

# The effect of rapid maxillary expansion on conductive hearing loss

İsmail Ceylan, DDS, PhD; Hüsametdin Oktay, DDS, PhD;  
Münir Demirci, MD

**A**uditory disorders are broadly classified as conductive (external and middle ear) and sensorineural (referring to lesions in the cochlea or involving the eighth nerve). The major dysfunction observed in conductive disorders is a loss in hearing sensitivity reflected by elevated air-conduction thresholds. The loss varies greatly depending on the severity and type of physical change imposed on the mechanical system of the outer or middle ear. Because the cochlea and eighth nerve are often unimpaired in individuals with only a conductive lesion, bone-conduction thresholds are found at normal or near-normal levels.<sup>1</sup>

The primary audiologic tests to distinguish a conductive hearing loss from a sensorineural one are the comparative measurements of air- and bone-conduction thresholds and acoustic

immittance as measured at the plane of the tympanic membrane. The magnitude of the hearing loss is usually measured at selected octave frequencies within the major useful range of human hearing (0.25 to 8.0 kHz).<sup>1</sup> Hearing abilities vary, and threshold values within 20 dB of 0 are considered normal hearing, according to the American National Standards Institute.<sup>2</sup>

In normal hearing, air- and bone-conduction thresholds interweave. The difference between these two thresholds—called the air-bone gap—provides an estimate of the magnitude of the conductive component and has considerable clinical usefulness. An air-bone gap of 20 to 30 dB indicates a mild or very early conductive loss; 30 to 45 dB a moderate conductive loss; and 45 to 60 dB a maximum conductive loss.<sup>1,3</sup>

Maxillary arch contraction or maxillary width

## Abstract

The effects of rapid maxillary expansion (RME) on conductive hearing loss were investigated in 14 subjects (11 females and 3 males). The subjects ranged in age from 10 years 4 months to 16 years 9 months (mean age 12 years 11 months  $\pm$  1 year 9 months) and had narrow maxillary arches and conductive hearing loss. Hearing levels were determined by means of pure-tone audiometric records. Three records were taken for each subject. The first was taken before RME, the second after sufficient midpalatal suture opening was achieved (mean=15 days), and the third after the retention period (mean=4.5 months). All the audiometric records were assessed by an otolaryngologist. Changes in both hearing level and air-bone gap were investigated by means of analysis of variance. It has been determined that hearing improved at a statistically significant level ( $P<0.05$ ) after the active treatment period, but that the improvement reversed at the end of the retention period. Five patients experienced significant and stable hearing improvement over the duration of this study.

## Key Words

Rapid maxillary expansion • Conductive hearing loss

Submitted: April 1995

Revised and accepted: August 1995

Angle Orthod 1996;66(4):301-308.

deficiency, concomitant with a high palatal vault, is a manifestation of a skeletal development syndrome that causes some rhinologic problems and has certain negative effects on the dentofacial pattern. Some of the more typical features of this syndrome are (1) decrease in nasal permeability resulting from nasal stenosis, (2) elevation of the nasal floor, (3) mouth breathing, (4) bilateral dental maxillary crossbite combined with a high palatal vault, (5) and a decrease in nasal airway size because of enlargement of the nasal turbinates.<sup>4</sup>

The dental manifestations of this malocclusion are generally treated orthodontically by rapid maxillary expansion (RME). RME is a dramatic procedure with a long history. This treatment procedure was introduced by E.C. Angell in 1860, and has gone through subsequent periods of popularity and decline.<sup>5</sup> Graber<sup>6</sup> advocated RME for the treatment of cleft lip and palate patients in the late 1940s. As a result of the studies made by Haas,<sup>7,8</sup> RME gained more attention in the 1960s. At the present time, this treatment procedure is being applied successfully to both children and young adults in most orthodontic departments.

