Original Article

Rapid Maxillary Expansion and Conductive Hearing Loss

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Abstract: The purpose of this study was to evaluate the effects of rapid maxillary expansion (RME) on conductive hearing loss in 35 subjects (21 girls and 14 boys) with an average age of 14 years 6 months. All patients had maxillary constriction with a high palatal vault and a conductive hearing loss. Pure-tone audiometric records were used to determine the hearing levels at four time intervals, namely, before RME, after sufficient midpalatal suture opening was obtained (mean: 18 days), after the retention period (mean: six months), and a final set two years after the retention period. Records were evaluated by the same otolaryngologist. Analysis of variance was used to assess the changes in the hearing level and the air-bone gap. Descriptive statistics were calculated for each subject at each period. The results indicated that significant changes occurred in both the hearing levels and air-bone gaps in both timing and frequency after the active treatment period ($P < .001$). For most patients (74%), these improvements were maintained two years after active treatment. (*Angle Orthod* 2003;73:669–673.)

Key Words: Maxillary constriction; Hearing level; Air-bone gap

INTRODUCTION

Rapid maxillary expansion (RME) increases the transverse dimension of the upper arch by separating the two maxillary halves and, in addition, the posterior teeth and alveolar processes move buccally.[1] The force for midpalatal splitting is delivered by the activation of the expansion screw. With such an approach, midpalatal suture is separated by the application of heavy intermittent forces (0.9–4.5 kg) for a short period (1–3 weeks).[2–7] The suture's vertical opening is triangular, with the greatest width at the prosthion and the least near the apex of the nasal cavity.[7] As the maxilla starts to separate, the translation of the maxillary segments occurs.[8] The lateral walls of the nasal cavity with the attached conchae move laterally, and the floor of the nose drops inferiorly as the alveolar processes bend laterally and the free margins of the horizontal palatine processes move inferiorly. The mechanical widening of the nose is said to facilitate nasal respiration.[7] Several investigators[6,9–15] have evaluated the effects of RME and reported a decrease in nasal resistance and an increase in nasal width after treatment. According to Wertz,[14] the stenosis caused by an obstruction in the more anterior-inferior portion of the nose could possibly be relieved by maxillary suture opening, whereas a stenosis located in a more posterior or superior area would not benefit from this procedure. Recently, Basciftci et al[16] also have reported that RME is effective in patients with respiratory problems.

RME is frequently used in the treatment of maxillary constriction with a bilateral posterior crossbite. Maxillary constriction, together with a high palatal vault, are two characteristics of the "skeletal development syndrome."[17] Laptook[17] described other features of this syndrome as (1) decreased nasal permeability resulting from nasal stenosis, (2) elevation of the nasal floor, (3) mouth breathing, (4) bilateral dental maxillary crossbite along with a high palatal vault, and (5) enlargement of the nasal turbinates causing a decrease in nasal airway size.

Conductive hearing loss is one of the auditory disorders characterized by elevated air-conduction thresholds. The loss in hearing varies according to the severity and type of the physical change imposed on the mechanical system of the outer or middle ear.[18] Another auditory disorder is of the sensorineural type and is characterized by lesions in the cochlea or involves the eighth cranial nerve.[18] Audiologic tests such as comparative measurements of air- and bone-conduction thresholds help distinguish a conductive hearing loss from a disorder of the sensorineural type.[18] The air- and bone-conduction thresholds interweave in normal hearing. The difference between these two thresholds is called air-bone gap. The air-bone gap provides information about the magnitude of conductive hearing loss. The air-bone gap of 20–30 dB indicates a mild conductive hearing loss, 30–

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45 dB a moderate conductive hearing loss, and 45–60 dB a maximum hearing loss.[18,19]

