increased from 186 to 198 mm^2 and from 259 to 284 mm^2, respectively. However, since the age of the patients differed slightly in this previous study,[16] the increment in anterior and posterior nasal airway CSAs after RME was 25.0 and 21.7 mm^2 in the control group, 19.3 and 35.3 mm^2 in the mucosa group, and 19.7 and 2.6 mm^2 in the adenoid group, respectively. Therefore, an enlargement effect of approximately 20 mm^2 was observed in the anterior region of the nasal airway in all three groups after RME; the enlargement effect was approximately 20–35 mm^2 in the posterior region in the control and mucosa groups, but there was no enlargement in the adenoid group. Thus, the posterior CSA reflected the ventilation condition of the nasal airway.

The CSAp of the control group was relatively large, and the nasal airways were expanded smoothly; as a result, the CSAp was of a sufficient size for nasal airway obstruction to improve. Therefore, the improvement rate increased. There was one case of non-improvement in the normal group; this subject had a deviated nasal septum, which we believe may be the cause for non-

improvement in the nasal obstruction. Future studies should evaluate cases with deviated nasal septums in detail.

The nasal mucosa group had small CSAp values before the expansion. Therefore, although the CSAp of the mucosa group was markedly increased following RME, the CSAp did not reach the size necessary to improve nasal airway ventilation, explaining the low improvement rate.

The CSAp of the adenoids group was intermediate in size before expansion. However, the effect of RME on improvement was insufficient. Therefore, expansion of the nasal airway did not occur until symptom improvement was achieved, and this group was associated with a low improvement rate.

Therefore, we concluded that nasal septum deviation and maxillary sinus mucosa hypertrophy were unlikely to have a significant impact on the goal of the current study, i.e., improvement of nasal airway ventilation condition. However, the result of our present study, which required the enrollment of patients undergoing RME, might be different from that of general cases. Therefore, we must continue to investigate these factors in the future. Since the ostia was broken and did not continue between the nasal and paranasal airways, we considered that the 3D nasal airway model of patients with no ostia would not show the paranasal airway. No ostia were observed in eight patients (five in the mucosa and three in the adenoid group, and only one adenoid patient improve nasal airway obstruction by RME).

Although children with mucosa hypertrophy had a significantly greater degree of maxillary sinus hypertrophy, the presence of the maxillary sinus mucosa hypertrophy did not improve the nasal airway ventilation condition after RME Therefore, we believe that there was negligible effect on maxillary sinus hypertrophy in this study, which evaluated the improvement effect of nasal airway ventilation after RME. Therefore, we considered that maxillary sinus hypertrophy did not have an improvement effect on nasal airway ventilation in this study. However, these are studies that only

included children who required RME, and other cases will need to be investigated in the future.

Clinical implications

From this study, we were able to elucidate improvements in nasal airway obstruction following RME in subjects with nasal mucosa and adenoidal hypertrophy. In other words, following RME, effective improvement of the nasal airway obstruction occurred in the absence of hyperplasia of the nasal mucosa and obstructed adenoids. Moreover, even in the case of mucosa hypertrophy, there was an improvement in constant nasal airway obstruction following RME, and improvements in nasal airway obstruction should arise from additional treatment (otolaryngological treatment, MFT, and other treatments). In contrast, improvement in the nasal airway obstruction following RME cannot be expected in cases with grade 4 adenoids (≥ 75%); a medical examination and an adenoidectomy by an otolaryngologist is required in such cases. Notably, we were also able to identify a reference value for the CSA of the posterior nasal airway necessary for improvements in nasal airway ventilation.

Limitations

The limitations of this study include the small sample size and the potential bias of the included subjects. However, because each variable recognized a statistically significant difference, it was assumed that the effect on results was minor. Therefore, it will be necessary to perform a randomized controlled trial examining real cases in the future. Furthermore, it is necessary to evaluate and compare the nasal airway improvement effects of otolaryngological treatments and MFT in subjects with nasal mucosa thickening and that of adenoidectomy in subjects with adenoids.[38] Because this is a retrospective study, this study did not include an examination by an otolaryngologist. Therefore, future research on RME with an otolaryngologist regarding clinical

manifestations such as nasal mucosa hypertrophy or obstructed adenoids will be required.

Conclusions

In our RME study on children with nasal airway obstruction, the nasal airway ventilation conditions were affected by the specific clinical condition (nasal mucosa hypertrophy and obstructed adenoids) of the nasal airway. Improvement in nasal airway obstruction following RME was influenced by the clinical condition (nasal mucosa hypertrophy and obstructed adenoids) of the nasal airway, too. In cases without nasal mucosa hypertrophy or obstructed adenoids, the improvement in the nasal airway obstruction following RME was 90%. Conversely, the improvement effect was low in cases with nasal mucosa hypertrophy and obstructed adenoids (31.6% and 23.1%, respectively). Nasal airway obstruction due to obstructed adenoids did not respond to RME.

Acknowledgments

We would like to thank Editage (www.editage.jp) for English language editing.

Funding

This work was supported by KAKENHI from Japan Society for the Promotion of Science (No. 18K09860, 20K18766, 20K23116, 20K10230, 21K17189).

References

1. Doruk C, Sokucu O, Sezer H, Canbay EI. Evaluation of nasal airway resistance during rapid maxillary expansion using acoustic rhinometry. Eur J Orthod 2004;26:397-401.