Although the main object of RME is to correct maxillary arch narrowness, its effects are not limited to the upper jaw. The maxilla is associated with 10 bones in the face and head, so RME may affect structures directly or indirectly related to the maxilla,<sup>4,5,9-12</sup> mandible,<sup>5,7,8,12-18</sup> nasal cavity,<sup>4,7,8,13-16,18-25</sup> pharyngeal structures,<sup>4,11,26</sup> temporomandibular joint,<sup>12</sup> middle ear,<sup>4,11,18,27,28</sup> and the pterygoid process of the sphenoid bone.<sup>11</sup> RME has also been reported to cause improvements in breathing, to correct dental crossbite and crowding,<sup>7,8,13,15,20,22-24,26,29</sup> and to restore conductive hearing loss due to middle ear and Eustachian tube problems.<sup>4,11,18,20,22,28</sup>

The Eustachian tube connects the tympanic cavity to the nasal part of the pharynx, and its orifice lies on its respective lateral nasal pharyngeal wall.<sup>4</sup> Physiologic obstruction of the Eustachian tube comes from the tensor veli palatini muscles, at their origins. The tensor and levator palatini muscles are part of the soft palate. This keeps the tube from opening in response to negative pressure in the middle ear. Negative pressure in the middle ear may, by itself, be another cause of tubal malfunction. The physiologic malformation is seen in all the diseases that affect the palatal musculature and the shape of the nasopharynx.<sup>30</sup> If the tube is blocked, air in the tympanic cavity is absorbed into the mucosal cells (and may at times be replaced with serous or

mucous secretions) with loss of pressure, increasing concavity of the tympanic membrane, and progressive deafness.<sup>30</sup> Braun<sup>20</sup> believed that a relationship existed between conductive hearing loss and maxillary width deficiency. Rudolph<sup>30</sup> stated that tubal malfunction is seen more frequently in children who have extremely high palatal arches, and that malformations of the palate and nasopharynx are predisposing factors for otitis media.

Gray<sup>22</sup> found that recurrent serous otitis media decreased remarkably in patients who had undergone RME. After Laptok<sup>4</sup> applied RME to a patient who had conductive hearing loss, he reported that hearing improved dramatically within the first 1.5 weeks and the improvement continued during the active treatment period. Timms<sup>18</sup> reported on the medical aspects of 200 patients treated with RME and cited a case that was related to a hearing problem. He reported that the patient's hearing problem improved within the first week of the expansion. Hazar and co-workers<sup>28</sup> also observed that a patient with conductive hearing loss experienced significantly improved hearing after four weeks of RME, and that the air-bone gap decreased.

The relationship between RME and changes in hearing levels has not been investigated statistically in a large sample. The purpose of this study was to determine whether or not RME has any effect on conductive hearing loss.

### Materials and methods

Pure-tone audiometric records of 14 subjects, 11 females and 3 males, who underwent RME at the Department of Orthodontics in the Faculty of Dentistry at Atatürk University were examined. Each patient had a severe maxillary width deficiency and deep palatal vault and was chosen at random. Hearing losses were rated minimal in one patient, severe in two patients, and mild in 11 patients. Some patients, especially those with minimal or mild hearing disorders, were unaware of their hearing loss. The age range of the subjects was 10 years 4 months to 16 years 9 months, and the mean age was 12 years 11 months $\pm$ 1 year 9 months.

Pure-tone audiometric records were taken for each patient at three different times under standard conditions, in a room isolated from outside sounds. The patient was seated, and the earphones of the test machine were placed on the patient's ears. A tone was presented at a reasonably loud level for recognition by the patient. The tone was then attenuated in large steps (10-15 dB) until the patient was unable to hear the

**Table 1**  
Range, mean, and standard deviation of pure tone thresholds  
at speech frequencies (decibel) (n=14)

Frequency (Hz)	First record				Second record				Third record			
	Min.	Max.	Mean	S.D.	Min.	Max.	Mean	S.D.	Min.	Max.	Mean	S.D.
250												
Right ear	10	60	23.21	14.76	10	50	18.93	10.03	10	50	23.57	10.27
Left ear	10	50	23.93	12.58	10	70	22.14	15.15	15	45	22.14	9.35
500												
Right ear	10	55	23.71	13.56	10	45	18.57	8.42	10	40	21.43	8.42
Left ear	10	65	24.29	14.92	10	75	21.43	16.34	10	40	20.71	9.38
1000												
Right ear	10	50	22.14	11.55	10	50	18.21	9.92	10	35	20.00	6.50
Left ear	5	55	19.29	13.42	5	70	18.21	16.24	10	35	17.86	6.11
2000												
Right ear	10	40	16.79	10.12	5	45	12.86	10.14	5	30	16.43	7.45
Left ear	5	40	16.07	8.81	5	65	16.79	14.76	5	30	13.93	6.26
Mean frequency												
Right ear	10	47	20.93	11.55	8	47	16.29	9.57	8	35	19.07	6.93
Left ear	7	53	19.86	12.01	7	70	18.79	15.62	8	35	17.29	6.73

stimulus. The threshold was then established by systematically increasing the tone in 5 dB steps until a response was observed. The lowest level at which the responses occurred in at least 50% of a series of ascending trials, or a minimum of two responses out of three at a constant level, was accepted as the estimate of threshold. The procedure for measuring bone-conduction thresholds was similar except that the signal was transduced by a vibrotactile stimulator, usually coupled to the mastoid process.

