Original Research

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

Key words: Rapid maxillary expansion, computer fluid dynamics, cone-beam computed tomography, nasal airway obstruction, nasal mucosa hypertrophy, obstructive 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 obstructive adenoids remains uncertain. In addition, nasal septum deviation and maxillary sinus mucosal hypertrophy may also affect it. 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 obstructive 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, and CBCT images were taken at time T1 (baseline phase I data obtained before RME insertion) and T2 (obtained after the removal of RME and before phase II treatment), 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, Table 1): 1) control group (20 subjects; six boys, mean T1 and T2 ages were 9.4 and 11.0 years, respectively, and the interval between T1 and T2 was 1.6 years); 2) mucosa group (20 subjects; eight boys, mean T1 and T2 ages were 9.1 and 11.1 years, respectively, and the interval between T1 and T2 was 2.0 years); and 3) adenoids group (20 subjects; seven boys, mean T1 and T2 ages were 9.0 and 10.7 years, respectively, and the interval between T1 and T2 was 1.8 years). The three groups were approximately matched in terms of sex, age, and dentition.

1) Control subjects were those without nasal mucosa hypertrophy or obstructive adenoids (Figure 1A). Nasal mucosa 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 obstructive adenoids were classified as nasal mucosa subjects (Figure 1B). Nasal mucosa 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, obstructive 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 (posterior cross-sectional area of the nasal airway/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 NMR 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]

After one week, the same evaluator (R. SI.) repeated all the measurements to confirm the reliability. Furthermore, after repeating the measurements, another author (T. I) along with the initial evaluator (R. SI.), confirmed the accuracy of these measurements. If an additional measurement was needed, the same evaluator (R. SI.) measured it again; then, 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, 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, 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. (Table 1).

The CSAa (anterior cross-sectional area) 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 (posterior cross-sectional area) 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 (nasal mucosal ratio; the posterior cross-sectional area of nasal airway/nasal area) did not significantly change in the control or adenoids groups following RME (NMR control group: approximately 30%; adenoid group: approximately 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.

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 I). 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 was also a significant negative correlation between pressure and CSAa and CSAp when all the stages were considered rather than by stage (Figure 3).

Maxillary sinus mucosa hypertrophy was observed before RME in 14 out of 20 patients (70%) 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. There was no significant difference between nasal airway obstruction and maxillary sinus mucosa hypertrophy before and after RME (Table IV).

Nasal septum deviation in the mucosa group was significantly larger than in the control and

adenoid groups at T1 and T2 (Table I). Nasal septum deviation and maxillary sinus mucosa hypertrophy significantly differed among the three groups (Table IV). However, the distribution of patients with nasal obstruction was not significantly different from 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).

Discussion

The present study showed that nasal airway ventilation conditions were affected by the specific clinical condition (nasal mucosa hypertrophy or obstructive 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 obstructive adenoids) of the nasal airway. In the absence of nasal mucosa hypertrophy and obstructive 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 obstructive 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 (anterior cross-sectional area) 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 (posterior cross-sectional

243 area). The anterior nasal airway, located in the proximal portion of the nasal cavity, is covered by
244 epithelium and has no erectile tissue, whereas the posterior nasal airway is covered by mucosa. The
245 posterior nasal airway is considered susceptible to nasal mucosa hypertrophy and obstructive
246 adenoids. In the presence of nasal airway obstruction, these results suggest that the posterior nasal
247 airway influences nasal airway ventilation conditions (Figure 3).[28]

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

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

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

262 From these reports[16,27-29] and the results of the present study, we concluded that the threshold
263 of nasal airway obstruction (more than 100 Pa, corresponding to 0.5 Pa/mL/s) [30] of CSAp in children
264 was approximately 250 mm^2 (Figure 3B).

265

266 Treatment change and nasal mucosa rate

NMR (nasal mucosal ratio; the posterior cross-sectional area of nasal airway/nasal area) was used to evaluate nasal mucosa hypertrophy in each group (Figure 4). The NMR of the control 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. On the other hand, 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, despite the improvement, the NMR remained at 22.0%. Otolaryngological treatment is considered necessary for further improvement. In addition, improved upper airway obstruction using oral myofunctional therapy (MFT) has recently been reported.[31] Therefore, in addition to otolaryngological treatment, MFT may also be effective.