2. Enoki C, Valera FC, Lessa FC, Elias AM, Matsumoto MA, Anselmo-Lima WT. Effect of rapid maxillary expansion on the dimension of the nasal cavity and on nasal air resistance. Int J Pediatr

Otorhinolaryngol 2006;70:1225-1230.

3. Iwasaki T, Saitoh I, Takemoto Y, Inada E, Kanomi R, Hayasaki H et al. Improvement of nasal airway ventilation after rapid maxillary expansion evaluated with computational fluid dynamics. Am J Orthod Dentofacial Orthop 2012;141:269-278.

4. DiCosimo C, Alsulaiman AA, Shah C, Motro M, Will LA, Parsi GK. Analysis of nasal airway symmetry and upper airway changes after rapid maxillary expansion. Am J Orthod Dentofacial Orthop 2021;160:695-704.

5. Oliveira De Felippe NL, Da Silveira AC, Viana G, Kusnoto B, Smith B, Evans CA. Relationship between rapid maxillary expansion and nasal cavity size and airway resistance: short- and long-term effects. Am J Orthod Dentofacial Orthop 2008;134:370-382.

6. Araújo MC, Bocato JR, Oltramari PV, de Almeida MR, Conti AC, Fernandes TM. Tomographic evaluation of dentoskeletal effects of rapid maxillary expansion using Haas and Hyrax palatal expanders in children: A randomized clinical trial. J Clin Exp Dent 2020;12:e922-e930.

7. Weissheimer A, de Menezes LM, Mezomo M, Dias DM, de Lima EM, Rizzatto SM. Immediate effects of rapid maxillary expansion with Haas-type and hyrax-type expanders: a randomized clinical trial. Am J Orthod Dentofacial Orthop 2011;140:366-376.

8. Lemos Rinaldi MR, Azeredo F, Martinelli de Lima E, Deon Rizzatto SM, Sameshima G, Macedo de Menezes L. Cone-beam computed tomography evaluation of bone plate and root length after maxillary expansion using tooth-borne and tooth-tissue-borne banded expanders. Am J Orthod Dentofacial Orthop 2018;154:504-516.

9. Leboulanger N. Nasal obstruction in children. Eur Ann Otorhinolaryngol Head Neck Dis 2016;133:183-186.

10. Baddam P, Thereza-Bussolaro C, Flores-Mir C, Graf D. Nasal cavity structural anomalies in children and adolescents at high risk of sleep-disordered breathing: An exploratory cone-beam

computed tomography study. Am J Orthod Dentofacial Orthop 2021;160:533-543.e532.

11. Iwasaki T, Sugiyama T, Yanagisawa-Minami A, Oku Y, Yokura A, Yamasaki Y. Effect of adenoids and tonsil tissue on pediatric obstructive sleep apnea severity determined by computational fluid dynamics. J Clin Sleep Med 2020;16:2021-2028.

12. Iwasaki T, Papageorgiou SN, Yamasaki Y, Ali Darendeliler M, Papadopoulou AK. Nasal ventilation and rapid maxillary expansion (RME): a randomized trial. Eur J Orthod 2021;43:283-292.

13. Iwasaki T, Suga H, Minami-Yanagisawa A, Hashiguchi-Sato M, Sato H, Yamamoto Y et al. Upper airway in children with unilateral cleft lip and palate evaluated with computational fluid dynamics. Am J Orthod Dentofacial Orthop 2019;156:257-265.

14. Baysal A, Karadede I, Hekimoglu S, Ucar F, Ozer T, Veli I et al. Evaluation of root resorption following rapid maxillary expansion using cone-beam computed tomography. Angle Orthod 2012;82:488-494.

15. Sperl A, Gaalaas L, Beyer J, Grünheid T. Buccal alveolar bone changes following rapid maxillary expansion and fixed appliance therapy. Angle Orthod 2021;91:171-177.

16. Iwasaki T, Yanagisawa-Minami A, Suga H, Shirazawa Y, Tsujii T, Yamamoto Y et al. Rapid maxillary expansion effects of nasal airway in children with cleft lip and palate using computational fluid dynamics. Orthod Craniofac Res 2019;22:201-207.

17. Smith T, Ghoneima A, Stewart K, Liu S, Eckert G, Halum S et al. Three-dimensional computed tomography analysis of airway volume changes after rapid maxillary expansion. Am J Orthod Dentofacial Orthop 2012;141:618-626.

18. Capelli M, Gatti P. Radiological Study of Maxillary Sinus using CBCT: Relationship between Mucosal Thickening and Common Anatomic Variants in Chronic Rhinosinusitis. J Clin Diagn Res 2016;10:Mc07-mc10.

19. De Backer JW, Vanderveken OM, Vos WG, Devolder A, Verhulst SL, Verbraecken JA et al.

Functional imaging using computational fluid dynamics to predict treatment success of mandibular advancement devices in sleep-disordered breathing. J Biomech 2007;40:3708-3714.

20. Gamiño B, Aguillón J. Numerical simulation of syngas combustion with a multi-spark ignition system in a diesel engine adapted to work at the Otto cycle. Fuel 2010;89:581-591.

