

Allergy in relation to orofacial dental deformities in children: A review

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Mouth breathing in the infant and child is frequently secondary to chronic nasal allergy. Malocclusion and V-shaped dental arch are commonly found in children who are decided mouth breathers. The anatomic, pathogenetic, prophylactic, and prognostic considerations are discussed.

It is hoped that further investigations will be conducted through the cooperative efforts of the orthodontist, the pediatrician, and the allergist.

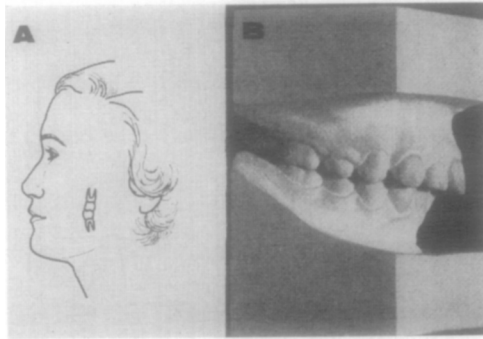
In only one recent text on orthodontics^{1, 2, 3} is allergy considered a factor in the development of malocclusion. Moyers³ mentioned it as one possibility among other causes. A recent article on "preventive orthodontics"⁴ in a pediatric quarterly symposium omitted allergy in the causation of this common childhood deformity. The literature on orthodontics is redundant with references to mouth breathing as a frequent etiologic factor, but the correlation between mouth breathing and chronic nasal allergy of infancy and childhood is rarely spelled out.

CRITIQUE OF LITERATURE

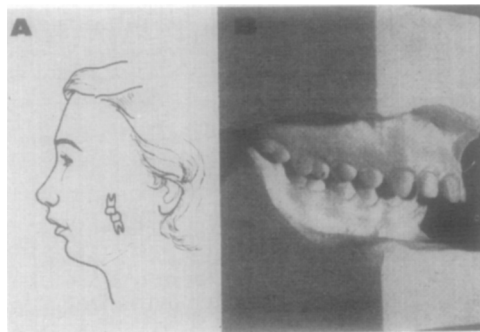
Miller⁵ contended that long-continued respiratory allergy had little to do with the occurrence of malocclusion. He compared two groups of students of both sexes at the University of Michigan for evaluation of dentition, one, a nonallergic, and the other, an allergic group. His major conclusions were: (1) that normal occlusion existed in about two-thirds of both allergic and non-allergic students; (2) that in Class I malocclusion (Angle system of classification) where there is a normal relationship between the maxillary and mandibular arch (Fig. 1), crowding of the maxillary incisors was 4.9 per cent greater in the allergic than in the nonallergic group; (3) that in Class II, Division 1,

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**Fig. 1**

Class I malocclusion (Angle system of classification). *A*, A normal relationship exists between the maxillary and mandibular arch (neutroclusion). *B*, This type of malocclusion develops most likely after the first year of life in children with milder symptoms of chronic nasal allergy and accompanying mouth breathing. (Reproduced, with permission, from Moyers, R. E.: *Handbook of Orthodontics*, Chicago, 1960, Year Book Publishers, Inc.)

**Fig. 2**

Class II, Division 1 malocclusion (Angle system), which includes the greatest number requiring orthodontic treatment. *A*, There is a distal relationship of the mandible to the maxilla (distocclusion). *B*, The maxillary incisors are in extreme labioversion. This condition may be traced to persistent and severe nasal allergy with accompanying mouth breathing occurring from earliest infancy. (Reproduced, with permission, from Moyers, R. E.: *Handbook of Orthodontics*, Chicago, 1960, Year Book Publishers, Inc.)

malocclusion (Angle system), where there is a "distal" relationship of the mandible to the maxilla (Fig. 2) and the maxillary incisors are in extreme labioversion, there was a 5.1 per cent lower incidence in the allergic than in the nonallergic group (this particular division includes by far the greatest number of individuals needing orthodontic care); (4) that palatal height and palatal width were not appreciably different in the nonallergic and the allergic groups.

One may criticize Miller's observations on the basis of the printed questionnaire used by him to determine respiratory allergy in the students. No one experienced in allergy personally interrogated the students. History of past or present mouth breathing, snoring, and clearing or dryness of the throat on awakening was not included. The question of early, repeated tonsil and adenoid infections in childhood was also omitted. No smears for nasal eosinophilia were

made. Many of those students included in the nonallergic group may have had nasal allergy without Miller's cognizance. Often, people are themselves unaware of symptoms of nasal allergy. Freeman and Johnson,⁶ in their study of allergy in adolescent school children, corroborated this fact.