NMR of the adenoid group was maintained at approximately 27% after RME, with no significant improvement. CFD also showed no significant improvement. However, the nasal airway CSA was significantly enlarged for both CSAa and CSAp. Based on the above, we infer that RME did not improve nasal ventilation.[32-34] In terms of adenoids and nasal airway relationships, obstructive adenoids have been linked to nasal mucosa hyperplasia in a previous study, and adenoidectomy has been linked to improved nasal airway ventilation.[35] In the case of conventional examinations[36,37] for the degree of nasal airway ventilation, nasal airway ventilation may be affected by obstructive 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 obstructive adenoids as it was derived from the CFD evaluation of a 3D nasal airway model except for the obstructive adenoids. Improvement of nasal airway obstruction was not observed following RME in the presence of obstructive adenoids; this may be because nasal

291 breathing occurred in a non-physiological situation.[32] It has been shown that nasal breathing with
292 an obstructive adenoid causes the nasopharynx to contract, resulting in fast airflow through the
293 nasopharyngeal airway (Figure 5). In other words, it is thought that the nasal mucosa may be
294 thickened by mechanical stimulation [12] of the nasal mucosa. Based on the results of the present study
295 and the above, when obstructive adenoids are detected, it is difficult to achieve an improvement in
296 nasal airway ventilation conditions following RME; hence otolaryngology treatment for the adenoids
297 is considered necessary.[32]
298
299 Differences in the improvement of nasal airway obstruction following RME
300 The sizes of the nasal airways before the expansion and improvement in nasal airway
301 obstruction following RME were different (Figure 6); thus, the improvement rates of each group in
302 this study were different.
303 Regarding the nasal airway CSA of the control group (before 11.1 years and after 13.4
304 years), in our previous study,[16] the anterior and posterior regions of the nasal airway increased from
305 186 to 198 mm^2 and from 259 to 284 mm^2, respectively. These showed that normal growth changes
306 of both the anterior and posterior regions of CSA were 12 mm^2 and 25 mm^2, respectively. However,
307 because the age of the patients in the present study did not differ significantly from this previous
308 study,[16] the normal growth amount of CSA in the present study patients was considered similar to
309 previous research results.[16] Therefore, the increment in the present study anterior and posterior nasal
310 airway CSAs after RME included both normal growth and treatment change, respectively.
311 Furthermore, the treatment change of anterior and posterior CSAs was subtracted by the normal
312 growth values. Both CSAs were expected to be 25.0 and 21.7 mm^2 in the control group, 19.3 and
313 35.3 mm^2 in the mucosa group, and 19.7 and 2.6 mm2 in the adenoid group, respectively. Therefore,
314 an enlargement effect of approximately 20 mm2 was observed in the anterior region of the nasal

315 airway in all three groups after RME; the enlargement effect was approximately 20–35 mm^2 in the
316 posterior region in the control and mucosa groups, but there was no enlargement in the adenoid group.
317 Thus, the posterior CSA reflected the ventilation condition of the nasal airway.
318 The CSAp of the control group was relatively large, and the nasal airways were expanded
319 smoothly; as a result, the CSAp was of a sufficient size for nasal airway obstruction to improve.
320 Therefore, the improvement rate increased. There was one case of no improvement in the control
321 group. Future studies should investigate such cases in detail.
322 The nasal mucosa group had small CSAp values before the expansion. Therefore,
323 although the CSAp of the mucosa group was markedly increased following RME, the CSAp did not
324 reach the size necessary to improve nasal airway ventilation, explaining the low improvement rate.
325 The CSAp of the adenoids group was intermediate in size before expansion. However,
326 the effect of RME on improvement was insufficient. Therefore, expansion of the nasal airway did
327 not occur until symptom improvement was achieved, and this group was associated with a low
328 improvement rate.
329
330 Influence of improvement effect of nasal airway obstruction by RME in deviated nasal
331 septum and maxillary sinus hypertrophy
332 The results of this study showed that the frequency of nasal septal curvature and maxillary
333 sinus hypertrophy was significantly higher in the mucosa group. However, when looking at all
334 subjects, there was no difference in the distribution of nasal septal curvature and maxillary sinus
335 mucosal hypertrophy in the improvement effect of nasal airway obstruction by RME and nasal
336 airway obstruction. Therefore, in this study, we conclude that nasal septum deviation and maxillary
337 sinus hypertrophy are unlikely to significantly affect the improvement effect of nasal airway
338 obstruction by RME, which is the goal of the current study. However, these studies only included

children requiring RME and need further investigation.

Clinical implications

This study showed following RME, effective improvement of the nasal airway obstruction occurred in the absence of hyperplasia of the nasal mucosa and obstructive 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 obstructive 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 obstructive adenoids) of the nasal airway. Improvement in nasal airway obstruction following RME was influenced by the clinical condition (nasal mucosa hypertrophy and obstructive adenoids) of the nasal airway, too. In cases without nasal mucosa hypertrophy or obstructive 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 obstructive adenoids (31.6% and 23.1%, respectively). Nasal airway obstruction due to obstructive adenoids did not respond to RME.