21. Iwasaki T, Sato H, Suga H, Takemoto Y, Inada E, Saitoh I et al. Relationships among nasal resistance, adenoids, tonsils, and tongue posture and maxillofacial form in Class II and Class III children. Am J Orthod Dentofacial Orthop 2017;151:929-940.

22. Yanagisawa-Minami A, Sugiyama T, Iwasaki T, Yamasaki Y. Primary site identification in children with obstructive sleep apnea by computational fluid dynamics analysis of the upper airway. J Clin Sleep Med 2020;16:431-439.

23. Kobayashi R, Miyazaki S, Karaki M, Kobayashi E, Karaki R, Akiyama K et al. Measurement of nasal resistance by rhinomanometry in 892 Japanese elementary school children. Auris Nasus Larynx 2011;38:73-76.

24. Dahlberg G. Stastistical methods for medical and biological syudents. London: Gerge Allen and Unwin; 1940.

25. Yajima Y, Oshima M, Iwai T, Kitajima H, Omura S, Tohnai I. Computational fluid dynamics study of the pharyngeal airway space before and after mandibular setback surgery in patients with mandibular prognathism. Int J Oral Maxillofac Surg 2017;46:839-844.

26. Shirazawa Y, Iwasaki T, Ooi K, Kobayashi Y, Yanagisawa-Minami A, Oku Y et al. Relationship between pharyngeal airway depth and ventilation condition in mandibular setback surgery: A computational fluid dynamics study. Orthod Craniofac Res 2020;23:313-322.

27. Garcia GJM, Hariri BM, Patel RG, Rhee JS. The relationship between nasal resistance to airflow and the airspace minimal cross-sectional area. J Biomech 2016;49:1670-1678.

28. Wandalsen GF, Mendes AI, Solé D. Correlation between nasal resistance and different acoustic

rhinometry parameters in children and adolescents with and without allergic rhinitis. Braz J Otorhinolaryngol 2012;78:81-86.

29. Van Holsbeke C, Vos W, Van Hoorenbeeck K, Boudewyns A, Salgado R, Verdonck PR et al. Functional respiratory imaging as a tool to assess upper airway patency in children with obstructive sleep apnea. Sleep Med 2013;14:433-439.

30. Crouse U, Laine-Alava MT, Warren DW. Nasal impairment in prepubertal children. Am J Orthod Dentofacial Orthop 2000;118:69-74.

31. Huang YS, Hsu SC, Guilleminault C, Chuang LC. Myofunctional Therapy: Role in Pediatric OSA. Sleep Med Clin 2019;14:135-142.

32. Brinckmann CA, Scapin FP, Bernd LA, Lubianca Neto JF. Effect of improved nasal airflow after adenoidectomy on nasal erectile tissue. Arch Otolaryngol Head Neck Surg 2008;134:832-836.

33. Shin SH, Heo WW. Effects of unilateral naris closure on the nasal and maxillary sinus mucosa in rabbit. Auris Nasus Larynx 2005;32:139-143.

34. Maurizi M, Paludetti G, Ottaviani F, Almadori G, Falcetti S. Mucociliary function and nasal resistance evaluation before and after adenoidectomy. Int J Pediatr Otorhinolaryngol 1986;11:295-300.

35. Calvo-Henriquez C, Branco AM, Lechien JR, Sandoval-Pacheco V, Maldonado-Alvarado B, Maza-Solano J et al. Assessing the effect of adenoidectomy on nasal resistance and airflow. A systematic review and meta-analysis. Int J Pediatr Otorhinolaryngol 2021;151:110969.

36. Clarke RW, Jones AS. The limitations of peak nasal flow measurement. Clin Otolaryngol Allied Sci 1994;19:502-504.

37. Cakmak O, Coskun M, Celik H, Buyuklu F, Ozluoglu LN. Value of acoustic rhinometry for measuring nasal valve area. Laryngoscope 2003;113:295-302.

38. Calvo-Henriquez C, Capasso R, Chiesa-Estomba C, Liu SY, Martins-Neves S, Castedo E et al. The role of pediatric maxillary expansion on nasal breathing. A systematic review and metanalysis.

Int J Pediatr Otorhinolaryngol 2020;135:110139.

Figure captions

Figure 1. Condition of the nasal airway.

A: control subjects, defined as not having nasal mucosa hypertrophy or adenoids. B: nasal mucosa subjects, defined as having nasal mucosa hypertrophy without adenoids. Nasal mucosa hypertrophy was considered to be present when one or both turbinates were enlarged and fused. C: adenoid subjects, defined as having adenoid hypertrophy without apparent nasal mucosa hypertrophy.

Figure 2. Measurement of the intermaxillary molar width, nasal airway width, nasal cross-sectional area, nasal area, nasal septum deviation, and maxillary sinus mucosa hypertrophy.

A, Intermaxillary molar width, the intermaxillary first molar width at the narrowest portion.

B, Nasal airway width, the widest portion of the nasal aperture.

C, Definition of nasal airway cross-sectional area (CSA). a, measurement site of the anterior CSA at the anterior nasal spine; b, measurement site of the posterior CSA at the maxillary first molar.

D, CSAa, the anterior CSA, inside the red line.

E, CSAp, the posterior CSA, inside the red line; the nasal area, inside the yellow line.

F, Nasal septum deviation was defined as the maximum difference between the actual septum and hypothetical straight septum in coronal sections at the level of maximal septum deviation.

