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Tutorial

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The Effect Of Airway Interference On The Growth And Development Of The Face, Jaws, And Dentition

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INTRODUCTION:

Two criteria of great importance for the well-being of most humans are good health and appearance. This paper is an attempt to examine the effect of allergies on both those criteria as far as the growth and development of the face and dentition are concerned.

A review of the literature shows that allergies are responsible for nasorespiratory blockage either through congestion of the nasal mucous membranes or through the hypertrophy of tonsils and adenoids. Several articles report the incidence and age of onset of these phenomena.

The development of immunocompetent lymphoid tissue in the nasopharynx is correlated with the growth of the nasopharygeal area. An imbalance in the equilibrium of differential growth of these organs can result in mouth breathing.

This paper will examine the consequences of mouth breathing as documented in studies performed on experimental animals, and as seen in clinical examinations of children. The review of the literature will also show that upon treatment of the allergic condition or surgical removal of the excessive tonsil and adenoid tissues it was found that the restoration of nasal breathing reduced the malformations present and resulted in a tendency towards normalization of health and appearance.

This paper, condensed and limited to those portions dealing with the effects of nasorespiratory blockage in facial development, presents a review of the literature available on the topic. Originally, it was presented in the People's Republic of China, in the Fall of 1981. It was titled: *The Effects of Allergies and Nasorespiratory Blockage on the Development of the Face and Dentition.* Five physicians and an orthodontist participated in a cultural exchange program on *Allergy and Immunology*, the purpose of which was to trade information and ideas with their counterparts in a variety of settings such as schools, hospitals, clinics, and several different types of medical facilities in communes throughout China.

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REVIEW OF THE LITERATURE:

A relationship between the development of the face and nasorespiratory blockage has long been suspected. In 1861 George Catlin,[15] renowned for his American Indian paintings and contributions to ethnography, published a classic text on mouth breathing. The title of this work was "The Breath of Life, or Malrespiration and Its Effects Upon the Enjoyment and Life of Man." The premise of his book was that primitive Indians were healthier than civilized Europeans because they slept on their backs and breathed through their noses. This book was reprinted regularly for thirty years and sold approximately 40,000 copies. Catlin felt that the bad habit of mouth breathing caused nightmares, snoring, nervous debility, yellow fever, cholera, infant death, deafness, crooked teeth, colds, and dreary faces. While some of his theories strike us as ridiculous today, parts of his observations have been proven correct as this paper will show through a review of the additional literature.

Essentially, allergies are frequently responsible for nasorespiratory blockage either through congestion of the nasal mucous membranes or the hypertrophy of tonsils and adenoids. The development of immunocompetent lymphoid tissue in the pharynx is correlated with the growth of the nasopharyngeal area. An imbalance in the equilibrium of the differential growth of these organs can result in mouth breathing. The following sequellae are then observed:

1. The child develops a facial appearance described as gaping or "adenoidal facies."
2. The lower face height is increased.
3. The mandibular angle is increased and the gonial angle becomes more open.
4. The tongue position is lowered.

5. A tongue thrust swallowing pattern is often retained from infancy.
6. There seems to be a smaller saggital depth to the maxillary and mandibular complex.
7. The occlusion is characterized by narrow upper arches, frequent cross-bites and anterior open-bites.
8. An alteration of head posture is noted.
9. A variety of other facial and oral disorders are in evidence. Among these are: edematous bags and dark circles under the lower eyelids; marginal eyelid eczema and infected conjunctivas; gingival hyperplasia; occasionally, very severe geographic tongue; and nocturnal bruxism.

It has been estimated that obstructions of varying degrees of severity in the nasal cavities exist in 85% of all children. These obstructions may be due to the enlargement of the turbinates as well as the result of air-borne irritants or allergens, marked deviation of the nasal septum, or adenoid hypertrophy. Mouth breathing in infancy often leads to severe dental and orofacial deformities.

In a study of 500 children Leech[9] reported that of the 19 percent (or 95 children) classified as mouth breathers, 13 percent (65 children) had obstructive adenoids and 6 percent (30 children) had allergic and infective rhinitis, nasal defects or other disorders.

Cooke[4] observed that nasal blockage was a frequent cause of malocclusion. He placed particular stress on the hypertrophied tonsils and adenoids, and on the obstruction of airway passages in children between the ages of two and eight. He claimed that changes in the nasal membranes due to allergies are not common before the sixth year. They are, however, the most frequent cause of deformities encountered by the orthodontist. He felt that nasal blockage should be treated early in life to prevent the development of malocclusion.