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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.

390 4. DiCosimo C, Alsulaiman AA, Shah C, Motro M, Will LA, Parsi GK. Analysis of nasal airway
391 symmetry and upper airway changes after rapid maxillary expansion. Am J Orthod Dentofacial
392 Orthop 2021;160:695-704.
393 5. Oliveira De Felippe NL, Da Silveira AC, Viana G, Kusnoto B, Smith B, Evans CA. Relationship
394 between rapid maxillary expansion and nasal cavity size and airway resistance: short- and long-
395 term effects. Am J Orthod Dentofacial Orthop 2008;134:370-382.
396 6. Araújo MC, Bocato JR, Oltramari PV, de Almeida MR, Conti AC, Fernandes TM. Tomographic
397 evaluation of dentoskeletal effects of rapid maxillary expansion using Haas and Hyrax palatal
398 expanders in children: A randomized clinical trial. J Clin Exp Dent 2020;12:e922-e930.
399 7. Weissheimer A, de Menezes LM, Mezomo M, Dias DM, de Lima EM, Rizzatto SM. Immediate
400 effects of rapid maxillary expansion with Haas-type and hyrax-type expanders: a randomized
401 clinical trial. Am J Orthod Dentofacial Orthop 2011;140:366-376.
402 8. Lemos Rinaldi MR, Azeredo F, Martinelli de Lima E, Deon Rizzatto SM, Sameshima G, Macedo
403 de Menezes L. Cone-beam computed tomography evaluation of bone plate and root length after
404 maxillary expansion using tooth-borne and tooth-tissue-borne banded expanders. Am J Orthod
405 Dentofacial Orthop 2018;154:504-516.
406 9. Leboulanger N. Nasal obstruction in children. Eur Ann Otorhinolaryngol Head Neck Dis
407 2016;133:183-186.
408 10. Baddam P, Thereza-Bussolaro C, Flores-Mir C, Graf D. Nasal cavity structural anomalies in
409 children and adolescents at high risk of sleep-disordered breathing: An exploratory cone-beam
410 computed tomography study. Am J Orthod Dentofacial Orthop 2021;160:533-543.e532.
411 11. Iwasaki T, Sugiyama T, Yanagisawa-Minami A, Oku Y, Yokura A, Yamasaki Y. Effect of
412 adenoids and tonsil tissue on pediatric obstructive sleep apnea severity determined by
413 computational fluid dynamics. J Clin Sleep Med 2020;16:2021-2028.
414 12. Iwasaki T, Papageorgiou SN, Yamasaki Y, Ali Darendeliler M, Papadopoulou AK. Nasal
415 ventilation and rapid maxillary expansion (RME): a randomized trial. Eur J Orthod 2021;43:283-
416 292.
417 13. Iwasaki T, Suga H, Minami-Yanagisawa A, Hashiguchi-Sato M, Sato H, Yamamoto Y et al.
418 Upper airway in children with unilateral cleft lip and palate evaluated with computational fluid
419 dynamics. Am J Orthod Dentofacial Orthop 2019;156:257-265.
420 14. Baysal A, Karadede I, Hekimoglu S, Ucar F, Ozer T, Veli I et al. Evaluation of root resorption
421 following rapid maxillary expansion using cone-beam computed tomography. Angle Orthod
422 2012;82:488-494.
423 15. Sperl A, Gaalaas L, Beyer J, Grünheid T. Buccal alveolar bone changes following rapid
424 maxillary expansion and fixed appliance therapy. Angle Orthod 2021;91:171-177.
425 16. Iwasaki T, Yanagisawa-Minami A, Suga H, Shirazawa Y, Tsujii T, Yamamoto Y et al. Rapid