G, Presence or absence of maxillary sinus mucosa hypertrophy was defined as the degree of thickening of the sinus mucosa $\geq$ 2 mm and < 2 mm, respectively.

Figure 3. Relationships between the cross-sectional area (CSA) of the nasal airway and pressure.

A: Relationship between pressure and CSAa. A weak but significant association was shown. CSAa: cross-sectional area of the nasal airway at the anterior nasal spine.

B: Relationship between Pmax and CSAp. A medium significant association was shown. CSAp was 250 mm^2 or more, and pressure was shown to be 100 Pa or less. CSAp: cross-sectional area of the nasal airway at the maxillary first molar.

Figure 4. Change of the nasal airway following RME. Upper: before expansion, lower: After expansion.

A: Control group; the nasal airway expanded following RME.

B: Nasal mucosa hypertrophy group; the nasal airway became constricted by nasal mucosa hypertrophy before expansion. However, the hypertrophy of the nasal mucosa was relieved after expansion and showed expansion of the nasal airway.

C: Adenoids group; there were no major changes in the size of the nasal airway following RME.

Figure 5. Airflow of the nasal airway without and with adenoid hypertrophy.

A: Adenoid hypertrophy.

B: Model with adenoids. Airflow showed a faster posterior part (red arrow). Due to the fast airflow, the site provides strong mechanical stimulation to the nasal mucosa.

C: Model without adenoids. The airflow was relatively slow in all parts.

Figure 6. The difference in improvements in the nasal airway ventilation condition following RME. A representative example is displayed (upper, before expansion; lower, after expansion).

A: Control, B: Nasal mucosa hypertrophy, C: Adenoids.

Before RME, all cases had a pressure of 100 Pa or more and showed nasal airway obstruction; after

RME, the Control subjects had a pressure of 100 Pa or less and showed improvement in the nasal airway obstruction. However, the nasal mucosa hypertrophy and adenoid subjects did not show improvements in the nasal airway obstruction, with the pressure remaining 100 Pa or more.