According to Laptook,[17] cases of conductive hearing loss often have some of the features of the skeletal development syndrome. He suggested that the orthopedic effect of the RME procedure helps improve hearing loss because of a more normal functioning of the pharyngeal ostia of the eustachian tubes. Braun[20] observed that mouth breathing is an aberrant respiratory function and can cause extensive tracking to the most distant corners of the system, from the eustachian tubes to the middle ear, and may cause hearing loss. One of the causes of nasal stenosis was maxillary constriction.[20] Rudolf[21] stated that tubal malfunction is more frequently seen in children who have extremely high palatal arches as well as malformations of the palate and nasopharynx that may predispose to otitis media. Gray[22] found that recurrent serous otitis media decreased remarkably in patients who had undergone RME. Laptook[17] used RME for the treatment of a patient who had conductive hearing loss and reported improved hearing in the first 1.5 weeks and also noted that this improvement continued during the active phase of treatment. Timms[23] also reported that patients had improved hearing and speech after RME. Hazar et al[24] reported on a patient with conductive hearing loss who showed significant improvement in hearing and a decrease in air-bone gap within four weeks of RME. In addition to these case reports, Ceylan et al[25] performed RME on 14 patients with conductive hearing loss, and they found that hearing levels were significantly improved during the active expansion period. They observed some relapse in the hearing level after the retention period, but it did not significantly affect the overall results obtained.

Although there are numerous reports in the literature on the short-term effects of RME on conductive hearing loss, there is scarcity of information on the long-term results. The purpose of this study was to evaluate the effects of RME on conductive hearing loss over a two-year period.

MATERIALS AND METHODS

The sample comprised 35 subjects, 21 girls and 14 boys, who underwent RME at the Department of Orthodontics, Faculty of Dentistry, Atatürk University. Each patient had severe maxillary arch constriction and high palatal vault. The age of the subjects ranged between 13 and 16 years, with a mean of 14 years 6 months. Each patient was examined for conductive hearing loss by an otolaryngologist with the help of pure-tone audiograms. The patient cooperation was very positive, and the otolaryngologist did not need any other examinations to determine hearing loss. Hearing losses were minimal in 11 patients, mild in 19 patients, and severe in five patients. Some patients, who had minimal or mild hearing loss, were unaware of their hearing disabilities. None of the patients had undergone any kind of medical treatment before RME treatment.

Pure-tone audiometric records of all subjects were taken at the Department of Otolaryngology, Faculty of Medicine, Atatürk University. The records were taken for each patient at four different times in a room isolated from outside sounds. The first pure-tone audiograms were taken before RME. Hyrax-type banded RME appliances were cemented to the maxillary teeth, and the patients were instructed to activate the screw three times a day for three days. After midpalatal suture opening (radiographically determined) and the creation of a midline diastema, activation was reduced to two times a day until the complete elimination of the posterior crossbite.

The second audiometric recording was taken after satisfactory expansion of the maxillary arch was obtained (approximately 18 days later). After the satisfactory expansion, same RME device was fixed in the mouth and used for retention device. The third recording was taken at the end of the retention period (approximately six months later), at which time the mineralization of the suture was completed. At the end of these six months, rigid transpalatal arch with the extension throughout anterior teeth was inserted to the mouth and used for two years. The fourth and final audiometric record was taken two years from the end of the retention period. None of the patients received medical treatment during the two-year follow-up period.

All audiometric records were evaluated by an otolaryngologist. The thresholds at four speech frequencies, 250, 500, 1000, and 2000 Hz, were obtained separately for each ear. In addition, air-bone gaps at frequencies of 500, 1000, and 2000 Hz were also recorded.

Statistical analysis

The descriptive statistics, including means and standard deviations for the measurements, were calculated at each of the four periods separately. The data were evaluated using the analysis of variance. To determine at which periods the changes were significant, the least square difference (LSD) test was used. Significance was predetermined at the .05 level of confidence.

RESULTS

Tables 1 and 2 summarize the means and standard deviations for the air-bone gaps and pure-tone thresholds at different speech frequencies for each ear. The results of the analysis of variance for hearing levels are presented in Table 3. There was a statistically significant difference in the hearing levels ($F = 12.45$ and $P < .001$). The results of the LSD test indicated that the improvements between the first and all other recordings were statistically significant ($P < .001$). On the other hand, the changes between the second and third recordings, the second and fourth recordings, and the third and fourth recordings were not statistically significant (Table 4).