In a sample of 41 patients with chronic nasal allergy, Straub[17] classified 22 patients as Class II, division 1 with maxillary constriction; two as Class I, also showing maxillary constriction with accompanying protrusion of the anterior teeth and retraction of the mandible or mandibular teeth. The remainder were classified into thirteen Class I; one Class II, division 2 and one Class III. In other words, malocclusion of some form or another were present.

Bowen[1] studied a group of 500 children between the ages of six and fourteen years. They were divided into subgroups of 100 each as follows: Indians, Caucasians, Negro children, crippled children in hospitals and finally children suffering from chronic nasal allergy. In the first four groups he found four to seven percent had dental and palatal deformities attributable to a variety of causes such as genetic condition, presence of adenoid tissue, detrimental thumb sucking and sleeping or learning habits. In the fifth group, the maxillary deformities were found to be in excess of twenty-four percent. The statistical significance of this study is questionable based on the sample selection which obviously skews the data.

Cole[3], in a survey of 200 children with allergic rhinitis, stated that ten percent showed onset of their symptoms under one year of age and seventy-four percent at the age of three or under. In ninety-two percent the onset was at age six or under.

Clein[2]cited a very similar figure of thirteen percent incidence of allergic rhinitis in infancy. The 206 infants he studied were chiefly allergic to cow's milk.

According to Todd[20] the deficiency of facial growth is apparent in the first year and allergic sensitivity most clearly evident in disturbances of the gastrointestinal tract. In the third year he described disturbances with high fever, beginning of nasal turbinate congestion, morning sneezing, and nasal discharges. He states that this occlusion of nasal passages results in a narrow, pinched nose and contraction of the dental arch. His findings would suggest that adverse effects of allergic conditions manifest themselves much earlier than the six years of age suggested by Cooke[4]. Todd postulates that since growth of the head takes place at an accelerated rate during the first year of life, the growth and development of the face will be most vulnerable to pathologic changes at that particular stage of life. If allergic rhinitis begins in infancy, growth of the face may be retarded in all three dimensions. If it begins in the second year, less pronounced deformities take place, especially in the lateral expansion of the hard palate. When onset of the symptoms occurs at five or six years of age, only a slight forward or downward growth will ensue. The study of sixty allergeric children by Todd and associates disclosed an incidence of seventy-five percent of children with orthodontic deformities.

The primary biological function of the nasopharynx is to provide passage for airflow from the nasal cavity to the oral pharynx, laryngeal pharynx, and eventually to the lungs. The nasophyarynx also provides space on its posterior and superior walls for the lymphoid tissue complex referred to as Waldeyer's ring. The enlargement of the adenoids may lead to partial or to total blockage of the nasopharyngeal pas-

sage making nasal respiration difficult or impossible. The potential disharmony of the adenoid tissue may be due to the different growth pattern of the bony nasopharynx and the tonsils per se. The shape and size of the nasopharyngeal cavity can be defined in terms of depth and height in the medium sagittal plane, and width in the frontal plane.

King[8] stated that the total depth of the nasopharynx is established in the first or second year of life. Increases in the height continue until maturity and are accounted for by the descent of the hard palate and cervical vertebrae from the cranium. The increases in width were registered as taking place after six years with acceleration at adolescence. In comparison to pharyngeal growth, the adenoids were first evident on lateral cephalometric radiographs by six months to one year. They became abundant, occupying about one-half of the nasopharyngeal cavity, by two to three years of life. Thereafter, the lymphoid mass continued to grow, peaking in size as early in life as nine to ten years, or as late as fourteen to fifteen years. It then atrophies progressively as the nasopharyngeal airway space increases greatly. In most instances growth of the adenoids and of the nasopharynx are in a state of equilibrium, and the developmental pattern just described is fairly well established. It is the differential timing of growth as well as the differential quantities of growth of all the organs involved that affect dentofacial development. Occasionally the equilibrium is disturbed and considerable variations are seen from age to age. It is speculated that these deviations are related to individual responses to the stresses of nasorespiratory infections and allergies.