426 maxillary expansion effects of nasal airway in children with cleft lip and palate using computational
427 fluid dynamics. Orthod Craniofac Res 2019;22:201-207.
428 17. Smith T, Ghoneima A, Stewart K, Liu S, Eckert G, Halum S et al. Three-dimensional computed
429 tomography analysis of airway volume changes after rapid maxillary expansion. Am J Orthod
430 Dentofacial Orthop 2012;141:618-626.
431 18. Capelli M, Gatti P. Radiological Study of Maxillary Sinus using CBCT: Relationship between
432 Mucosal Thickening and Common Anatomic Variants in Chronic Rhinosinusitis. J Clin Diagn Res
433 2016;10:Mc07-mc10.
434 19. De Backer JW, Vanderveken OM, Vos WG, Devolder A, Verhulst SL, Verbraecken JA et al.
435 Functional imaging using computational fluid dynamics to predict treatment success of
436 mandibular advancement devices in sleep-disordered breathing. J Biomech 2007;40:3708-3714.
437 20. Gamiño B, Aguillón J. Numerical simulation of syngas combustion with a multi-spark ignition
438 system in a diesel engine adapted to work at the Otto cycle. Fuel 2010;89:581-591.
439 21. Iwasaki T, Sato H, Suga H, Takemoto Y, Inada E, Saitoh I et al. Relationships among nasal
440 resistance, adenoids, tonsils, and tongue posture and maxillofacial form in Class II and Class III
441 children. Am J Orthod Dentofacial Orthop 2017;151:929-940.
442 22. Yanagisawa-Minami A, Sugiyama T, Iwasaki T, Yamasaki Y. Primary site identification in
443 children with obstructive sleep apnea by computational fluid dynamics analysis of the upper airway.
444 J Clin Sleep Med 2020;16:431-439.
445 23. Kobayashi R, Miyazaki S, Karaki M, Kobayashi E, Karaki R, Akiyama K et al. Measurement of
446 nasal resistance by rhinomanometry in 892 Japanese elementary school children. Auris Nasus
447 Larynx 2011;38:73-76.
448 24. Dahlberg G. Stastistical methods for medical and biological syudents. London: Gerge Allen
449 and Unwin; 1940.
450 25. Yajima Y, Oshima M, Iwai T, Kitajima H, Omura S, Tohnai I. Computational fluid dynamics
451 study of the pharyngeal airway space before and after mandibular setback surgery in patients with
452 mandibular prognathism. Int J Oral Maxillofac Surg 2017;46:839-844.
453 26. Shirazawa Y, Iwasaki T, Ooi K, Kobayashi Y, Yanagisawa-Minami A, Oku Y et al. Relationship
454 between pharyngeal airway depth and ventilation condition in mandibular setback surgery: A
455 computational fluid dynamics study. Orthod Craniofac Res 2020;23:313-322.
456 27. Garcia GJM, Hariri BM, Patel RG, Rhee JS. The relationship between nasal resistance to
457 airflow and the airspace minimal cross-sectional area. J Biomech 2016;49:1670-1678.
458 28. Wandalsen GF, Mendes AI, Solé D. Correlation between nasal resistance and different acoustic
459 rhinometry parameters in children and adolescents with and without allergic rhinitis. Braz J
460 Otorhinolaryngol 2012;78:81-86.
461 29. Van Holsbeke C, Vos W, Van Hoorenbeeck K, Boudewyns A, Salgado R, Verdonck PR et al.

462 Functional respiratory imaging as a tool to assess upper airway patency in children with obstructive
463 sleep apnea. Sleep Med 2013;14:433-439.
464 30. Crouse U, Laine-Alava MT, Warren DW. Nasal impairment in prepubertal children. Am J
465 Orthod Dentofacial Orthop 2000;118:69-74.
466 31. Huang YS, Hsu SC, Guilleminault C, Chuang LC. Myofunctional Therapy: Role in Pediatric
467 OSA. Sleep Med Clin 2019;14:135-142.
468 32. Brinckmann CA, Scapin FP, Bernd LA, Lubianca Neto JF. Effect of improved nasal airflow
469 after adenoidectomy on nasal erectile tissue. Arch Otolaryngol Head Neck Surg 2008;134:832-836.
470 33. Shin SH, Heo WW. Effects of unilateral naris closure on the nasal and maxillary sinus mucosa
471 in rabbit. Auris Nasus Larynx 2005;32:139-143.
472 34. Maurizi M, Paludetti G, Ottaviani F, Almadori G, Falcetti S. Mucociliary function and nasal
473 resistance evaluation before and after adenoidectomy. Int J Pediatr Otorhinolaryngol
474 1986;11:295-300.
475 35. Calvo-Henriquez C, Branco AM, Lechien JR, Sandoval-Pacheco V, Maldonado-Alvarado B,
476 Maza-Solano J et al. Assessing the effect of adenoidectomy on nasal resistance and airflow. A
477 systematic review and meta-analysis. Int J Pediatr Otorhinolaryngol 2021;151:110969.
478 36. Clarke RW, Jones AS. The limitations of peak nasal flow measurement. Clin Otolaryngol Allied
479 Sci 1994;19:502-504.
480 37. Cakmak O, Coskun M, Celik H, Buyuklu F, Ozluoglu LN. Value of acoustic rhinometry for
481 measuring nasal valve area. Laryngoscope 2003;113:295-302.
482 38. Calvo-Henriquez C, Capasso R, Chiesa-Estomba C, Liu SY, Martins-Neves S, Castedo E et al.
483 The role of pediatric maxillary expansion on nasal breathing. A systematic review and metanalysis.
484 Int J Pediatr Otorhinolaryngol 2020;135:110139.