The results of the analysis of variance for the air-bone

TABLE 1. The Mean and Standard Deviations (SD) for Air-Bone Gap Measurements at Different Speech Frequencies and Time Intervals in Decibels

Frequency (Hz)	First Record		Second Record		Third Record		Fourth Record	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
500								
Right ear	25.6	6.2	18.2	7.8	16.8	7.6	17.2	7.5
Left ear	24.8	4.9	17.2	4.8	16.6	5.4	14.8	4.7
1000								
Right ear	24.0	3.8	19.6	3.5	16.4	4.2	15.6	3.9
Left ear	24.0	4.1	20.6	3.5	19.8	4.0	20.0	3.5
2000								
Right ear	23.8	4.4	19.2	4.3	19.4	3.9	19.6	5.0
Left ear	23.2	4.8	19.8	4.2	19.8	5.3	20.0	4.8

TABLE 2. The Mean and Standard Deviations (SD) of Pure-Tone Thresholds at Different Speech Frequencies and Time Intervals in Decibels

Frequency (Hz)	First Record		Second Record		Third Record		Fourth Record	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
250								
Right ear	27.2	8.3	22.8	7.9	23.6	8.0	24.4	7.7
Left ear	27.6	8.3	23.2	8.4	22.8	8.8	23.0	8.8
500								
Right ear	27.4	8.7	22.6	7.5	24.0	7.9	23.8	7.8
Left ear	27.8	8.9	23.6	7.8	23.0	7.8	22.6	7.8
1000								
Right ear	25.4	6.8	20.8	7.6	22.2	8.3	21.4	7.3
Left ear	22.8	6.5	20.4	6.6	19.2	6.7	19.0	7.6
2000								
Right ear	19.0	6.1	14.8	5.3	16.8	5.8	16.0	5.6
Left ear	18.4	6.1	16.8	5.6	16.0	5.8	15.8	6.0

TABLE 3. Result of the Analysis of Variance for Hearing Levels

Sources of Variation	*F* value
Recording time	12.45***
Speech frequency	47.66***
Ear (right-left)	0.22
Ear × recording time	0.44
Ear × speech frequency	0.44
Recording time × speech frequency	0.99
Ear × Recording time × speech frequency	1.00

*** $P < .001$.

TABLE 5. Results of the Analyses of Variance for Air-Bone Gaps

Sources of Variation	*F* Value
Recording time	53.58***
Speech frequency	5.98**
Ear (right-left)	0.29
Ear × recording time	0.50
Ear × speech frequency	0.004**
Recording time × speech frequency	0.002**
Ear × Recording time × speech frequency	0.46

** $P < .01$.
*** $P < .001$.

gaps are presented in Table 5. There was a statistically significant relationship (F = 53.58 and $P < .001$) present between the recording time and the decrease in the air-bone gap. The results of the LSD test (Table 4) indicated that the improvements between first and all other recordings were significant ($P < .001$). The differences between the second and fourth recordings were also statistically significant ($P < .05$). On the other hand, the differences between the second and third recordings and that between the third and fourth recordings were not significant (Table 4).

DISCUSSION

RME is one of the treatment modalities for the correction of maxillary constriction with posterior crossbites. RME increases the width of the nasal passages and improves respiration.[5–7,26] Braun[20] indicated that maxillary constriction is one of the causes of nasal stenosis, which may be associated with mouth breathing, can affect the Eustachian tubes and the middle ear, and result in hearing loss.

The maxilla articulates with 10 other bones of the face and cranium. Because of their relative rigidity, skeletal tissues offer immediate resistance to the expansion force. The main resistance to midpalatal suture opening is not in the suture itself but mainly in the surrounding structures, particularly the sphenoid and zygomatic bones.[9] There are two distinct stages in palatal expansion, active adjustment of the screw and the passive retention to allow healing. These stages are mediated by the stabilization of the appliance.[7] A number of authors[10,11,27–33] suggested that a three- to six-month retention period is sufficient for the ossification of the midpalatal suture and reorganization and stabilization of the other maxillary sutures. Other authors[7,27,34] suggest that a longer retention period is needed.