The first pure-tone audiograms were taken before RME and the cementing of a Biedermann-type RME appliance<sup>31</sup> with Hyrax screw (Dentaurum 602-813) to the maxillary teeth of the patients with orthodontic requirements. The patients were instructed to activate the screw 0.2 mm three times a day for 3 days. After the screw was widened 2 mm, the patients were examined clinically and radiographically.

After a midline diastema occurred, indicating sutural opening, widening of the screw continued with two turns of 0.2 mm per day until the posterior crossbite was eliminated. The second audiometric record was taken after satisfactory widening was obtained at the midpalatal suture, and the third record after the ossification of the suture was completed, approximately 4.5 months later.

All the audiometric records were evaluated by an otolaryngologist, and the pure-tone threshold for each patient was determined. The thresholds at the speech frequencies of 250, 500, 1000, and

2000 Hz were recorded separately for each ear. In addition, the means of the measurements at the frequencies of 500, 1000, and 2000 Hz were computed for each patient. The air-bone gaps at the frequencies of 500, 1000, and 2000 Hz also were recorded. The data regarding the hearing level and the air-bone gaps were analyzed by analysis of variance with randomized block designs with  $3 \times 5 \times 2 \times 14$  factors and  $3 \times 3 \times 2 \times 14$  factors,<sup>32</sup> respectively. The least square difference (LSD) test was applied to reveal between which periods the changes of measurements were significant.

## Results

The mean durations of active widening and retention were  $14.92 \pm 1.90$  days and  $4.50 \pm 1.16$  months, respectively. The mean midpalatal suture opening of  $6.20 \pm 0.60$  mm was achieved at the end of the active widening period. Tables 1 and 2 summarize the ranges, the mean values, and the standard deviations of the pure-tone thresholds and air-bone gaps for each ear. The results of the analysis of variance testing the changes at the hearing levels are shown in Table 3.

As indicated in Table 3, hearing levels improved at a statistically significant level ( $P = 0.043$ ). According to the results of the LSD test applied to explain the significance in Table 3, the difference between the first and second records was statistically significant ( $P < 0.05$ ), but the changes between the first and third records and

**Table 2**  
The ranges, means, and standard deviations for  
air-bone gap measurements (decibel) (n=14).

Frequency (Hz)	First record				Second record				Third record			
	Min.	Max.	Mean	S.D.	Min.	Max.	Mean	S.D.	Min.	Max.	Mean	S.D.
250												
Right ear	10	35	23.21	7.75	15	40	22.50	5.80	10	30	20.71	6.16
Left ear	10	55	22.64	11.41	0	55	20.50	12.97	5	25	19.29	5.50
500												
Right ear	15	30	21.79	3.73	15	40	22.86	6.11	10	30	18.93	5.94
Left ear	5	45	19.64	8.87	0	50	18.57	11.34	10	35	18.93	6.26
1000												
Right ear	10	35	19.64	6.64	10	40	17.14	8.48	5	30	16.07	7.12
Left ear	5	35	17.86	6.99	10	55	20.00	11.09	10	25	15.71	5.50

between the second and third were not significant. As Table 1 shows, an important improvement in hearing was achieved after the active widening period, but this positive effect reversed at a nonsignificant level during the retention period.

The results of the variance analysis concerning the air-bone gaps are shown in Table 4. A nearly significant relationship ( $P=0.061$ ) was found between the recording time and the decrease at the air-bone gap. According to the results of the LSD test applied to the recording time, the measurements of the air-bone gap decreased at a statistically significant level ( $P<0.05$ ) only between the first and third records.

