Effect of Mouth Breathing on Dental Occlusion

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The role of mouth breathing in the development of malocclusion has been discussed at length in dental literature.[3,5,7,13] Mouth breathing has been stated to have serious effects on the development of the facial skeleton and occlusion of teeth on account of the displacement of normal lateral, buccal, and lingual muscular forces. It is believed to cause disfigurement of features and the undermining of general health. Despite considerable interest in the problem, no uniform opinion exists regarding the association of the mouth breathing habit and the development of malocclusion. Mouth breathing is a habit in which the lower jaw is dropped, the lips are parted, and the tongue is depressed from its normal position.[7] Enlarged adenoids and tonsils are regarded as common causes.[1,5,8,11]

In mouth breathing the air is received directly into the lungs without being cleansed, warmed, and moistened and it tends to lift the palate high.[5] McCoy[12] stated that the mouth is held open constantly and, as a result, the muscles which depress the mandible exert a backward muscular pull upon the mandible with each inspiration. With time this may influence the bone to modify and bring the lower teeth distal to the normal. Once this distal relationship of the molars is established, the permanent teeth will also assume a similar malrelation, and these mechanics of malocclusion operate constantly. The tongue is not held in the roof of the mouth due to mandibular depression and the upper teeth are deprived of their muscular support and lateral pressure in consequence. Because of this imbalanced relationship between external and internal muscular forces about the mouth, the buccinator muscle causes a lateral pressure on the maxillary arch resulting in its narrowing.[4] The upper lip is so little used that it exerts very little influence on the maxillary anterior teeth and is often short. The lower lip rests between the mandibular and maxillary incisors and becomes a factor in pushing the maxillary anterior teeth forward. During the swallowing process the lower lip is usually forced against the mandibular incisors and they are moved lingually and elevated resulting in an exaggerated curve of Spee.[7]

Joshi[10] found that Class II, Division 1 was more common in cases with mouth breathing. However, Brash[3] and Hartsook[6] pointed out that the theory of mechanical forcing of the palate upwards by compression of the cheeks during mouth breathing could not be accepted as it lacked sufficient proof. Ballard[2] believed that the adenoid face and the accompanying malocclusion were due to heredity. Linder-Aronson and Backstrom[11] found no significant differences in overjet, overbite, width of the dental arch and in the percentage distribution of post- and prenormal occlusion in mouth breathers and nose breathers.

The present investigation has been undertaken to compare the occlusion of teeth in mouth breathers and nose breathers and to find out the morphologic differences, if any.

Material and Method

The observations were made on one

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Dental Surgery at the Lucknow University, Lucknow, India.

hundred male individuals of 15-20 years of age divided equally into experimental and control groups. The experimental group of mouth breathers was selected on the basis of having no other abnormal habit or apparent local cause which could be responsible for the malocclusion of teeth. The control group of nose breathers had clinically excellent occlusion. None of the individuals had received any orthodontic treatment in the past and there were no extracted or missing teeth.

To select mouth breathers a cotton wisp test was employed. A small cotton wisp was held alternatively in front of each nostril and the mouth of all the individuals. No movement of cotton wisp when held in front of the nose, but evidence of its movement in front of the mouth indicated mouth breathing. Individuals showing movement of cotton wisp when placed before the nose and no movement in front of the mouth indicated normal nasal breathing. The latter were included in the control group. Care was taken in applying this test in so far as the patient was not under apprehension, or made conscious of this test.

Impressions of maxillary and mandibular dental arches of the selected individuals were taken with irreversible hydrocolloid impression material and various measurements were taken on the study casts.

Landmarks Used

A soft pencil was used to locate the landmarks on the casts. All of these points were placed lingually at the gingival margin of the teeth on the assumption that these points are not affected by attrition or malposition of the teeth.

Bilateral:

1. Mesiodistal midpoint of canines.
2. Mesiodistal midpoint of first premolars.
3. Midpoint of mesiolingual cusp of first molars.
4. Contact point of second premolar and first molar (taken only in maxillary cast).
5. Contact point of first molar and second molar.

Unilateral:

1. Point between the two upper central incisors (intradentale superioris linguale).
2. Point between the two lower central incisors (intradentale inferioris linguale).

Measurements

Intercanine, interpremolar, and intermolar widths were recorded in fractions of a millimeter from the vernier scale. To avoid obstruction from the curvature of the teeth while taking these measurements, the outside calipers B and B′ at the terminal points (shown in Fig. 1) were used.