In animal studies, RME resulted in cranioskeletal dis-

TABLE 4. Results of Least Significant Difference (LSD) Tests Comparing the Mean Hearing Levels and Air-Bone Gaps

	Before RME, 1	After RME, 2	After six mo, 3	After two y, 4	Comparisons of the Mean Differences 1 − 2	1 − 3	1 − 4	2 − 3	2 − 4	3 − 4
Hearing levels	24.45	20.63	20.95	20.75	***	***	***	NS	NS	NS
Air-bone gaps	24.23	19.10	18.13	17.86	***	***	***	NS	*	NS

NS indicates not significant; * $P < .05$ and *** $P < .001$.

placements further from the site of actual expansion.[35,36] The skeletal changes that occur in the mouth, oropharynx, nasal cavity, and nasopharynx tend to modify the soft tissue architecture overlying these bony structures.[4,37] In addition, the soft tissue response plays an important role vis-a-vis the stability of the results.[4,17,37] Some patients affected with hearing loss also have a history of recurrent upper respiratory tract infections.[20,21,38] The general improvement in nasal physiology as a result of RME minimizes the drying of the pharyngeal mucosa and decreases the upper respiratory tract infections and otitis media. The latter is a common cause of conductive hearing loss.[13,21,23,39]

Progressive deafness occurs through an increase in the tympanic membrane concavity as a result of pressure loss. Chronic otitis media is an example of conduction deafness because in this disorder air conduction is impaired.[21,38,40] With RME, palatal and pharyngeal soft tissues can be modified and tubal ostia may function more normally.[17,39] As a result, air passes through the tube, and pressures on both sides of the tympanic membrane are balanced. Thus, the tympanic cavity and the ossicular chain can vibrate freely and function normally.[21,39]

Pure-tone air- and bone-conduction threshold testing provides a good profile of an individual's hearing.[25] Conductive hearing loss due to middle ear stiffness primarily affects low frequencies.[41] Hence, the thresholds at high frequencies of 4000 and 8000 Hz affected by middle ear mass or by inner ear nerve damage were excluded from this study.

In this study, significant improvements in both hearing levels and air-bone gaps were achieved at the completion of the active expansion period. These findings are similar to the observations by Laptook,[17] Timms,[23] and Hazar et al[24] as well as the study by Ceylan et al.[25] Although Laptook,[17] Timms,[23] and Hazar et al[24] reported that the results were not transient, Ceylan et al[25] found that at the end of the 4.5-month retention period, some of the improvement was lost but not at a significant level. In addition, they stated that the decrease in air-bone gap measurements affected hearing positively. In this study, 26 of 35 patients (74%) demonstrated clinically significant and stable improvement in their hearing. The improvements in hearing levels and air-bone gaps appeared to be evident at the third and fourth recordings, ie, two years after expansion.

The middle ear is connected by the eustachian tube to the nasopharynx, which in turn communicates with the nasal cavities and the oropharynx. These latter structures and the palate may both influence the functioning of the eustachian tube.[42] RME has certain effects on the nasal cavity and palate. Hence, it may also indirectly affect the eustachian tube functions. In addition, tensor veli palatini muscle may affect hearing improvements. This muscle has its origin at or near the eustachian tube orifices and inserts into the soft palate and plays a role in the opening of eustachian tube orifices.[42] The relation between tensor veli palatini muscle and tubal function was shown in the literature.[43,44] After RME, this muscle may extend and open the eustachian tube orifice. As a result, air passes through the tube, and the ossicular chain functions normally.

According to long-term studies evaluating the stability of RME, most authors[4,5,26,45–47] observed that some relapse occurred during the retention period, whereas others[26,29,34] did not. The nonsignificant reversal in the hearing level observed in the third recording in this study could be related to the relapse tendency of the hard and soft tissues. On the other hand, the stability of the hearing improvements in this study could be attributed to the rigid retention device used and the longer retention period when compared with the other RME and conductive hearing loss studies. According to Timms,[48] the most important feature of RME is that no relapse of the basal bone occurs if adequate retention is maintained initially. This has been shown similarly in respiration.[12] Similarly, in our study, nonsignificant relapse was observed in hearing due to adequate retention maintenance.

Improvement in conductive hearing loss is considered as a possible additional benefit of RME treatment. It does not indicate that people with conductive hearing loss should consider this a treatment approach without an accompanying maxillary constriction.