### Discussion

Braun<sup>20</sup> pointed out that constriction of the maxillary dental arch causes nasal stenosis and oral respiration, and that the aberration from normal breathing patterns can even affect the Eustachian tube and the middle ear, and thus result in hearing loss in some instances. According to Timms,<sup>18,31</sup> RME can be applied to patients with nasal stenosis even in the absence of posterior dental crossbite, since its medical priorities are higher and the risk of worsening the malocclusion is not great. Most orthodontists accept this procedure as a treatment modality for the correction of a posterior dental crossbite due to maxillary deficiency. It is expected, however, that the operation has various effects on structures directly or indirectly related to the maxilla.

The question then must be raised as to how, with even limited bone displacement at regions far from the jaws, improvements in hearing occur with the RME. Laptok,<sup>4</sup> Wertz and Dreskin,<sup>16</sup> and Starnbach and Cleall<sup>26</sup> stressed the importance of the soft-tissue response. These authors stated that skeletal changes that occur in the mouth, oropharynx, nasal cavity, and nasopharynx tend to modify the soft-tissue architecture overlying these bony structures. Timms,<sup>11</sup> Hershey and co-workers,<sup>23</sup> and Rudolph<sup>30</sup> noted that the changes occur not only in the configuration but also in the tissue response. The improvement of nasal air flow results in an improvement in the nasal physiology, a cessation of drying of pharyngeal mucosa, and a decrease in upper respiratory infections and otitis media—the most common cause of conductive hearing loss.<sup>11,23,30</sup>

It might be thought that the muscle and fibrous attachments affect hearing improvements. The tensor and levator veli palatini muscles originate at or near the pharyngeal orifice of the Eustachian tube and end in the soft palate.<sup>33</sup> The fibrous portion of the soft palate attached to the posterior margin of the bony palate is the palatal aponeurosis, which continues laterally with the tendons of the tensor veli palatini muscle.<sup>34</sup> It has been shown by several types of surgical alterations of this muscle that the tensor veli palatini muscle is related to middle ear aeration and tubal function.<sup>34,35</sup> It is possible that the stretching that occurs in this muscle following

**Table 3**  
**Variance analysis of hearing levels**

Sources of variation	Degree of freedom	Sum square	Mean square	F Value	P Value
Recording time	2	562.18	281.09	3.18	0.043
Frequency	4	2489.06	622.27	7.05	0.000
Ear (right-left)	1	0.15	0.15	0.00	0.967
Recording time x frequency	8	77.54	9.69	0.11	0.999
Recording time x ear	2	337.23	168.62	1.91	0.149
Frequency x ear	4	91.44	22.86	0.26	0.904
Recording time x frequency x ear	8	43.10	5.39	0.06	1.000
Patients	13	16895.66	1299.67	14.72	0.000
Error	377	33280.63	88.28		

**Table 4**  
**Variance analysis of air-bone gaps.**

Sources of variation	Degree of freedom	Sum square	Mean square	F Value	P Value
Recording time	2	297.06	148.53	2.83	0.061
Frequency	2	601.56	300.78	5.73	0.004
Ear (right-left)	1	73.40	73.40	1.40	0.238
Recording time x frequency	4	26.02	6.50	0.12	0.974
Recording time x ear	2	8.72	4.36	0.08	0.920
Frequency x ear	2	61.56	30.78	0.59	0.557
Recording time x frequency x ear	4	141.97	35.49	0.68	0.609
Patients	13	3500.67	269.28	5.13	0.000
Error	221	11604.62	52.51		

midpalatal suture opening opens the pharyngeal orifice of the Eustachian tube, thus allowing air to enter or leave the tympanic cavity. By allowing air to pass through the tube, pressures on either side of the tympanic membrane are balanced, so the tympanic cavity and the ossicular chain can vibrate freely and function normally.<sup>4,11</sup>

Pure-tone audiometry used in the present study is the initial and critical measurement for auditory assessment. It provides a description of the magnitude of hearing loss and the pattern of audiometric configuration.<sup>1</sup> Testing by both air- and bone-conduction indicates how much of a hearing loss is due to problems in the transmission or conduction of sound and how much is due to inner ear or nerve damage. Thus, pure-tone air- and bone-conduction threshold testing provides a good profile of an individual's hearing. Generally, conductive hearing loss due to middle ear pathology is due either to middle ear

stiffness, primarily affecting the low frequencies, or to middle ear mass, primarily affecting the high frequencies.<sup>36</sup> Because high frequencies are affected by middle ear mass or by inner ear nerve damage, thresholds at speech frequencies of 4000 and 8000 Hz were excluded from this study.