Not all mouth breathing is due to an edematous nasal mucous membrane secondary to chronic allergic rhinitis. In a small percentage of persons, the lips lie more naturally apart than together. This may be a familial, hereditary problem. An inherent coaptation of the tongue and soft palate persists in these "open lip" children and still prevents actual mouth breathing. This can be demonstrated clinically and radiographically.^{7, 8}

Balyeat and Bowen⁹ noted an incidence of protruding maxillary incisor teeth and a grooved, V-shaped palate in about 5 per cent of the child population. In their study, 24 per cent of children had an arch-dental deformity, and, of these, 56 per cent had had their tonsils and adenoids removed early in life. Of 24 children with malocclusion, 8 had parents with the same deformity, 3 of the latter having had chronic nasal allergy since infancy. The authors postulated two etiologic factors for the production of a V-shaped palate and overriding maxillary incisor teeth: (1) heredity and (2) some pathologic condition in infants and children that interfered with normal development of the paranasal sinuses.

Duke¹⁰ held that the depression of the nasal process of the maxilla and flattening of the zygoma in children with chronic nasal allergy was caused by maldevelopment of the ethmoid cells and antra. He inferred that mucus and edematous mucous membranes impeded the entrance of air to the cells, creating a vacuum, and resulting in inadequate expansion and development of these sinuses. Rosenberger¹¹ showed that actual disease of the sinuses in childhood did not interfere with their normal pneumatization, growth, and development. In my own study¹² of allergic "shiners" with facial deformity in 60 children, less than one third had involvement of the nasal sinuses, as shown by roentgenographic studies.

Todd and co-workers¹³ pointed out that, during the first year of life, the head, particularly the brain and face, has an extraordinarily accelerated growth. Since all rapidly growing structures are vulnerable to pathologic change, disturbances of growth in the face may develop from nasal allergy. If perennial allergic rhinitis begins in early infancy, the face may be retarded in all three dimensions. If it begins in the second year, less pronounced deformities occur, especially in the lateral expansion of the hard palate. When symptoms commence at 5 or 6 years of age, only slight diminution in either forward or downward growth will ensue. The study of 60 allergic children by Todd and associates disclosed an incidence of 75 per cent with orthodontic deformity.

Straub¹⁴ studied a group of 104 children under orthodontic treatment, 58 girls and 46 boys. Forty-one (39.4 per cent) of this group were definitely allergic. Thirty-nine were available for classification, according to Angle. Twenty-four (61.5 per cent) had contracted maxillary arches with protruding anterior teeth and retrusion of the mandible. Twenty-two were classified as being in Class II, Division 1 (Fig. 2), and two as Class I (Fig. 1). The latter

two had maxillary arch contraction with protrusion of the anterior teeth. The frequency of 61.5 per cent dentofacial deformities in allergic children in Straub's group of 104 patients was about two and one-half times that reported by Bowen.

ANATOMY AND PATHOGENESIS

The blood supply of the maxillae, nasal cavities, and the paranasal sinuses is chiefly derived from the internal maxillary vessels. The divisions of the internal maxillary artery ramifying in this region are: (1) The anterior superior alveolar branches of the infraorbital artery (a continuation of the internal maxillary) supply the maxillary incisors and canine teeth and the mucous membranes of the antra. (2) The posterior superior alveolar branch descends upon the tuberosity of the maxilla and divides into numerous branches, some of which enter the alveolar canals to supply the molar and premolar teeth and the lining of the maxillary sinus. (3) The descending palatine artery drops downward through the pterygopalatine canal and emerges from the greater palatine foramen running forward in a groove on the medial side of the alveolar border of the hard palate to the incisive canal. The terminal branch of this artery passes upward through this canal to anastomose with the sphenopalatine artery. (4) The sphenopalatine artery passes through the sphenopalatine foramen into the cavity of the nose at the back part of the superior meatus. Here it gives off the posterior lateral nasal branches which spread forward over the conchae and meatuses, anastomosing with the ethmoidal arteries and the nasal branches of the descending palatine, and assist in supplying the frontal, maxillary, ethmoidal, and sphenoidal sinuses. Crossing the undersurface of the sphenoid, the sphenopalatine artery ends on the nasal septum as the posterior septal branches. These anastomose with the ethmoidal arteries and the septal branches of the superior labial (of the external maxillary artery), one branch descending in a groove on the vomer to the incisive canal and anastomosing with the descending palatine artery. (5) The buccinator artery, a branch of the second portion of the internal maxillary artery, supplies the outer surface of the buccinator muscle. The buccinator artery anastomoses with branches of the external maxillary, whose branches supply the upper orbicularis oris fibers. It also anastomoses with the infraorbital artery.