CONCLUSIONS

In this study, RME had a positive and significant effect on the hearing levels of subjects with conductive hearing loss after the expansion period. At the end of the retention period, the improvement tended to reverse, but the reversal was clinically of small magnitude and not statistically significant two years after the retention period. On the other hand, in 26% of the patients, improvements in hearing levels were not statistically significant. The difference in the improvement of the hearing levels among patients should be the subject of further studies.

REFERENCES

1. Da Silva Filho OG, Villas Boas MC, Capelozza Filino L. Rapid maxillary expansion in the primary and mixed dentitions: a cephalometric evaluation. *Am J Orthod.* 1991;100:171–181.
2. Biederman W, Chem B. Rapid correction of Class III malocclusion by midpalatal expansion. *Am J Orthod.* 1973;63:47–55.
3. Timms DJ. *Rapid Maxillary Expansion.* Chicago, Ill: Quintessence Publishing Co Inc; 1981:51–53.
4. Wertz R, Dreskin M. Midpalatal suture opening: a normative study. *Am J Orthod.* 1977;71:367–381.
5. Wertz RA. Skeletal and dental changes accompanying rapid midpalatal suture opening. *Am J Orthod.* 1970;58:41–66.
6. Haas AJ. Rapid expansion of the maxillary dental arch and nasal cavity by opening the midpalatal suture. *Angle Orthod.* 1961;31:73–90.
7. Haas AJ. The treatment of maxillary deficiency by opening the midpalatal suture. *Angle Orthod.* 1965;35:200–217.
8. Braun S, Bottrel JA, Lee KG, Lunazzi JJ, Legan HL. The bio-