Our finding, in which a statistically significant improvement was achieved after the active widening period, agrees with the results reported by Laptok,<sup>4</sup> Timms,<sup>18</sup> and Hazar and co-workers.<sup>28</sup> Contrary to our finding, these authors did not report that the results were transient and reversed during the ongoing days. It should be noted, however, that Timms' finding<sup>18</sup> was of a subjective nature, and no records regarding hearing status were taken before or after the RME. The author also had some doubts as to whether a relapse would occur in the improvement of the hearing observed in the first week. Laptok<sup>4</sup> found that the improvement in hearing did not relapse 1.5 years after the operation. In the

present study, five patients demonstrated clinically significant and stable improvement in hearing; in two patients, improvement of about 35 dB was observed, but these results were not supported by statistical analysis of the entire sample. After evaluating all 14 patients, it was determined that the improvements observed in this study were transient and did not appear to be evident at the time records were taken for the third time.

According to long-term studies carried out on the results of RME,<sup>13,15,16,19,37-39</sup> an inevitable relapse occurs during the retention period of the widening procedure. It is possible that the relapse in hearing level observed during the retention period could be due to the relapse in the hard and soft tissues or due to soft tissue adaptation. It is not known whether this reversal in hearing improvement will continue after the retention period.

### Conclusion

It has been observed that RME has a positive and statistically significant ( $P<0.05$ ) effect, during the active widening period, on the hearing levels of subjects with conductive hearing loss. At the end of the retention period, the improvement reversed at a nonsignificant level, causing the change in hearing levels to become nonsignificant. The decrease in air-bone gap measurements between the first and third records could be considered to positively affect hearing.

The fact that a significant improvement was observed in the hearing levels of some subjects, but was not supported by statistical analysis of the entire sample, denotes the need to investigate this topic using a larger sample and a longer investigation period. All audiological records should be included, and researchers should try to determine why some subjects show improvement and others do not.

### Acknowledgment

We express our gratitude to Mr. Yavuz Konca for his help in writing this article in English.

### Author Address

Dr. Hüsametdin Oktay  
Atatürk Üniversitesi Dis Hekimligi  
Fakültesi Ortodonti Anabilim Dalı  
Erzurum, Turkey

İsmail Ceylan, assistant professor, Department of Orthodontics, Faculty of Dentistry, Atatürk University, Erzurum, Turkey.

Hüsametdin Oktay, professor, Department of Orthodontics, Faculty of Dentistry, Atatürk University, Erzurum, Turkey.

Münir Demirci, associate professor, Department of Otolaryngology, Research Hospital, Atatürk University, Erzurum, Turkey.

This study was presented as a poster at the 4th international congress of the Turkish Orthodontic Society, Antalya, Turkey, September, 4-9, 1994.