The veins generally follow the branches of the internal maxillary artery, and, from the foregoing, one can see the intimate connection between the vascular supply of the maxillae and the nasal and paranasal cavities. The pterygoid plexus receives venous tributaries corresponding with the branches of the internal maxillary artery. Thus, it receives the sphenopalatine, the superior alveolar, buccinator, and descending palatine veins.

In an earlier communication,¹² I called attention to the etiology of discoloration of the lower orbitopalpebral grooves in allergic children (allergic "shiners"). This is caused by venous stasis from mucosal edema secondary to perennial allergic rhinitis. The same mechanism causing venous stasis may affect alveolar bone and the primary teeth of the maxilla, leading to premature exfoliation or prolonged retention. An abnormal eruptive and resorptive pattern

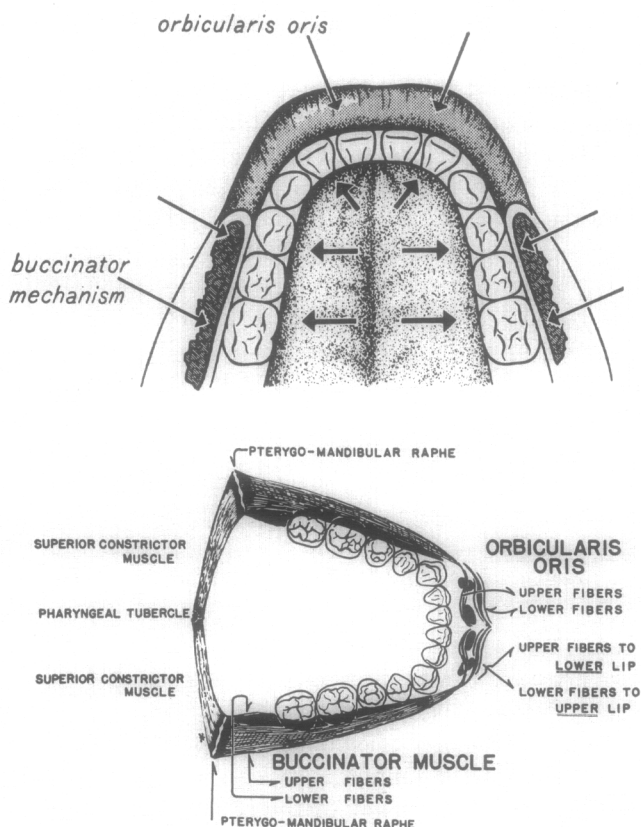
of the primary teeth in the maxilla may affect the normal eruption of the secondary teeth and lead to malocclusion. Rywlin¹⁵ stated that the dental alveolus may be affected by factors stemming from a local acidosis. He expressed the view that "venous puddling" in the maxilla from any cause might bring about local tissue acidosis and interfere with normal growth and development of alveolar bone and teeth.¹⁶ The metabolism of the tissues in this area may be further affected by the action of histamine on the capillaries, resulting in marked dilatation and slowing of the circulation.¹⁷ Thus, it can be seen that venous and capillary stasis in the maxilla, secondary to mucosal edema from long-standing perennial allergic rhinitis, may be a factor in the occurrence of local alveolar acidosis, with resulting arch-dental changes.