mechanics of rapid maxillary sutural expansion. *Am J Orthod.* 2000;118:257–261.
9. Bishara SE, Staley RN. Maxillary expansion: clinical implications. *Am J Orthod.* 1987;91:3–14.
10. Hartgerink DV, Vig PS, Abbott DW. The effect of rapid maxillary expansion on nasal airway resistance. *Am J Orthod.* 1987;92:381–389.
11. Melsen B. Palatal growth studied on human autopsy material: a histologic microradiographic study. *Am J Orthod.* 1975;68:42–54.
12. Linder-Aronson S, Aschan G. Nasal resistance to breathing and palatal height before and after expansion of the median palatal suture. *Odont Revy.* 1963;14:254–270.
13. Hershey HG, Stewart BL, Warren DW. Changes in nasal airway resistance associated with rapid maxillary expansion. *Am J Orthod.* 1976;69:274–284.
14. Wertz RA. Changes in nasal airflow incident to rapid maxillary expansion. *Angle Orthod.* 1968;38:1–11.
15. Memikoglu UT, Iseri H. Effects of a bonded rapid maxillary expansion appliance during orthodontic treatment. *Angle Orthod.* 1999;69:251–256.
16. Basciftci FA, Mutlu N, Karaman AI, Malkoc S, Kucukkolbasi H. Does the timing and method of rapid maxillary expansion have an effect on the changes in nasal dimensions? *Angle Orthod.* 2002;72:118–123.
17. Laptook T. Conductive hearing loss and rapid maxillary expansion. *Am J Orthod.* 1981;80:325–331.
18. Dirks DD, Morgan DE. Auditory function tests. In: Bailey BJ, ed. *Head and Neck Surgery—Otolaryngology.* Vol 2. Philadelphia, Pa: Lippincott; 1993:1489–1504.
19. Glasscock ME, Shambaugh GE, Johnson GD. *Surgery of the Ear.* Philadelphia, Pa: Saunders; 1990:60.
20. Braun F. A contribution of the problem of bronchial asthma and extension of the palatal suture. *Trans Eur Orthod Soc.* 1966;42:363.
21. Rudolph AM. *Pediatrics.* 16th ed. New York, NY: Appleton-Century-Crofts; 1997:954–968.
22. Gray LP. Results of 310 cases of rapid maxillary expansion selected for medical reasons. *J Larnygol Otol.* 1975;89:601–614.
23. Timms DJ. Some medical aspects of rapid maxillary expansion. *Br J Orthod.* 1974;1:127–132.
24. Hazar S, Günbay MU, Sandikçioğlu M, Kırkım G. Hızlı üst çene genişletmesi ve iletim tipi işitme kaybı. *Ege Ortod.* 1992;1:15–17.
25. Ceylan İ, Oktay H, Demirci M. The effect of rapid maxillary expansion on conductive hearing loss. *Angle Orthod.* 1996;66:301–308.
26. Haas AJ. Just the beginning of dentofacial orthopedics. *Am J Orthod.* 1970;57:219–255.
27. Korkhaus G. Present orthodontic thought in Germany. *Am J Orthod.* 1960;46:187–206.
28. Bell RA. A review of maxillary expansion in relation to rate of expansion and patient's age. *Am J Orthod.* 1982;81:32–37.
29. Haas AJ. Long term posttreatment evaluation of rapid palatal expansion. *Angle Orthod.* 1980;50:189–217.
30. Mew J. Relapse following maxillary expansion. *Am J Orthod.* 1983;83:56–61.
31. Chaconas SJ, Caputo AA. Observation of orthopedic force distribution produced by maxillary orthodontic appliances. *Am J Orthod.* 1982;82:492–501.
32. Storey E. Tissue response to the movement of bones. *Am J Orthod.* 1973;64:229–247.
33. White RE. A cephalometric appraisal of changes in the maxillofacial complex resulting from palatal suture expansion utilizing fixed appliance therapy [abstract]. *Am J Orthod.* 1972;61:527–528.
34. Thörne NAH. Expansion of the maxilla. Spreading the midpalatal suture; measuring the widening of the apical base and the nasal cavity on serial roentgenograms [abstract]. *Am J Orthod.* 1960;40:626.
35. Haas AJ. *Gross Reactions to the Widening of the Maxillary Dental Arch of the Pig by Splitting the Hard Palate* [master's thesis]. Chicago, Il: University of Illinois; 1957.
36. Gardner GE, Kronman JH. Cranioskeletal displacements caused by rapid palatal expansion in the rhesus monkey. *Am J Orthod.* 1970;58:41–65.
37. Starnbach HK, Cleall JF. The effects of splitting the midpalatal suture on the surrounding tissues. *Am J Orthod.* 1964;50:923.
38. Nelson WE. Textbook of Pediatrics. In: Vaughan VC, McKay RJ, eds. *The respiratory system.* 7th ed. Philadelphia, Penn, London, England: WB Saunders Company; 1959:764–770.
39. Timms DJ. A study of basal movement with rapid maxillary expansion. *Am J Othod.* 1980;77:500–507.
40. Selkurt EE (editor). *Physiology.* 2nd ed. Boston, Mass: Little, Brown & Company; 1966:110–113.
41. Lee KJ. *Essential Otolaryngology. Head and Neck Surgery.* New York, NY: Med Ex Pub Co.; 1991;26–33.
42. Fingeroth AI. Orthodontic-orthopedics as related to respiration and conductive hearing loss. *J Clin Pediatr Dent.* 1991;15:83–89.
43. Bumsted RM. Velopharyngeal incompetence. In: English GM, ed. *Otolaryngology.* Vol 4. Philadelphia, Pa: Lippincott; 1988:1–3.
44. Cantekin EI, Phillips DC, Doyle WJ, Bluestone CD, Kimes KK. Effect of surgical alterations of the tensor veli palatini muscle on eustachian tube function. *Ann Otol Rhinol Laryngol.* 1980;89:47–53.
45. Krebs AA. Rapid expansion of mid-palatal suture by fixed appliance. An implant study over a 7 year period. *Trans Eur Orthod Soc.* 1964;141–142.
46. Timms DJ. Long term follow-up of cases treated by rapid maxillary expansion. *Trans Eur Orthod Soc.* 1976;211–215.
47. Hicks EP. Slow maxillary expansion: a clinical study of the skeletal vs. dental response to low magnitude force. *Am J Orthod.* 1978;73:121–141.
48. Timms DJ. Rapid maxillary expansion in the treatment of nasal obstruction and respiratory disease. *Ear Nose Throat J.* 1987;66:242–247.