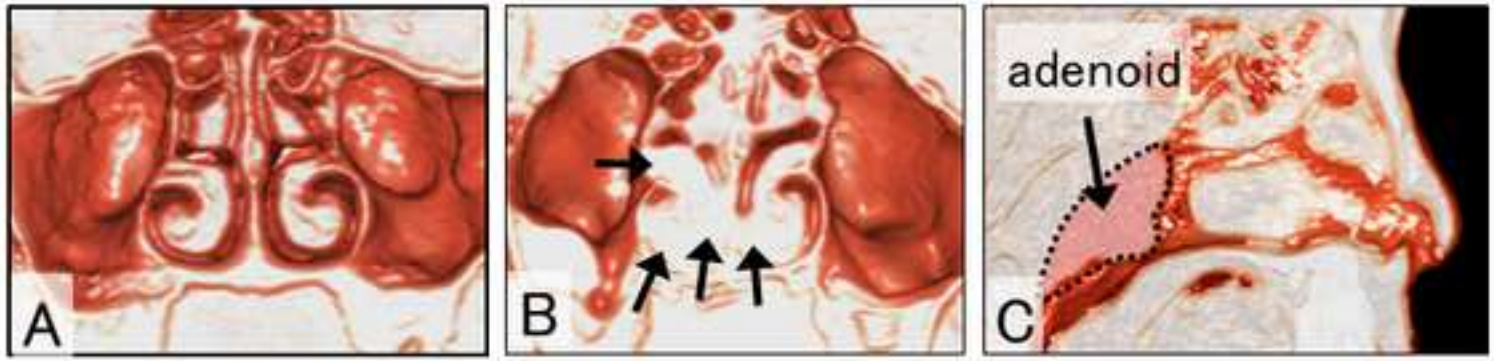

Fig 1

Figure 2

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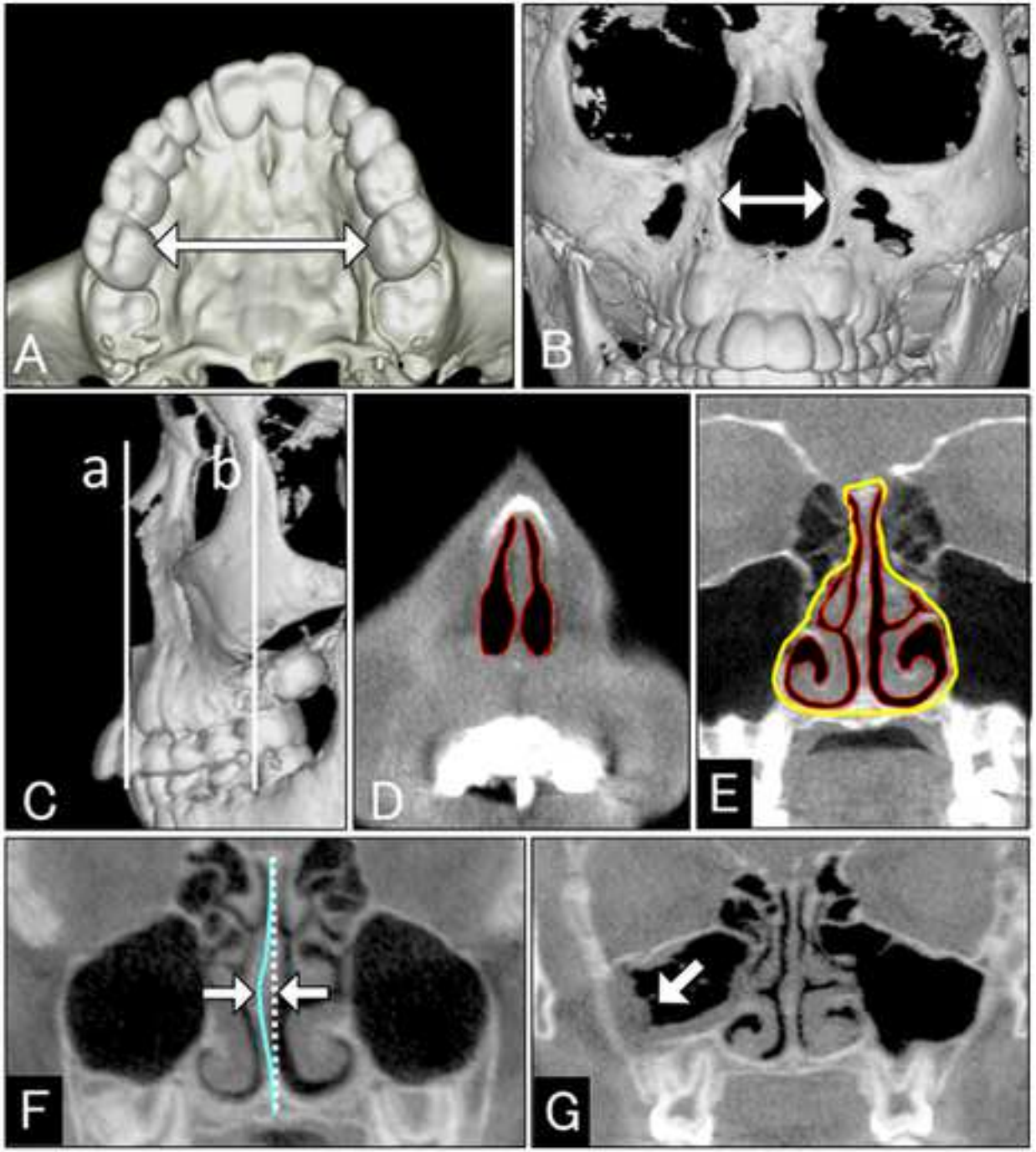

Fig 2

Figure 3

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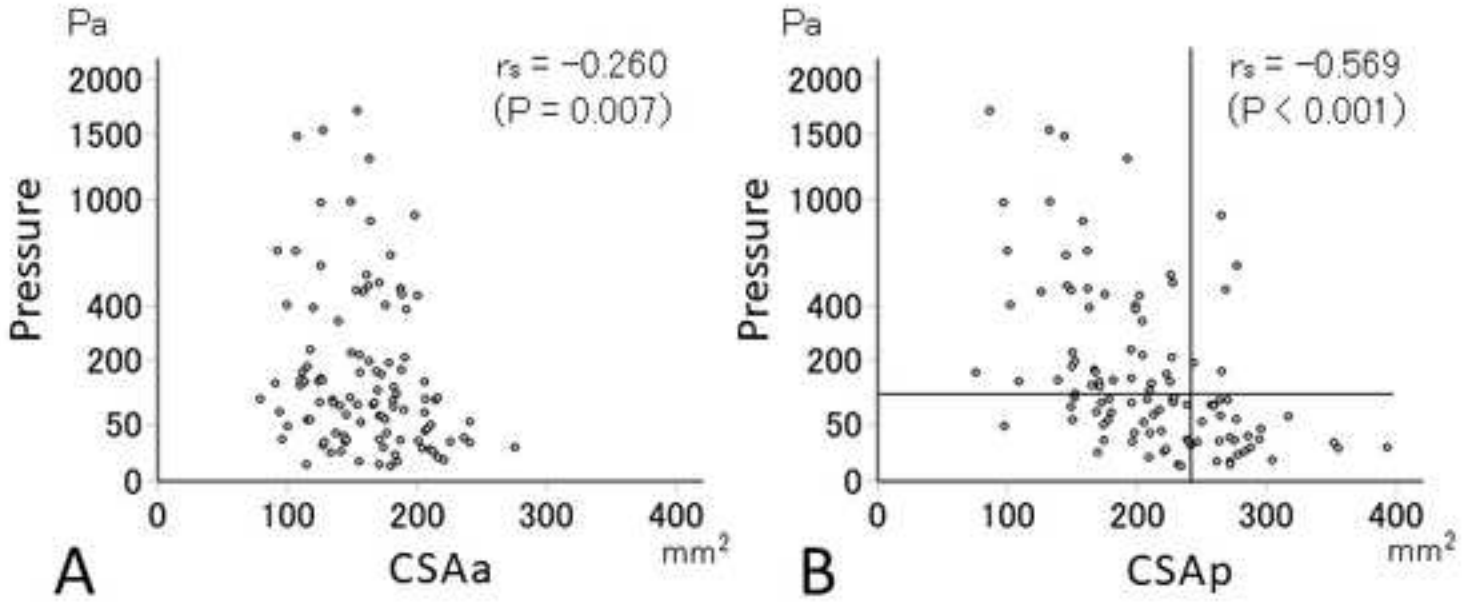