The adenoid tissue is attached to the roof of the nasopharynx and may extend as far forward as the posterior nasal choanae. At times it may be seen to obstruct, in a vertical relationship, a major portion of the posterior nasal choanae. It may even extend as far downward as the nasal surface of the soft palate, in which case the respiratory passages are abnormally reduced. When airflow is thus impeded, oral rather than nasal respiration becomes the mechanism of survival.

If the growth of the nasomaxillary complex is related to functional response to nasorespiratory intake, the anatomy of the nasal cavities should be examined as potential contributors to developmental problems. Frontal cephalometric radiographs should be checked, not only for width of the nasal cavities, but also for the shape and size of the nasal septum and the degree of hypertrophy of the turbinates located on the lateral walls of the nasal cavities. These turbinates are lined with respiratory mucosa which becomes chronically swollen incident to inflammatory and allergic stresses. Therefore, the turbinates, particularly the inferior turbinates, when observed to approximate the nasal septum can mechanically impede airflow. A greater incidence of deviated nasal septum has been recorded in the allergic than in the nonallergic child. Little is known about the growth of the turbinates or the effect of enlarged turbinates on maxillary growth itself, but it seems reasonable, in view of the deformities attributed to mouth breathing, to believe that a viable nasal respiratory system is necessary for propitious development of both nasomaxillary and mandibular complexes.

Concurrent with the necessity for mouth breathing, changes in facial development were noted by several investigators. Clinical observations suggest a close association between nasal obstruction, mouth breathing, facial maldevelopment, and dental malocclusion, but a direct cause and effect relationship had not been established until Harvold's[7] primate experiments on oral respiration.

In this experiment, forty-two Rhesus monkeys were used. Half the animals were used as controls, and the other half had their noses obstructed with silicone plugs which induced mouth breathing and were left in place for three years. Before and after the experiment the data recorded were analyzed by standard statistical methods for intra-pair and group differences. It was found that all mouth breathing animals developed a notched upper lip; the tongue shape was altered to secure an oral airway; and the continuous forward positioning of the tongue produced a long, slender tongue with a median groove.

The changes in face height and alterations in mandibular morphology were most extensive in those animals that acquired a low postural position of the mandible. This lowering of the mandible was followed by a downward displacement of the maxilla, and also by an increased extrusion of the teeth.

The lowering of the chin for oral respiration gradually resulted in a steeper mandibular plane and a more open gonial angle. These changes in mandibular growth were dependent on the altered activity in the facial and cervical musculature. All of the experimental group gradually developed some type of malocclusion, usually dependent on mandibular and lingual position.

Clinical observations in the human population closely parallel the results which were described in these experimental animals, Subtelny[18, 19] states that

while mouth breathing the mandible is depressed, and that with the forward posture of the tongue to maintain an oropharyngeal airway passage, more vertical facial growth may follow. He conjectures that continued eruption of the posterior teeth is instrumental in increasing anterior lower face height, and also the potential for an anterior open bite.

Examination of the nasomaxillary complex on cephalometric head plates confirmed a clinical observation that the hard palate tends to tip down in the posterior region, away from the cranial base. This could be measured and was therefore not an optical illusion related to the posturing of the while head in an upward and forward direction away from the cervical column. This head posture is a characteristic common to mouth breathers.

In a different study conducted by Ricketts[16], subjects with excessive adenoid masses showed a posterior vertical height of the nasomaxillary complex that was significantly greater than was found in the control subjects. The mandibular plane was steeper, diverging more as it progressed from the gonial region to the symphysis. In addition there was a greater divergence of the lower border of the mandible relative to the ramus which added to the comparatively greater anterior lower facial height. A further clinical observation showed some degree of maxillary retrusion and reduction of maxillary width. Upper face height was not affected.

Ricketts gave us the following description: "A patient would display cross-bite; tonsils and adenoids would be present; an open-bite would be exhibited; the tongue would thrust on swallowing. If the maxillae were constricted bilaterally, the mandible would be deflected to one side in a functional cross-bite. If the constriction were more severe, the patient would reveal a bilateral cross-bite with no lateral mandibular deflection, but a forward deflection would be observed. Concomitant with the narrow U-shaped maxillary arch, the upper incisors were usually procumbent and and labially flared. This is attributed to the reduced pressure from a short upper lip, deficient in muscular tonus on one side, and to the proclining force of the lower lip against the lingual surfaces of the upper incisors from the other side. The occlusion most commonly seen was a Class II in which the maxillary first permanent molars are anterior to the lower ones."