485

486

487 **Figure captions**

488 Figure 1. Condition of the nasal airway.

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

493

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.

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.

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

519 A: Adenoid hypertrophy.

520 B: Model with adenoids. Airflow showed a faster posterior part (red arrow). Due to the fast airflow,

521 the site provides strong mechanical stimulation to the nasal mucosa.

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

523

524 Figure 6. The difference in improvements in the nasal airway ventilation condition following RME.

525 A representative example is displayed (upper, before expansion; lower, after expansion).

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

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

528 RME, the Control subjects had a pressure of 100 Pa or less and showed improvement in the nasal

529 airway obstruction. However, the nasal mucosa hypertrophy and adenoid subjects did not show

530 improvements in the nasal airway obstruction, with the pressure remaining 100 Pa or more.

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 (years)											
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	
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, anterior cross sectional area of nasal airway at ANS; ANS, anterior nasal spine; CSAp, posterior cross sectional area of nasal airway at maxillary first molar; NMR, nasal mucosal rate: (CSAp/nasal area)*100, 12, control group vs nasal mucosa hypertrophy group; 13, control group vs adenoiid group; 23, nasal mucosa hypertrophy group vs adenoiid group; *, statistically significant Between T1 and T2 at $P < 0.05$

Table II Subject distributions and incidence by 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 as 3D obstruction or maximum pressure of more than 100 Pa; Non obstruction, defined as 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 III. Spearman rank correlation coefficients and P values of relationship 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, anterior cross sectional area of nasal airway at ANS, CSAp, posterior cross sectional area of nasal airway at maxillary molar; NMR, nasal mucosal ratio (CSAp/nasal area)*100; T1. before maxillary expansion; T2, after maxillary expansion, ** $P < 0.01$

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 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 hypertr	17	26	0.093
	Maxillary sinus mucosa hypertroph	3	14	
After RME	No maxillary sinus mucosa hypertr	30	19	0.684
	Maxillary sinus mucosa hypertroph	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.