Fig 3

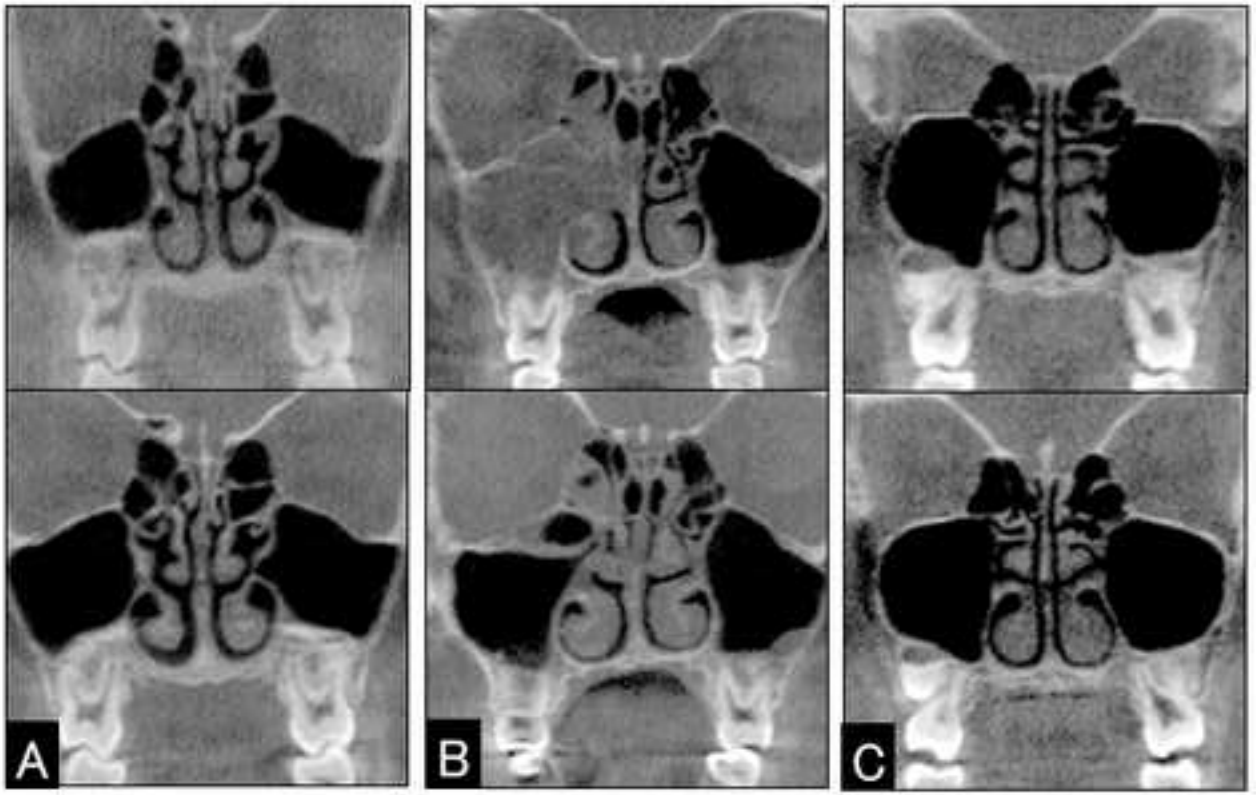

Fig 4

Figure 5

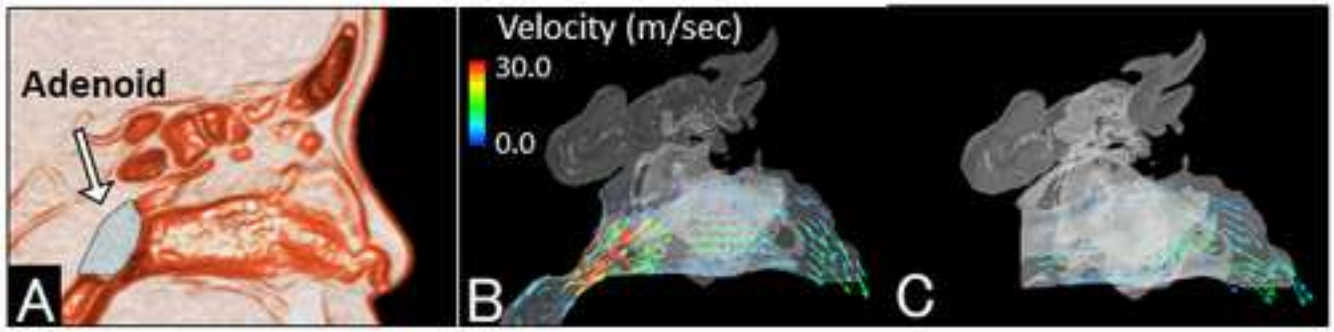

Fig 5

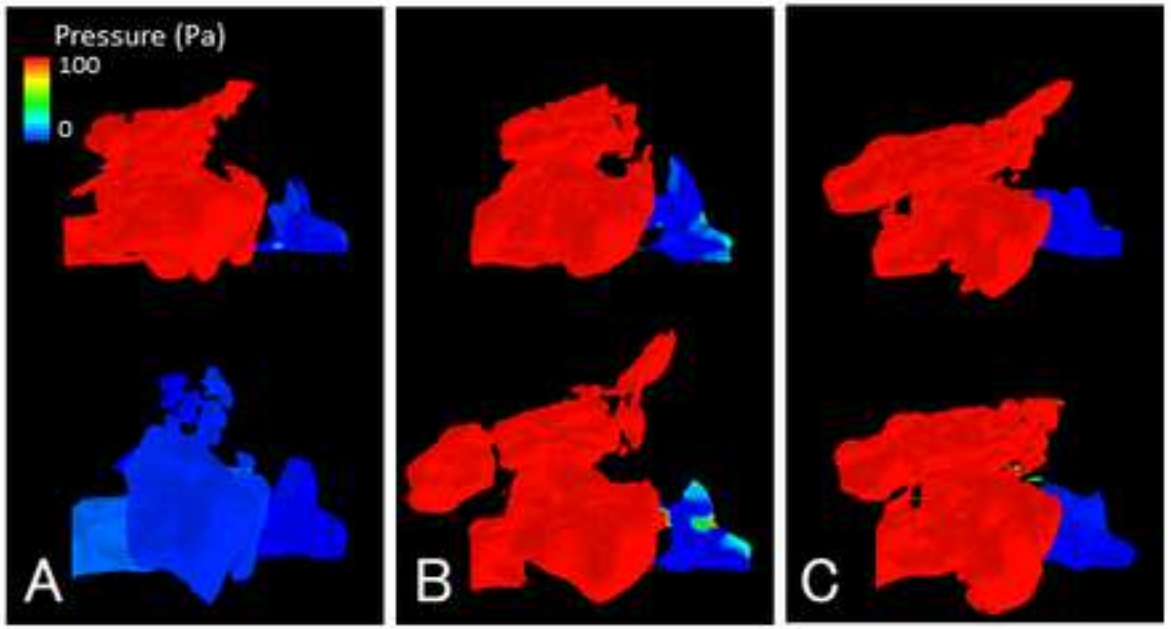

Fig 6

Table 1 Click here to access/download;Table;Table 1.xlsx

Table I Comparison of the three groups

	Control group (n = 20)			Nasal mucosa hypertrophy group (n = 20)			Adenoid group (n = 20)			ANOVA or Kruskal–Wallis	post hoc Bonferroni
	mean	SD		mean	SD		mean	SD		P	
age (year)											
T1	9.44	1.36		9.05	1.01		8.90	0.79		NS	
T2	11.03	1.49		11.06	1.26		10.65	0.91		NS	
T2–T1	1.59	0.87		2.01	1.08		1.76	1.05		NS	
Maxillary molar width (mm)											
T1	34.4	2.9		34.6	2.4		33.6	2.5		NS	
T2	38.3	3.4		39.0	2.6		38.2	3.0		NS	
T2–T1	3.9	1.9	*	4.4	1.7	*	4.6	1.7	*	NS	
Nasal airway width (mm)											
T1	28.5	1.4		28.4	2.0		27.9	2.9		NS	
T2	30.9	2.0		30.8	1.7		30.4	2.8		NS	
T2–T1	2.4	1.3	*	2.4	1.4	*	2.5	1.5	*	NS	
Nasal septum deviation (mm)											
T1	0.6	0.8		2.3	1.9		1.2	1.3		0.018	
T2	0.8	1.2		2.4	2.0		1.2	1.2		0.016	
T2–T1	−0.2	0.5		−0.1	0.4		0.0	0.6		0.926	
CSAa (mm^2)											
T1	154.1	29.3		134.3	28.9		141.8	36.6		NS	