Linder-Aronson[10, 11, 12] made extensive studies on respiratory function in relation to facial morphology and the dentition. He compared a sample of eighty-one mouth breathing patients with hypertrophic adenoids with a similar number of control cases of the same age and sex but without nasal obstructions. After adenoidectomies were performed, he followed up his patient sample for a period of five years. Most of his findings corroborated those previously described; however, a few discrepancies appeared and should be mentioned. Agreement was seen on the following points: twenty-six percent of the mouth breathing population presented the adenoid facies appearance already described as compared to four percent of the control group. Increases in both the lower and total face height were recorded. The sagittal depth of the nasopharynx was less, and the tongue position was lower. In addition, the upper jaw was narrow, crossbites were frequent, and so were open-bites.

In disagreement with previous investigation, Linder-Aronson reports retroclined upper and lower incisors (often crowded), normal palatal heights, and normal antero-posterior relationship between the upper and lower jaws.

Upon postoperative resumption of nasal breathing, a reversal of some of these malformations in Linder-Aronson's experimental group were seen over the interval of five years. The inclination of both upper and lower incisors became normal. The arch width, as measured between the upper first molars, increased by an average of nine millimeters as compared to five millimeters in the control group during the first postoperative year. A normalization of the depth of the nasopharynx also took place during this year as the result of the alteration from mouth to nose breathing.

The normalization of the mandibular inclination took place throughout the five-year observation period, and as a consequence lower face height was decreased. The alteration of tongue position has already been described, and the term "tongue thrust" introduced. This concept deserves elaboration. Tongue thrusting is an infantile swallowing reflex normally present at birth. As the baby develops, this tongue protrusion, sucking type of swallowing reflex changes, and with the eruption of teeth a mature somatic swallow results. However, when this sucking swallow is retained, and does not progress normally to the mature swallow, this is considered abnormal behavior.

A number of explanations have been offered concerning the tongue thrust behavior when it is seen later in life. Hanson and Cohen[6] suggest that any of the following factors or a combination thereof are potential factors in the persistence of tongue-thrust swallow throughout the mixed dentition: mouth breathing, digit sucking, enlarged tonsils, allergic rhinitis, a high or narrow palatal arch, and anterior malocclusion in the deciduous dentition. Eighty-five percent of habitual mouth breathers show underdevelop-

ment of the oral musculature and varying degrees of distocclusion. A declining incidence of mouth breathing parallels a decline in tongue thrust. Practically every individual with a stubborn tongue thrust exhibits a breathing problem of some form. The importance of this can be appreciated when one reads Graber's[5] description of the sequellae of this habit. With constant thrusting, the tongue drops lower in the mouth and no longer approximates the palate. It elongates as it thrusts forward, and if an anterior openbite is already present, the tongue will thrust between the incisors to create an oral seal. Since the tongue force is greater than the opposing lip force, the incisors are usually pushed further labially. With changes in tongue, cheek, and lip function, the net effect is a significant narrowing of the maxillary arch and overeruption of the posterior teeth resulting in increased lower face height. A cross-bite is frequently created by the narrowing of the maxillary arch, and a convenience swing or mandibular displacement results in a lateral deflection of the lower jaw. Unless normal activity can be restored and a mature somatic swallowing habit achieved, the malocclusion will perpetuate itself and be aggravated until a balance is established with hereditary pattern, basal bone limits, tooth size, and the functions of respiration, speech, mastication, deglutition and posture.

This description is analogous to that of the sequellae of mouth breathing. Since they both occur so frequently in conjunction with each other, one could speculate on the extent of the interrelationship, and of the cause-effect of the factors involved. It is clear, however, that whether we are dealing with a chronic allergy, an adenoid or tonsillar nasopharyngeal obstruction, or habitual tongue thrust, the correction of dental deformity will be doomed to failure unless these conditions are recognized and treated first.

The malformations resulting from mouth breathing have usually been attributed to the interplay of altered muscle function with concomittantly deformed skeletal structures. The debate between investigators continues as to whether form affects function or viceversa. It would seem reasonable to consider them as interrelated and affecting each other, dependent on the stage of development. At present an interesting concept has been put forward relative to the mechanism of growth of the craniofacial structures called the "functional matrix theory." In essence, it is hypothesized that craniofacial growth is closely associated with the functional activities carried out by different components of the head and neck area and soft tissues involved in these functions. Respiration is one such functional element while mastication and deglutition would be examples of other such functional activities. Considering this doctrine of functional matrices, if there are obstructions in the nasal and oropharyngeal respiratory areas, some influence may be exerted on the direction of growth of the skeletal structures of the face incident to the need for continued reposturing of soft tissues and skeletal structures.