Also to be considered in the production of a V-shaped palate and overriding maxillary incisor teeth are the upper decussating fibers of the orbicularis oris and buccinator muscles. These muscles comprise the anterior and lateral components, respectively, of the buccinator mechanism, and, with the tongue, exert pressures on the maxillary dental arch (Fig. 3). The upper portion of the orbicularis oris and the mucous membrane of the upper lip are supplied by the superior labial artery, a branch of the external maxillary. The superior labial also gives off several branches to the nose and septum. There are also widespread anastomoses between this artery, the buccinator, and infraorbital vessels. Venous stasis in the above muscles, as a result of persistently swollen nasal mucous membranes from earliest infancy, can impair their function. Thus, weakness of the orbicularis oris leads to flaccidity and hypotonia of the upper lip, allowing the powerful anterior thrust of the tongue to force the maxillary incisor teeth into a protruded position. Some inefficiency of the muscular action of the buccinator also permits the lateral aspects of the tongue to deflect the premolar and molar teeth toward the cheeks (Fig. 4).

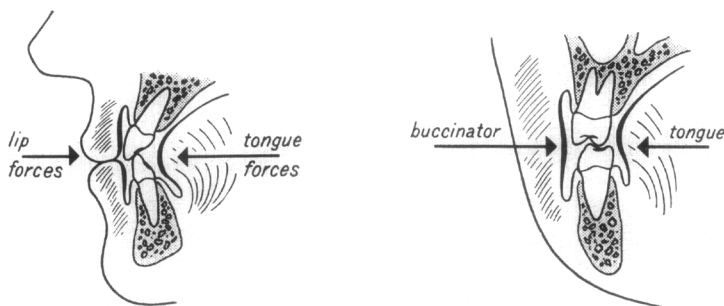
A threefold etiology may be responsible for formation of the V-shaped dental arch: (1) local metabolic tissue changes in alveolar and palatal bone from venous and capillary stasis; (2) contact and repeated pressures of the high-lying, arched tongue against the hard palate in suckling infants (slow-motion films have shown that a rubber teat placed on the dorsum of the tongue is repeatedly compressed against the hard palate by successive waves of contraction in the transverse fibers of the tongue)¹⁸; (3) residual tonus in the buccinator, which contracts upon an already weakened arch.

COMMENT

Most orthodontists include mouth breathing as a major contributing factor in the development of dentofacial deformities. Moyers³ stated that mouth breathers have an unusually high incidence of malocclusion. He stated further that mouth breathing involves contraction of the maxillary denture, labioversion of the maxillary anterior teeth, crowding of the anterior teeth in both arches, hypertrophy of the lower lip, hypotonicity and apparent shortening of the maxillary lip, and frequently marked overbite. Mouth breathing, he believed, is one symptom of disturbed muscle balance and is associated with many factors not conducive to normal facial and occlusal development.

**Fig. 3**

The buccinator mechanism. The upper decussating fibers of the orbicularis oris and buccinator muscles together with the tongue exert balanced molding pressures on the maxillary dental arch. (Reproduced, with permission, from Graber, T. M.: *Orthodontics: Principles and Practice*, Philadelphia, 1961, W. B. Saunders Company.)

**Fig. 4**

Protruding maxillary incisor, premolar, and molar teeth. The powerful anterior and lateral thrust of the tongue against the weakened fibers of the orbicularis oris and buccinator muscles leads to this condition. (Reproduced, with permission, from Graber, T. M.: *Orthodontics: Principles and Practice*, Philadelphia, 1961, W. B. Saunders Company.)

Allergic edema of the mucosa of the nasal cavity and paranasal sinuses exerts a major role in mouth breathing.^{18, 19} Mouth breathing secondary to chronic nasal allergy in early infancy often leads to severe dental and orofacial deformities. Some newborn infants are already confirmed mouth breathers in the first few days of life. These infants have feeding difficulties and often need formula substitutions. Other neonates, who are apparent mouth breathers, possess a good posterior seal between the soft palate and the tongue, allowing for free nasal breathing; thus, no interference with suckling occurs.¹⁸

Clein²⁰ cited a 13 per cent incidence of allergic rhinitis in infancy. The 206 infants studied by him were chiefly allergic to cow's milk. Cole,²¹ in a survey of 200 children with allergic rhinitis, stated that 10 per cent had the onset of their symptoms under 1 year of age and 74 per cent at the age of 3 or under. In 92 per cent the onset was at the age of 6 or under. Freeman and Johnson⁶ reported an over-all 6.4 per cent incidence of perennial allergic rhinitis in adolescent school children and a 25 per cent incidence in the allergic group. Nonseasonal rhinitis was a more frequent complaint among younger allergic school children than among older ones. Unlike other workers, however, they found allergic rhinitis to develop more frequently after the age of 5 than before.