T2	191.2	39.9		165.6	34.2		173.5	34.5		NS	
T2–T1	37.0	37.6	*	31.3	25.7	*	31.7	25.7	*	NS	
CSAp (mm^2)											
T1	218.1	43.8		123.0	34.2		194.4	41.0		< 0.001	12, 23
T2	264.8	48.5		183.3	55.5		222.0	54.1		< 0.001	12, 23
T2–T1	46.7	51.4	*	60.3	49.5	*	27.6	36.9	*	NS	
Nasal area (mm2)											
T1	743.9	78.0		707.6	58.2		728.4	116.6		NS	
T2	859.7	100.9		838.2	98.1		827.1	116.9		NS	
T2–T1	115.8	46.7	*	130.6	58.0	*	98.7	45.5	*	NS	
NMR (%)											
T1	29.3	5.1		17.4	4.5		26.9	4.8		0.001	12, 23
T2	30.8	4.1		22.0	6.5		26.8	5.2		0.001	12, 23
T2–T1	1.5	6.1	NS	4.6	6.0	*	0.0	4.7	NS	0.035	23
Nasal airway pressure (Pa)											
T1	214.6	338.2		564.5	494.2	(n = 15)	301.2	397.1	(n = 17)	0.019	12
T2	35.9	36.0		179.5	161.0	(n = 16)	197.1	238.2	(n = 19)	< 0.001	12, 13
T2–T1	178.7	346.6	*	400.2	492.6	(n = 15) *	82.5	366.6	(n = 17) NS	NS	
Nasal airway velocity (m/sec)											
T1	17.7	13.3		34.8	18.7	(n = 15)	27.5	18.2	(n = 17)	0.014	12
T2	7.4	4.7		17.6	11.3	(n = 16)	20.6	20.0	(n = 19)	<0.001	13
T2–T1	10.4	14.8	*	18.1	18.3	(n = 15) *	6.3	21.6	(n = 17) NS	NS	