# References

1. Dirks DD, Morgan DE. Auditory function tests. In Bailey BJ, ed. Head and neck surgery-Otolaryngology, Vol 2. Philadelphia: Lippincott, 1993: 1489-504.
2. Austin DF. Mechanics of hearing. In Glasscock ME, Shambaugh GE, Johnson GD, ed. Surgery of the ear. Philadelphia: Saunders, 1990: 299.
3. Glasscock ME, Shambaugh GE, Johnson GD. Surgery of the ear. Philadelphia: Saunders, 1990: 60.
4. Lupton T. Conductive hearing loss and rapid maxillary expansion: Report of a case. Am J Orthod 1981; 80: 325-31.
5. Bishara SE, Staley RN. Maxillary expansion: clinical implications. Am J Orthod Dentofac Orthop 1987; 91: 3-14.
6. Graber TM, Swain BF. Dentofacial orthopedics. In: Current orthodontic concepts and techniques, Vol 1. Philadelphia: Saunders, 1975.
7. Haas AJ. Rapid expansion of the maxillary dental arch and nasal cavity by opening the mid-palatal suture. Angle Orthod 1961; 31: 73-90.
8. Haas AJ. The treatment of maxillary deficiency by opening the mid-palatal suture. Angle Orthod 1965; 35: 200-17.
9. Gardner GE, Kronman JH. Cranioskeletal displacements caused by rapid palatal expansion in the rhesus monkey. Am J Orthod 1971; 59: 146-55.
10. Fried KH. Palate-tongue reliability. Angle Orthod 1971; 61: 308-23.
11. Timms DJ. A study of basal movement with rapid maxillary expansion. Am J Orthod 1980; 77: 500-7.
12. Bell RA. A review of maxillary expansion in relation to rate of expansion and patient's age. Am J Orthod 1982; 81: 32-7.
13. Haas AJ. Just the beginning of dentofacial orthopedics. Am J Orthod 1970; 57: 219-55.
14. Haas AJ. Longterm posttreatment evaluation of rapid palatal expansion. Angle Orthod 1980; 50: 189-217.
15. Wertz RA. Skeletal and dental changes accompanying rapid mid-palatal suture opening. Am J Orthod 1970; 58: 41-66.
16. Wertz RA, Dreskin M. Mid-palatal suture opening: A normative study. Am J Orthod 1977; 71: 367-81.
17. Gryson JA. Changes in mandibular interdental distance concurrent with RME. Angle Orthod 1977; 47: 186-92.
18. Timms DJ. Some medical aspects of rapid maxillary expansion. Br J Orthod 1974; 4: 127-32.
19. Krebs AA. Rapid expansion of mid-palatal suture by fixed appliance. An implant study over a 7 year period. Trans Eur Orthod Soc 1964; 141-2.
20. Braun F. A contribution of the problem of bronchial asthma and extension of the palatal suture. Trans Eur Orthod Soc 1966; 363.
21. Kressner A. Maxillary orthopaedics and otolaryngology. Trans Eur Orthod Soc 1966; 355-60.
22. Gray LP. Results of 310 cases of rapid maxillary expansion selected for medical reasons. J Laryngol Otol 1975; 89: 601-14.
23. Hershey HG, Steward BL, Warren DW. Changes in nasal airway resistance associated with rapid maxillary expansion. Am J Orthod 1976; 69: 274-84.
24. Montgomery W, Vig PS, Staab EV, Matteson SR. Computed tomography: A three-dimensional study of the nasal airway. Am J Orthod 1979; 76: 363-75.
25. Pavlin D, Vuvcevik D. Mechanical reaction of facial skeleton to maxillary expansion determined by laser holography. Am J Orthod 1984; 85: 498-507.
26. Starnbach HK, Cleall JF. The effects of splitting the mid-palatal suture on the surrounding structures. Am J Orthod 1964; 50: 923-4.
27. Fingerhuth AI. Orthodontic-orthopedics as related to respiration and conductive hearing loss. J Clin Pediatric Dent 1991; 15: 83-9.
28. Hazar S, Günbay MU, Sandıkçıoğlu M, Kırkım G. Hızlı üst çene genişletmesi ve iletim tipi işitme kaybı. Ege Ortodonti 1992; 1: 15-7.
29. Frank SW, Engel GA. The effects of maxillary quad-helix appliance expansion on cephalometric measurements in growing orthodontic patients. Am J Orthod 1982; 81: 378-89.
30. Rudolph AM. Pediatrics, ed. 16, New York: Appleton-Century crofts, 1977; 954-68.
31. Timms DJ. Rapid maxillary expansion. Chicago, Berlin, Rio de Janeiro, and Tokyo: Quintessence, 1981:45-47,86.
32. Yıldız N, Bircan H. Arastırma ve deneme metodları. Erzurum: Atatürk Üniversitesi yayınları. No: 697 Ders Kitapları Serisi No:57, 1994: 122-47.
33. Ross MA. Functional anatomy of the tensor palati: Its relevance in cleft palate surgery. Arch Otolaryng 1971; 93 : 1-8.
34. Bumsted RM. Velopharyngeal incompetence. In: English GM, ed. Otolaryngology, Vol 4. Philadelphia: Lippincott, 1988: 1-3.
35. 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.
36. Lee KJ. Essential otolaryngology. Head and Neck Surgery. New York: Med Ex Pub Co 1991; 26-33.
37. Timms DJ. Long term follow-up of cases treated by rapid maxillary expansion. Trans Eur Orthod Soc 1976; 211-5.
38. Hicks EP. Slow maxillary expansion: A clinical study of the skeletal vs. dental response to low magnitude force. Am J Orthod 1978; 73: 121-41.
39. Linder-Aronson S, Lindgren J. The skeletal and dental effects of rapid maxillary expansion. Br J Orthod 1979; 6: 25-9.
40. Inoue N. Radiographic observation of rapid expansion of human maxilla. Bull Tokyo Med Dent Univ 1970; 17: 249-61.