Marks[13, 14] takes a totally different approach in his interpretation. He relates the deficiencies in development to the metabolism of tissues as they are affected by circulatory changes. Neuromuscular and circulatory imbalances occur between the buccinator, orbicularis oris, and the tongue. Most of the blood vessels supplying the palate, the dental arch, and the perioral musculature anastomose with the vessels supplying the nasal cavities and the infraorbital area. Venous stasis in these muscles, as a result of persistently swollen nasal mucous membranes from infancy, can impair their function. Thus, for example, weakness in the orbicularis oris leads to flaccidity and lack of tonus of the upper lip, allowing anterior displacement of the incisor teeth by the tongue. This theory could be disputed, since it is the strength, not the weakness of the buccinator muscles which would be responsible for the narrowing of the maxilla as the tongue, due to its lowered position, is no longer able to maintain palatal arch width. Therefore the theory of muscle weakness due to venous stasis deserves further investigation.

While the main aspects of deformations resulting from allergies and excess lymphoid tissue have been discussed, a few more disturbances are noted which deserve brief attention. Bruxism or tooth grinding is an oral habit indulged in by many adults and children. Dentists usually attribute bruxism to psychogenic factors, traumatic occlusion, and hypertonicity of the muscles of mastication concurrent with temporomandibular joint disturbances. However, in a study conducted in the pediatric and pediatric allergy clinics at Jackson Memorial Hospital in Miami, Florida, the incidence of tooth grinding in non-allergic children was found to be twenty percent while in a similar group of allergic children matched by age, sex, and color, the bruxism incidence was sixty percent. There is no clear-cut evidence to explain this. One possible cause might be that malocclusion, which is prevalent in allergic children, promotes traumatic occlusion. It seems more likely, however, that the disturbance would originate from the middle ear. Electro-acoustic impedance bridge studies have shown a high incidence of increased negative pressures in the middle ear cavities with a concomitant auditory tubal dysfunction in allergic bruxists. Repetitive nocturnal bruxism may pos-

sibly be initiated as a reflex action by increased negative or disturbed pressures in the tympanic cavities from allergic edema of the mucosa of the Eustachian tubes.

That allergy plays a definite role in nocturnal bruxism is evidenced during exacerbation of the perennial allergic rhinitis, asthma attacks, upper respiratory tract infections and excessive exposure to allergens, which induce more tooth grinding. Some parents can correlate an increase in bruxism with ingestions of certain foods of highly allergenic potential. Conversely, when modern allergy management is begun early, emphasizing strict environmental and dietary control measures along with immunotherapy when indicated, most parents noted that nocturnal bruxism diminished or ceased completely.

A minor allergic manifestation associated with food allergy is geographic tongue. Shiny circular or oval patches with white elevated margins appear on the dorsum, edges, or tip of the tongue. These patches migrate, disappear, then re-appear. They are due to a thickening of the filliform papillae and hypertrophy of the epithelium surrounding areas of desquamation. Since these lesions are asymptomatic, no treatment is indicated.

Persistent mouth breathing has been reported as leading to development of gingival hyperplasia. This condition can become so severe as to resemble the hyperplasia seen with long term medication with dilatin (phenytoin sodium). The primary lesion is marginal, with interdental swelling of the gingiva around the incisors, and to a lesser degree, the premolars and molars. The hyperplasia may be caused by circulatory disturbances, sensitization from inhalents, exposure to tobacco, smoke, food, and food additives. Tooth displacement may occur. A blood sample taken from the gingival tissues of allergic patients will show an eosinophil count as high as fifteen to forty-five percent.

CONCLUSION:

The sequellae of airway interferences have been reviewed. It was shown that the removal of these interferences tended to promote a reversal of symptoms towards normalization of the developmental pattern of the individual. The review of the literature also reveals the interdependance of the various responses exhibited by the different anatomical systems involved. Research has documented many correlations among the various symptoms, but very few direct cause and effect relationships. It is also impressive to consider the number of allied professional fields involved in the treatment of these problems, and the need for more communication and team work between the disciplines involved, to better benefit the patient.

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