T1, before RME; T2, after RME; T2-T1, treatment-associated variation; CSAa, cross sectional area at ANS; ANS, anterior nasal spine; CSAp, cross sectional area at maxillary first molar; NMR, (CSAp/nasal area)*100, 1, control group vs nasal mucosa hypertrophy group; 13, control group vs adenoiid group; 23, nasal mucosa hypertrophy group vs adenoiid group; NMR, nasal mucosa rate: nasal cross sectional area/nasal area; *, statistically significant Between T1 and T2 at $P < 0.05$

Table 2

Table II subject distributions and incidences on nasal airway ventilation condition

Before RME	After RME		Control group (n = 20)	Nasal mucosa hypertrophy group (n = 20)	Adenoid group (n = 20)	Fisher exact test P
		Before RME (year)	9.4 ±1.4	9.1 ±1.0	8.9 ±0.8	
		After RME age (year)	11.0±1.5	11.1±1.3	10.7±0.9	
Non obstruction	Non obstruction (case)		10	1	6	
	Obstruction (case)		0	0	1	
Obstruction	Non obstruction (improve) (case)		9	6	3 (1*)	
	Obstruction (non improve) (case)		1	13 (5*)	10 (2*)	
	nasal obstruction Improvement incidence (%)		90.0 (9/10)	31.6 (6/19)	23.1 (3/13)	0.004
Before obstruction incidence (%)			50.0 (10/20)	95.0 (19/20)	65.0 (13/20)	0.020

Obstruction, defined 3D obstruction or maximum pressure of more than 100 Pa; Non obstruction, defined maximum pressure of less than 100 Pa; Before obstruction incidence, (before obstruction case/20 case)*100; nasal obstruction improvement incidence, (improvement case/before obstruction case)*100; *; 3D obstruction case

Table 3

Table III. Spearman rank correlation coefficients and P values between nasal airway cross sectional area and nasal airway ventilation condition

			CSAa			CSAp			NMR		
			T1	T2	T2−T1	T1	T2	T2−T1	T1	T2	T2−T1
T1 (9.1±1.1 years)	Pressure	r_s	−0.141			−0.592**			−0.572**		
	Velocity	r_s	−0.198			−0.555**			−0.534**		
T1 (10.9±1.2 years)	Pressure	r_s		−0.088			−0.523**			−0.547**	
	Velocity	r_s		−0.038			−0.445**			−0.492**	
T2−T1 (1.8±10 years)	Pressure	r_s			−0.006			0.527**			0.513**
	Velocity	r_s			0.098			0.387**			0.391**

CSAa, cross sectional area at ANS, CSAp, cross sectional area at maxillary molar; NMR,(CSAp/nasal area)*100; T1. before maxillary expansion; T2, after maxillary expansion, ** $P < 0.01$

Table 4

Table IV Distribution of three groups of nasal septum deviation and maxillary sinus mucosa hypertrophy

		Control group (n= 20)	Nasal mucosa hypertrophy group (n= 20)	Adenoid group (n= 20)	Fisher exact test P
Before RME	No nasal septum deviation (case)	16	5	13	0.001
	Nasal septum deviiation (case)	4	15	7	
After RME	No nasal septum deviation (case)	16	6	13	0.004
	Nasal septum deviiation (case)	4	14	7	
Before RME	No maxillary sinus mucosa hypertrophy (case)	20	6	17	< 0.001
	Maxillary sinus mucosa hypertrophy (case)	0	14	3	
After RME	No maxillary sinus mucosa hypertrophy (case)	20	11	18	< 0.001
	Maxillary sinus mucosa hypertrophy (case)	0	9	2	

Table 5

Table V Distribution of ventilation obstruction of nasal airway accrding to nasal septum deviation and maxillary sinus mucosa hypertrophy

		Non nasal airway obstruction	Nasal airway obstruction	Fisher exact test P
Before RME	No nasal septum deviaton (case	12	22	0.712
	Nasal septum deviaton (case)	8	18	
After RME	No nasal septum deviaton (case	23	12	0.286
	Nasal septum deviaton (case)	13	12	
Before RME	No maxillary sinus mucosa hype	17	26	0.093
	Maxillary sinus mucosa hypertro	3	14	
After RME	No maxillary sinus mucosa hype	30	19	0.684
	Maxillary sinus mucosa hypertro	6	5	

Table VI Distribution of improvement effect of nasal airway obstruction by RME accrding to nasal septum deviation and maxillary sinus mucosa hypertrophy

		Improve	Not improve	Fisher exact test P
Before RME	No nasal septum deviation (case)	13	12	0.891
	Nasal septum deviiation (case)	11	11	
After RME	No nasal septum deviation (case)	14	12	0.671
	Nasal septum deviiation (case)	10	11	
Before RME	No mxillary sinus mucosa hypertrophy (case)	16	15	0.917
	Maxillary sinus mucosa hypertrophy (case)	8	8	
After RME	No mxillary sinus mucosa hypertrophy (case)	18	18	0.792
	Maxillary sinus mucosa hypertrophy (case)	6	5	

Improve; Before RME was nasal airway obstruction and after RME was no nasal airway obstruction, Not improve; Before and after RME were nasal airway obstruction.