On the maxillary cast the length of the dental arch was measured from intradentale superioris linguale along the midsagittal plane where it is intersected by a line drawn across the palate connecting the contact point between first and second molars. Arch length in mandible was measured in the same manner. In cases of diastema between the central incisors, the point on the alveolar ridge between the two central incisors in the midsagittal plane was marked for measuring the arch length. A plastic block for removing parallax was used to locate these points on a cellophane paper from which direct arch length measurements were taken.

Height of the palatal vault was measured in the midline from a coronal plane passing through the proximal contact of the maxillary first molar and second premolar by means of an attachment put on the vernier calipers (Fig. 2). The vernier was opened until the central rod touched the palate in midline and the palate height was re-

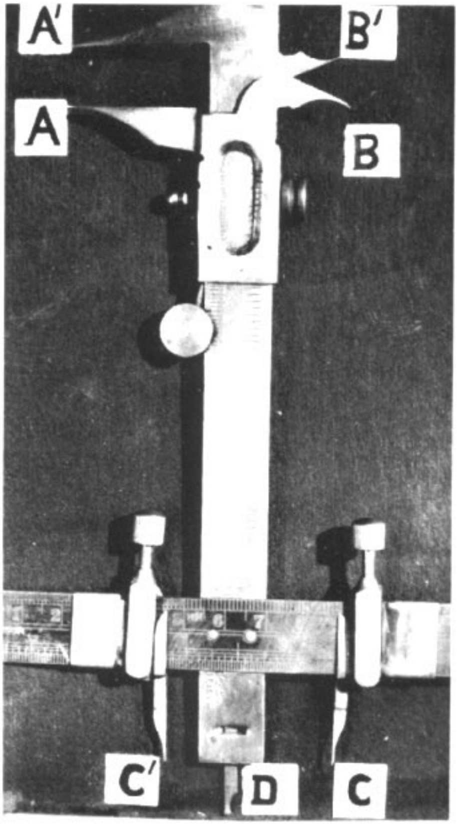

Fig. 1 Vernier calipers with the attachment CC' and D for measuring the height of palatal vault. BB' for measuring intermolar, interpremolar and intercanine width. AA' for recording the overbite.

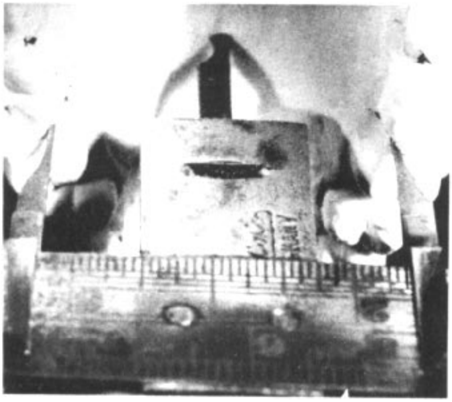

Fig. 2 Method for measuring the height of the palatal vault.

corded from the vernier reading.

The overjet was recorded in the midline by placing a modified one-half millimeter scale against the labial surface of the mandibular incisors and taking the reading where the incisal edge of the maxillary central incisor touched the scale. In cases of unequal protrusion of maxillary incisors, the maximum overjet was recorded from the incisal edge of either right or left maxillary central incisors.

The dental casts in centric occlusion were held at eye level and a fine horizontal scratch was made by a chisel on the labial surface of the mandibular central incisors. Overbite was recorded by measuring the mandibular incisor from its edge to the scratch mark with the inside of calipers A-A′.

The error in location of landmarks and in recording measurements was not found to be statistically significant for all measurements.

Findings

The mean values of arch width measurements in both of the dental arches were found to be lower in the experimental group than the control group (Fig. 3). However, only the intermolar width was found to be statistically significant at the 5 per cent level. The mean value of maxillary arch length was found to be higher by 3.26 mm in the experimental group as compared with the controls. This difference was significant at the 0.1 per cent level. The mean value of palatal height was slightly lower in mouth breathers but the difference was not statistically significant. The mean values of overjet and overbite were found to be higher in the experimental group by 5.48 mm and 1.10 mm, respectively. These differences were found to be highly significant ($P < .001$).

Seventy-four per cent of the mouth breathers had a Class II malocclusion.