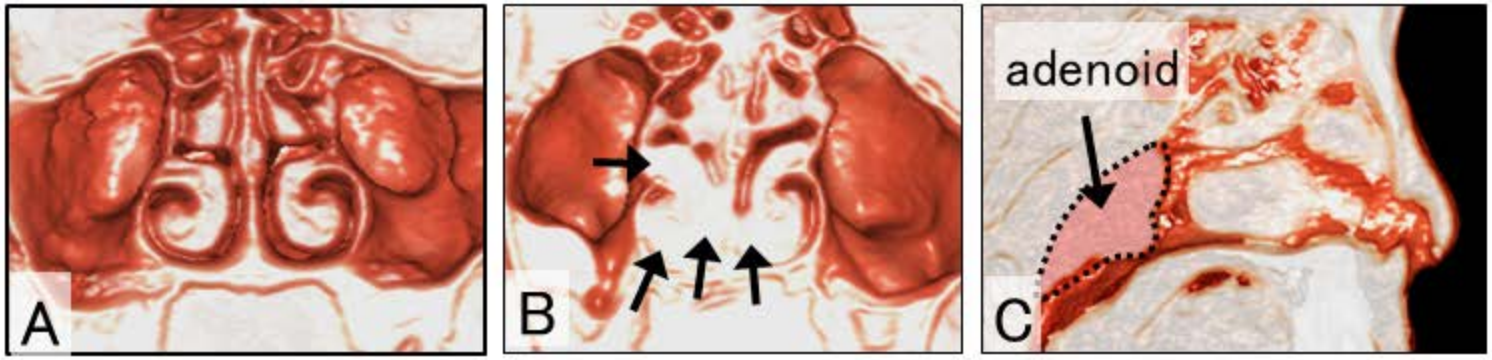

Fig 1

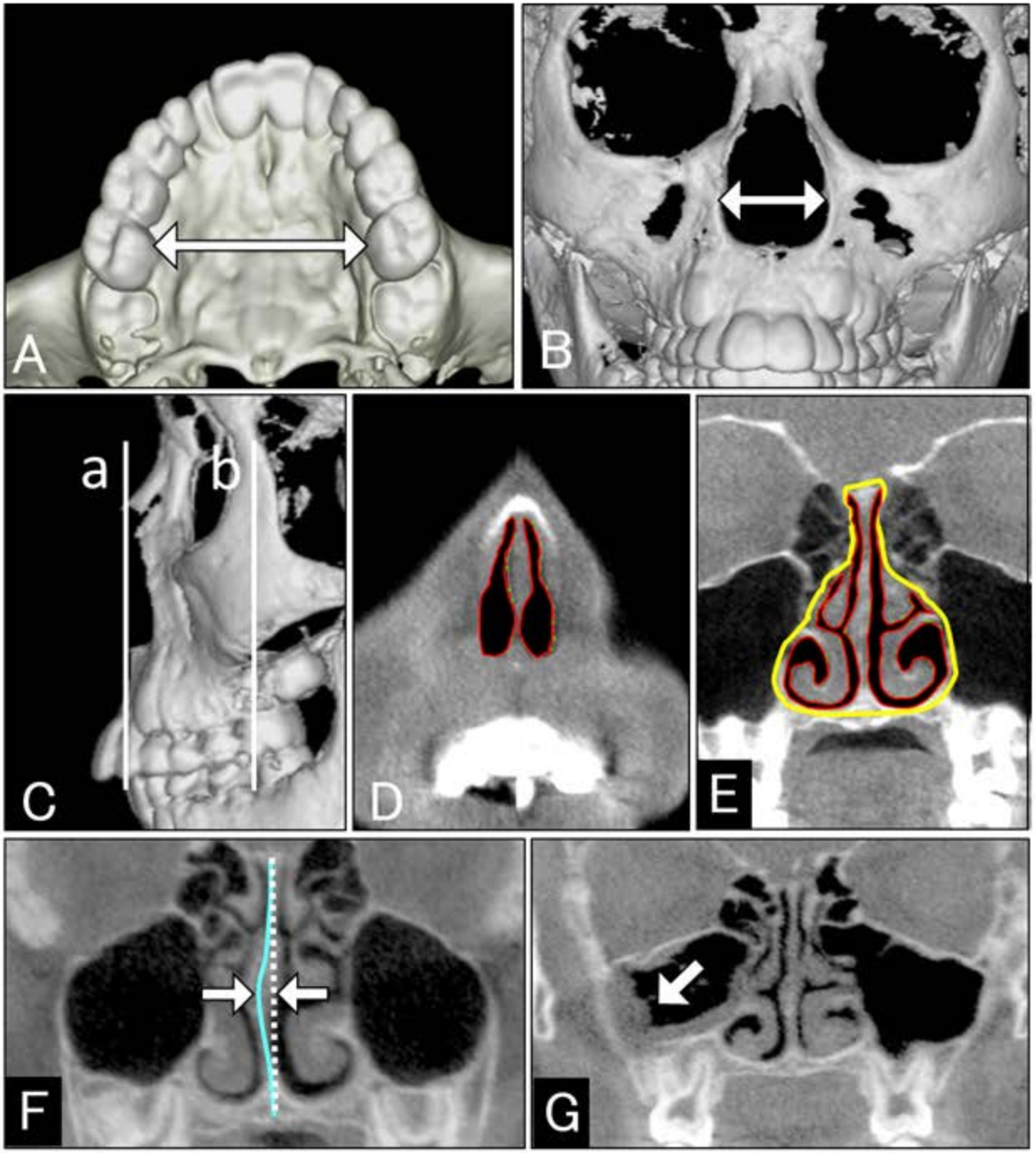

Fig 2

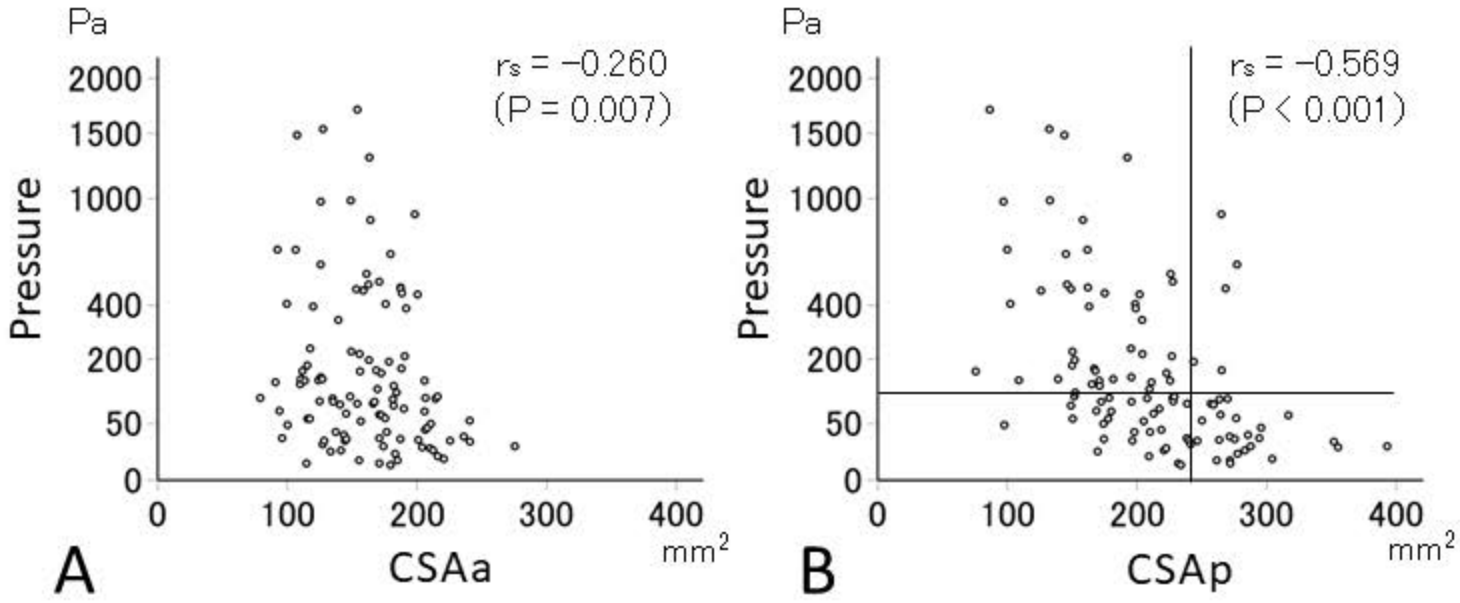
Pa
2000
1500
1000
400
200
50
0
Pressure
rs = −0.260
(P = 0.007)
0
100
200
300
400
mm2
CSAa
A
Pa
2000
1500
1000
400
200
50
0
Pressure
rs = −0.569
(P < 0.001)
0
100
200
300
400
mm2
CSAp
B

