Letters

Effect of rapid maxillary expansion on hearing loss

In their article on rapid maxillary expansion, Ceylan and co-authors examine a neglected aspect of orthodontic treatment: namely, its relationship to medicine. (Ceylan I, Oktay H, Demirci M. Effect of rapid maxillary expansion on hearing loss. Angle Orthod 1996;66(4):301-308.) In this case, the authors looked at the relationship of rapid maxillary expansion (RME) to conductive hearing loss (CHL). Their conclusion does not show RME in a particularly good light, and the results are somewhat out of step with much that has already been published.

Weaknesses stem from the protocol of this mutidisciplinary study, e.g., the selection of orthodontic cases with buccal crossbites and some degree of CHL. The patients in the study were treated with RME so that any changes in CHL should rightfully be described as probable side effects. Selection ought to have been made on the basis of CHL in the first place, not malocclusion, and then treatment focused on the reduction of CHL through its causes. This commentator wonders if so much attention would have been paid to the malocclusions and their treatments if the hypothesis had been tested primarily by otolaryngologists.

The article does not give the individual levels of CHL, only the ranges and means, and that is not good enough. The lowest was only 10 dB, although cases under 20 dB are generally considered normal. How many were under 20 dB? Such cases should never have been included in the sample as they distort the results by pulling down the averages. It is foolish enough to treat normal patients and even more foolish if their nonexistent or marginal improvements are included in the statistics.

The most common cause of CHL in children is otitis media (OM), usually in the form of acute or serous "glue ear." The acute form is often due to the spread of upper respiratory tract infection (URTI) associated with poor nasal ventilation and mouth breathing (stuffy nose syndrome). Dilation by RME reduces nasal airway resistance, improves nasal breathing (often changing the primary mode from mouth to nose), and restoring the defense mechanism of filtration (mucocillary transportation) with consequent inflammatory reduction. Serous OM is due to poor drainage of the middle ear because of eustachian tube dysfunction. RME, by increasing the width between the pterygoid hamuli, lengthens the course of the tensor veli palatini, increasing their tension and improving their action in opening the tubes on swallowing. Admittedly, this has not been firmly established.

These anatomic and physiologic points are covered in the discussion, but not applied to the individual subjects in the study. We are left in limbo regarding their otorhinolaryngologic histories. Which, if any, of the cases were mouth breathers, snorers, or had URTI? Were their nasopharyngies clear or were any nasal obstructions attributable to anterior stenoses? In other words, are we sure that the cases treated might reasonably respond to RME, and what, if any, were the respiratory changes?

Turning to the RME itself: "The mean midpalatal suture opening of 6.20±0.60 mm was achieved." This must be a misprint and apply to the dental expansion. To accomplish such a suture opening would require a colossal dental expansion, but the figure would fit a crossbite correction. However, in RME, partial relapse is inevitable and overexpansion is necessary. The relapse is in the dental crowns, not in the basal area. Consequently, any improvements in the airway and ventilation are not lost, as implied in the text. Relapse was mentioned, but it was not clear if overexpansion was used to compensate as "...widening of the screw continued... until the posterior crossbite was eliminated." Expansion of 6.20 mm suggests an absence of overcorrection. If so, the chance of gaining extra basal dilation was lost.

Regarding the appliance that was used, the Biedermann is the least rigid of those habitually employed, allowing more tipping of the teeth and less basal expansion. This is compared with appliances incorporating palatal pads or bonded full-crown capping. A question remains as to what extent the nasal airway was enlarged and the interhamular width increased, so doubt is expressed over the physiologic changes essential to a positive conclusion. There are some simple tests, such as holding a cold mirror before the nose/mouth, and according to Sorensen et al. (1980), snoring is a good indicator of high nasal airway resistance and mouth-breathing, the cessation of which with RME usually marks a change to normal respiration.

This work requires a more comprehensive presentation of data, especially the CHL figures for each case together with any perceived changes in respiratory pattern that might indicate the efficacy of the RME. Not a big task for 14 cases, or fewer if those with under 20 dB are excluded. It is also probable that better therapeutic application could have been made of the RME.

I believe the details mentioned above are responsible for the negative results, not the failure of RME. I have cited only one reference for support. All other issues raised are found within the excellent list provided by the authors, which perhaps should have been more diligently read before embarking on this project.

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References

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Author's response

We would like to express our thanks to Dr. Timms for his close interest in our article.

We based our study on scientific criteria and reported the results objectively. All the subjects included in the study applied to our department of orthodontics with orthodontic complaints, not audiological complaints. The main objective of treatment in these patients was to eliminate the orthodontic problems, and rapid maxillary expansion (RME) formed only the first stage of the treatment. As is well known, otolaryngologists prefer treatment techniques other than RME to treat conductive hearing loss (CHL).

Forty-two values were measured for each patient, 24 relating to hearing levels and 18 to air-bone gap. To report all these values for all the patients would simply be confusing and of no benefit to the readers.

Since the hearing levels were measured in 250, 500, 1000, and 2000 Hz, the number of patients with hearing loss over 20 dB may be said to be eight, even though it was difficult to determine on the basis of distribution. As a matter of fact, one individual might be included in the normal hearing level when measured on one frequency but in the group with hearing loss when measured on another. According to the air-bone gap measurements, hearing loss was severe (55 dB) in one ear, moderate (30 to 35 dB) in four ears, and mild (20 to 25 dB) in twelve ears.

Patients with severe maxillary deficiency were treated based on their orthodontic requirements, not their hearing levels; changes in their hearing levels were observed. I consider Dr. Timms' approach to be illogical not to include in the statistics the nonexistent or marginal improvements. Dr. Timms reported in one of his articles[1] that he treated 200 patients by means of RME, but he gave the audiological records of only one patient. Was it because he couldn't observe any positive improvement in the other patients?

I do not believe that it would be of use to the readers to mention the otolaryngological history of the patients.

All the cases treated by RME responded positively to the treatment. It was determined by means of occlusal X-rays that there was sutural opening in all of the cases. It was clinically observed that nasal resistance either disappeared or decreased.

I do not share of Dr. Timms' thoughts regarding the Bidermann Appliance. As Spolyar[2] stated, "Hyrax-type banded appliances are the rigid appliances most commonly encountered in the literature." In our cases, 0.9 mm stainless steel

wires were soldered on the premolar and molar bands in order to increase the rigidity of the appliances used for RME.

Dr. Timms claims that there would be a relapse in dental crowns, but not in basal areas after RME. On the contrary, Krebs[3-5] placed metallic implants in infrazygomatic ridges and observed a relapse in basal area after the fixed retention. He demonstrated that approximately 30% of the basal expansion was lost after a period of 15 months.

On the other hand, Dr. Mew mentioned in his kind letter to me that his findings agreed with ours. He stated, "The nasal airway improves after expansion but tends to occlude again a few months latter in the same way as the hearing worsens." He also said, "I have noted that those cases which maintain the improvement in hearing and airway tend to be those that learn to keep their mouths closed."

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References

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