Guest Editorial

Maxillary expansion and breathing function: Where we are now?

Since originally proposed by Angell[1] in 1860, the rapid maxillary expansion (RME) treatment has become a popular treatment to correct skeletal transverse maxillary discrepancy. However, the efficiency of such procedures was demonstrated only 100 years later[2,3] following the advent of proper radiographical recordings.

According to the anatomical proximity between nasal cavity and hard palate, an orthopedic expansion of the former might occur as consequence of the RME treatment. This hypothesis has initially been investigated decades ago. In particular, earlier studies[4,5] evaluated the advantages of RME treatment in improving nasal airflow in patients with nasal stenosis. It was later suggested that RME treatment triggers effects on nasal width[6-8] and volume.[9-14] Indeed, some studies[15,16] showed a reduction in nasal airway resistance after RME treatment. Consistently, a more recent investigation[17] reported up to 45% increase in nasal cross-sectional areas after expansion. In spite of this evidence, considering the V-shaped opening pattern of the midpalatal suture,[4,5] the only purpose of increasing respiratory performance has been reported as not sufficient to indicate an RME treatment.[17]

More in detail, airway changes upon RME treatment have been studied using different methodologies including acoustic rhinometry,[18] two-dimensional,[4,5] and three-dimensional (3D)[19] cephalometrics. One of the most used morphological techniques nowadays is represented by the 3D cone-beam computed tomography (CBCT) that allows a full 3D and reliable quantification of anatomical changes even for the airway compartments. Other functional diagnostic tools that can be employed to investigate the effects of RME on airflow include the polysomnography examination. This recording widely employed in obstructive sleep apnea patients,[20] gives useful information about breathing pattern, and showing quantitative data such as oxygen saturation (SpO_2) and apnea/hypopnea index (AHI). Indeed, a morphological modification of the airway spaces does not necessarily implies a greater respiratory performance (i.e., function) or *vice versa*, and studies including only the anatomical investigations of the RME treatment on airway compartments volume might be limited in their conclusions.

Several previous studies[9-14] evaluated airway volume changes after RME treatment dividing airway in different compartments to better describe effects at different levels. Indeed, an important distinction should be performed between anatomical skeletal changes and airway changes. The former modifications, in fact, might be of different amounts according to the amount of expansion that is related to maxillary transverse discrepancy and are influenced by the resistance of the sutures around maxillary bones.[21] According to a recent study using CBCT,[22] RME produces significant skeletal transverse augmentations in the palatal and nasal regions. These increments are bigger in the lower portion of the nasal cavities. Moreover, RME is able to increase significantly skeletal nasal cavity volume. The volume increase is equally distributed between the anterior and the posterior part of the nasal cavity. Greater increases in width were observed in the nasal floor region rather than in the middle nasal width region, thus supporting the reverse "V-" shape opening model of the craniofacial complex.[5]

On the contrary, airway changes are related to more complex variables and indeed to the breathing pattern of the patient. Unfortunately, skeletal widening of the nasal cavity does not necessarily imply a proportional improvement of the airway since airway obstruction causes might be not related to skeletal anatomical reasons. The airway might be divided in upper (from the nostrils to posterior nasal spine), middle (from posterior nasal spine to the basis of the tongue), and lower compartment (from the basis of the tongue to epiglottis) and in every portion, different mechanisms responsible for improvement or worsening of breathing might take place. In a recent study,[23] airway was examined as upper, middle, and lower compartments and as a whole. According to their results, only nasal cavity had a significant increase in volume after RME treatment. These findings are explained by the close anatomical proximity of the upper airway compartment, i.e., nasal cavity, with the hard palate subjected to orthopedic expansion. Similar results were reported previously.[11-13,24] In particular, Smith *et al*.[13] divided the airway volume in nasal cavity, nasopharynx, and oropharynx showing a significant increase in the nasopharynx volume after RME treatment. The apparent inconsistency between those results and the present evidence may be related to the different separations or combination of the

nasopharynx and oropharynx followed. This multiplicity of results could be related to the presence or absence of adenoid tissue in nasopharynx before treatment. Chang *et al*.[14] reported bony expansion and significant cross-sectional area increase immediately posterior to the hard palate after RME treatment and suggested that effects on the upper airway would be local, and it diminishes farther from the maxillary suture, possibly as a result of soft-tissue adaptation.

Iwasaki *et al*.[25] used CBCT and computational fluid dynamics to estimate the effects of RME on nasal airflow function (pressure and velocity). In the most of the examined patients, the pressure and velocity of nasal ventilation after RME resulted significantly lower than before treatment indicating an improvement in nasal breathing.[25] Fastuca *et al*.[26,27] evaluated changes in airway volumes and respiratory performance in 15 patients with a mean age of 7.5 years undergoing RME to determine whether any correlation exists between the morphological and respiratory functional modifications. On CBCT, the airway regions were segmented, and the volumes were computed to detect variations after the removal of the maxillary expander 12 months later. The multiple logistic regressions showed that the more a participant presented with a reduced nasal volume in the middle and lower compartments, the more he or she would benefit from RME in terms of improved SpO_2. The AHI can be used to indicate the severity of sleep apnea. Evaluating AHI as a secondary outcome, Fastuca *et al*.[27] found an improvement in the index with a reduction in apneic events of 4.2/h. Not only the upper and nasal airways but also the middle and lower airway compartments underwent significant volume increases. Such increases were greater for the nasal cavity and slightly lower for the middle and lower compartments.[27]

The study by Zhao *et al*.[10] is the only one that included an untreated control group and saw no significant changes between treated and controls in airway volumes after RME treatment. Moreover, more complex mechanisms are involved in respiratory function changes after RME. Iwasaki *et al*.[28] recently compared changes of the tongue posture with changes in the nasal airway ventilation pattern after RME treatment. According to their findings, children with nasal airway obstruction have a low tongue posture regardless of RME treatment meanwhile improvement of the nasal airway ventilation condition might be associated with improved low tongue posture after RME.

Even though encouraging results were recently arisen, especially with the means of new 3D technologies, long-term follow-up needs to be investigated.

Matsumoto *et al*.[29] investigated long-term effects of RME on nasal cavity using acoustic rhinometry, computed rhinomanometry, and posteroanterior cephalometric radiography demonstrating an increase in nasal osseous width with less significant increases in nasal area and nasal resistance and suggested that the effects of RME could be more evident at the bony level[18,22] than at the mucosal level and this might be due to compensatory hypertrophy of the nasal mucosa after expansion. A recent review concluded that the stability of the results can be expected for at least 11 months after the orthopedic therapy.[30] Further randomized and blinded controlled studies are needed to strengthen the evidence of the long-term RME effects on airway dimensions and functions.

Rosamaria Fastuca
Department of Medical, Surgical and Health Sciences,
University of Messina, Messina, Italy

Address for correspondence: Dr. Rosamaria Fastuca,
Department of Medical, Surgical and Health Sciences,
University of Messina, Messina, Italy.
E-mail: rosamariaf@hotmail.it

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