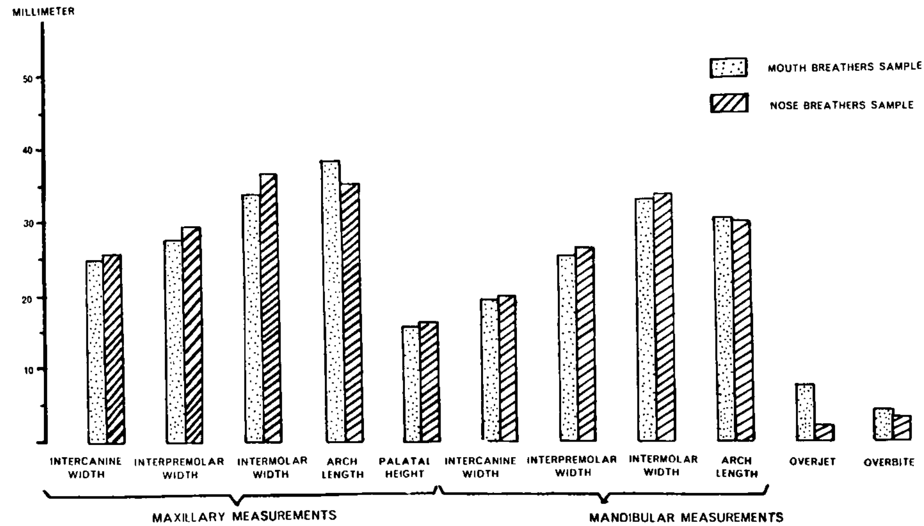

Fig. 3 Comparison of means of the dental cast measurements in the mouth breathing and nasal breathing samples.

The remaining showed Class I malocclusion. Chi-square test indicated that the association of Class II malocclusion with mouth breathing was statistically significant.

Discussion

Mouth breathing as a cause of malocclusion of teeth has been controversial. This is a clinical study to assess the role of mouth breathing on dental occlusion.

A high palatal vault which is commonly believed[14,17] to be associated with mouth breathing was not observed in this study. Palatal height in the experimental and the normal group was not significantly different.

Subtelny[18] believed that a high palate in cases of mouth breathing was probably an optical illusion resulting from contraction of the maxillary arch. The contraction conveys the impression that the vault is much higher than it really is. This investigation also showed a contracted maxillary arch in the experimental group, but this was evident only from the intermolar width. The differences in the normal and experimental groups for intercanine and interpremolar widths were insignificant. According to Brodie,[4] the teeth have a position of equilibrium between two strong muscular components, namely, the buccinator group and the muscles of the tongue. The change in equilibrium due to depressed position of the mandible induces the buccinator muscle to cause a lateral pressure on the maxillary arch. This pressure is more in the molar and premolar regions than the canine area; hence, little change may be expected in the canine area. However, the role of muscles in causing a narrow maxillary arch is not always accepted. While Angle,[1] Brodie,[4] Hawkins,[7] and Moyers[13] agree that muscular imbalance produces a narrow maxillary arch in mouth breathers, Brash,[3] Linder-Aronson and Backstrom[11] hold contrary views.

The maxillary arch length in the mouth breather group was 3.26 mm longer. This difference, which is significant at the 0.1 per cent level may be explained on the basis of the lengthening of the maxillary arch due to contraction of its width.

Mouth breathing does not seem to affect the mandibular arch. Statistically, no significant differences were found in mandibular arch width and arch length in the experimental and the control groups. Angle and Hawkins also could not find any changes in the mandibular arch due to mouth breathing. Angle thought that the tongue, which lies between the lateral halves of the lower arch, resists any dimensional changes in the mandibular arch.

A marked maxillary protrusion often occurs in persons breathing through the mouth. A significant increase of overjet in the experimental group was seen in this study ($P < .001$). The increase in overjet is due to loss of lip tone. The upper lip in mouth breathers is often short and exerts little or no influence on the upper anterior teeth while the lower lip turns outward and rests between the upper and lower arches, further pushing the upper anterior teeth forward.

Along with the excessive overjet, a deep overbite was also noticed in mouth breathers. The difference in mean value was significantly higher ($P < .001$) in the experimental group. Hawkins and Rachel considered the excessive overbite to be due to lack of vertical growth in the molar and premolar regions and to supraocclusion of mandibular incisors.