Sensitization of nasal mucous membranes from early feeding of cow's milk gives rise to persistent edema. Cow's milk in the neonatal period may be the priming, sensitizing factor which propels the allergic infant toward progressively severe allergic manifestations, one of which may be dentofacial deformity. One should strongly consider Glaser's^{22, 23} concept of prophylactic avoidance of cow's milk for potentially allergic newborn infants. Allergically edematous nasal and paranasal mucous membranes cause venous stasis in the maxillary dental arch and surrounding structures from pressure on veins. In addition, the action of histamine causes marked dilatation of the capillary bed¹⁷ in the affected nasal, palatal, and alveolar areas. As Rywlin^{15, 16} pointed out, venous and capillary stasis in alveolar bone may result in local tissue acidosis, leading to dental orofacial disorders.

Primitive peoples were said to be comparatively free from allergic diseases. Balyeat and Bowen⁹ remarked on the paucity of allergic manifestations and dentofacial abnormalities in Oklahoma Indians. Sincock²⁴ stated that he had never seen Chippewa Indians with hay fever, asthma, or eczema. He attributed these findings to Indian babies' being breast-fed for fully nine months or longer without supplemental foods or water. When early feeding of cow's milk and adult foods was introduced, eczema and other allergic manifestations appeared.

Wallis²⁵ noted the low incidence of malocclusion in collections of skulls 400 to 500 years old. He believed that mouth breathing and abnormal occlusion of the teeth led to a high infant mortality. Ingestion of natural foods, breast feeding, pollutant-free atmospheres, and the like were not considered by Wallis. These factors may have been responsible for the rarity of abnormal occlusion in the ancient skulls. Ballard²⁶ found a correlation between lips-apart posture, malocclusion, and the incidence of asthma and recurrent upper respiratory tract infections. He thought, also, that there was an association between this



Fig. 5A

Four-year-old boy with perennial allergic rhinitis, mouth breathing, V-shaped palate, and pectus excavatum. Has been a mouth breather since birth.



Fig. 5B

Four-year-old boy with V-shaped palate.

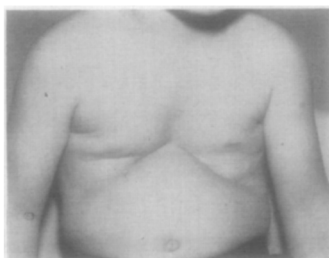


Fig. 5C

Four-year-old with pectus excavatum. Pigeon breast may be associated with mouth breathing.

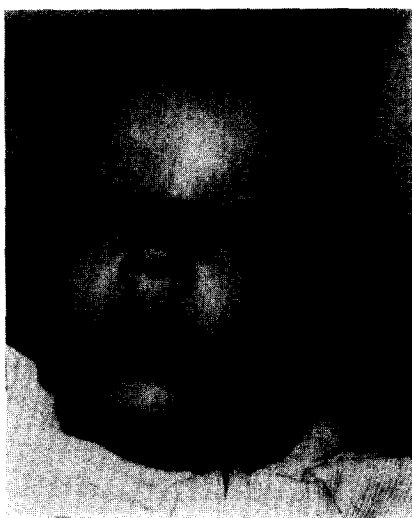


Fig. 6

Three-month-old male infant with severe nasal allergy. Markedly sensitive to cow's milk. Has been a mouth breather since birth.

type of facial morphology and congenital defect of the chest wall, particularly in ectomorphic persons (Fig. 5).

Persistent mouth breathing in the neonatal and postnatal periods is uncommon. Generally, the open-mouth habit in infancy and childhood should be viewed as an expression of nasal allergy (Fig. 6).

PROGNOSTIC IMPLICATIONS

Although mouth breathing is usually self-corrected before adolescence, the patient with chronic nasal allergy already may have malocclusion of the teeth, with orofacial deformities. Mouth breathing in the infant and child should be regarded as an allergic manifestation until proved otherwise. Early diagnosis and early efficient modern allergy management can often forestall at least one major factor contributing to the progressive effects of dentofacial deformity.

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1680 MERIDIAN AVENUE

ERRATUM

In the article, "Pollen and mold survey in Hawaii, July, 1963, to June, 1964" by Alexander Roth, in the March-April issue of the *JOURNAL*, page 186, in Table I the total pollen and mold count as given refers to cubic meters rather than cubic millimeters as stated.