Fig 3

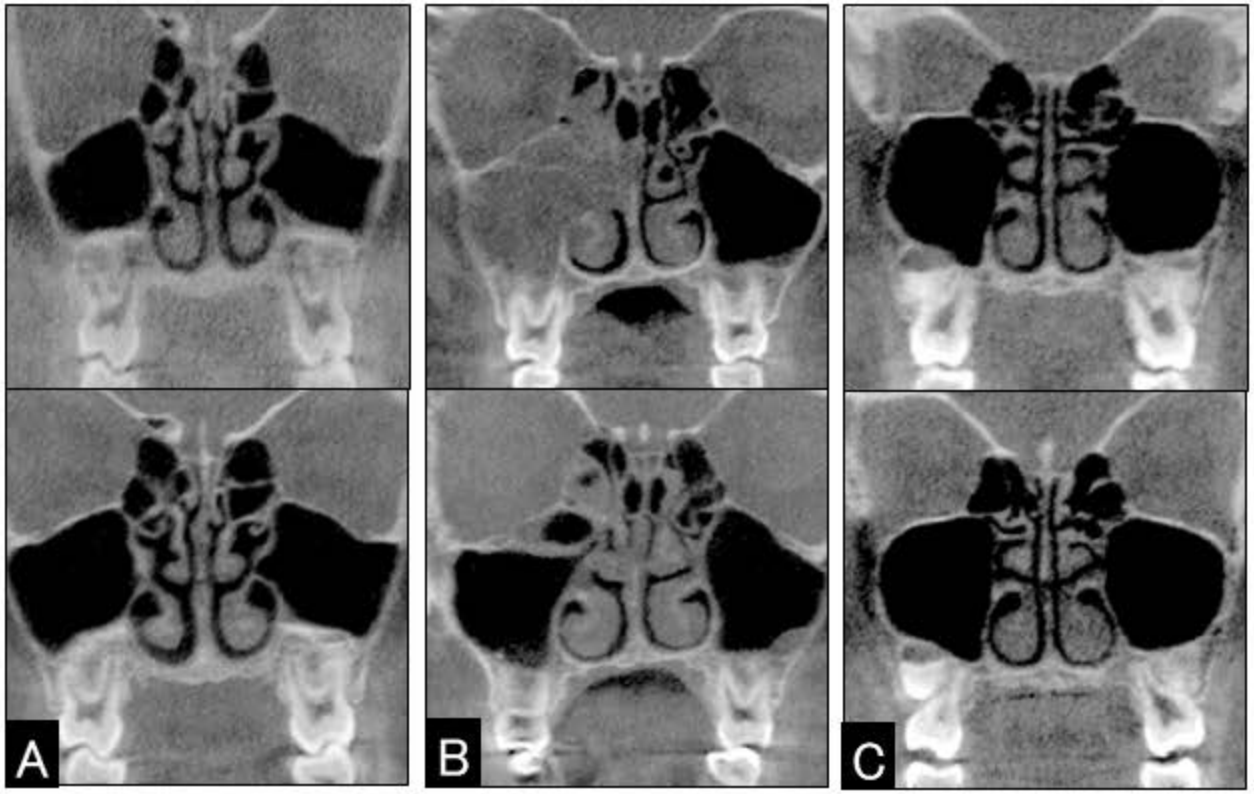

Fig 4

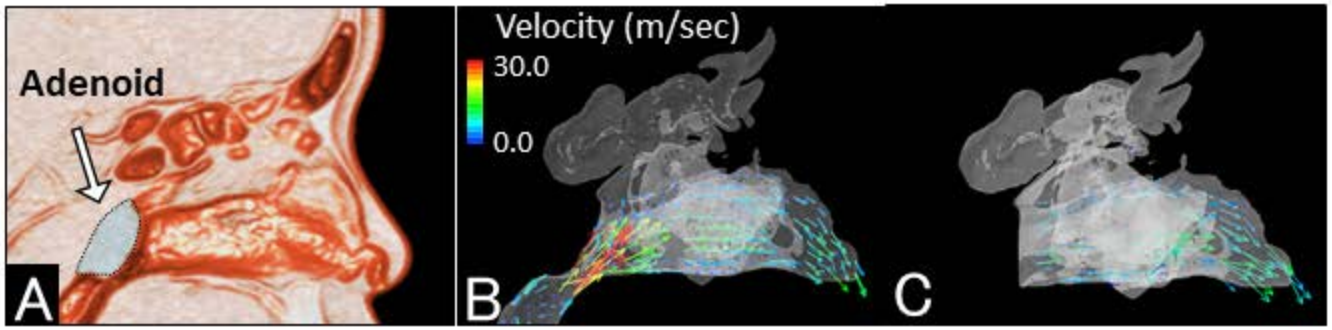

Fig 5

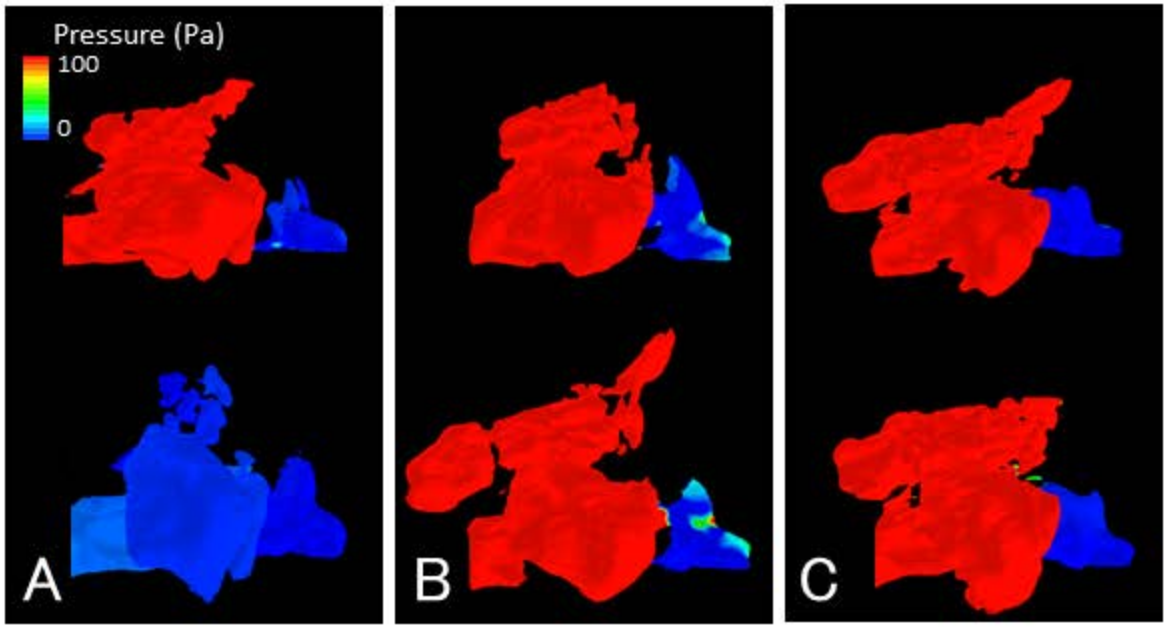

Fig 6