Mouth breathing is believed to be the primary factor in development of Class II, Division 1 malocclusion. Angle, Huber and Reynolds,[9] and Moyers reported that mouth breathing was associated with all classes of malocclusion. Howard[8] found many mouth breathers with normal occlusion. The occurrence of various types of malocclusion in mouth breathers does not indicate mouth breathing itself as a primary causative factor for Class II, Division 1 malocclusion (Salzmann[16]). To test the validity of their opinion, the experimental sample in the present study was divided according to Angle's classification. It was noted that seventy per cent of the sample had Class II, Division 1, and 26 per cent had Class I. Only two cases were observed in Class II, Division 2. Chi-square test was applied to statistically test the difference between various percentages of Angle's classifications. The value of Chi-square was 16.08 ($P < .005$) which was significant.

Summary

The present investigation regarding dental occlusion was carried out by the dental cast analysis of one hundred male individuals, 15-20 years of age divided equally into mouth breathers and nasal breathers. Various measurements were recorded on the dental casts and Angle's classification of malocclusion was also noted for each case.

From the present study it can be concluded that the effect of mouth breathing was confined to the changes in maxillary arch dimensions. There was contraction of maxillary arch and increase in maxillary arch length. An increased overjet and deep overbite were present in these cases. The palate appeared high, not because its height was actually increased, but due to the contraction of the maxillary arch. A higher percentage of Class II, Division 1 malocclusion was seen in mouth breathers.

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REFERENCES

1. Angle, E. H.: *Treatment of malocclusion of the teeth.* 7th ed., Philadelphia, S.S. White Dental Mfg. Co., 111-117, 1907.
2. Ballard, C. F.: Mouth breathing. *Year Book of Dentistry* 1959-1960 series, p. 166-167. *Proc. Roy. Soc. Med.* 51:282-285, 1958.
3. Brash, J. C.: *The etiology of irregularity and malocclusion of the teeth.* London, Dental Board of U.K., p. 212-226, 1929.
4. Brodie, A. G.: Muscular factors in the diagnosis and treatment of malocclusion. *Angle Orth.* 23:71-77, 1953.
5. Cooke, R. A.: The role of allergy in medico-dental problems. (In Anderson, G. M., ed. *Proceedings of the Dental Centenary celebration,* Baltimore, Waverly Press, 1940. VII. p. 1061) quoted by Hartsook p. 91.
6. Hartsook, J. T.: Mouth breathing as a primary aetiologic factor in production of malocclusion. *J. Dent. Child.* 13:91-94, 1946.
7. Hawkins, A. C.: Mouth breathing as the cause of malocclusion and other facial abnormalities. *Texas Dent. J.* 83:10-15, 1965.
8. Howard, A. J.: Obstructed nasal respiration and its relation to dental abnormalities. *Dental Cosmos.* 50:453-458, 1908.
9. Huber, R. E., and Reynolds, J. W.: A dentofacial study of male students at the University of Michigan in the physical hardening program. *Am. J. Orthodont. and Oral Surg.* 32:1-21, 1946, Quoted by Hartsook, p. 91.
10. Joshi, M. R.: Study of dental occlusion in nasal and pro-nasal breathers in Maharashtrian children. *J. All India D.A.* 36:219-239, 247-249, 1964.
11. Linder-Aronson, Sten and Backstrom, Arne: Comparison between mouth and nose breathers with respect to occlusion and facial dimensions. A biometric study. *Odont. Revy.* 11:343-376, 1961.
12. McCoy, J. D., and Shephard, E. E.: *Applied Orthodontics.* 7th Ed. Lea and Febiger, 1956, pp. 97-99.
13. Moyers, R. E.: *Hand Book of Orthodontics.* The Year Book Inc. Chicago, p. 122, 1958.
14. Neivert, H.: The lymphoid tissue problem in the upper respiratory tract. *Am. J. Orthodont. and Oral Surg.* 25:544-554, 1939.
15. Rachel, Sclare: Habits and the orthodontists. *Dental Magazine and Oral Topics.* 66:9-17, 1949.
16. Salzmann, J. A.: *Orthodontics Principles and Prevention.* Philadelphia and Montreal, J. B. Lippincott, Co., 1957.
17. Shultis, W. K.: Habits, the saboteurs of occlusion. *J.A.D.A.* 15:473-479, 1949.
18. Subtelny, J. D.: Significance of adenoid tissues in orthodontics. *Year Book of Dent.* 1955-1956 series, p. 142-143, 1954.