The long face syndrome and impairment of the nasopharyngeal airway

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The switch from a nasal to an oronasal breathing pattern induces functional adaptations[1] that have — for at least a century — been associated with a deviant craniofacial growth pattern. The typical dental and morphological characteristics associated with nasal impairment have been reviewed in detail elsewhere[2] and can be summarized as follows: an increase in total anterior face height[3,4,6,7] which is mostly contributed to by a more vertical development of the lower anterior face.[4,5,7,11] Concomitantly, an increase in the mandibular plane[3-7] and gonial angles[6] and a tipping of the palate[3,4,6,8-10] can be found. Facial prognathism is reported to be decreased.[3,5,6,12] The typical physionomy of the so-called "adenoid facies" is depicted in Fig. 1.

The features described bear a remarkable resemblance to those reported as typical for an extreme vertical growth pattern resulting in the long face syndrome[2,13] (Fig. 2) and as a consequence the associations found in the clinical studies mentioned above have prompted the hypothesis that mouthbreathing must be regarded as a major etiological factor in inducing excessive vertical growth.

Historically, several mechanisms have been suggested to act on the development of the maxilla and the other skeletal structures during impaired nasal breathing, e.g. atrophy of the nasal cavity by inactivity,[14,15] upward direction of the airstream on the palate[16,17] and raised negative pressure in the nasal cavity.[18,19] The prevailing belief today is that alteration in postural muscle activity influences the position of the teeth as well as the growth behavior of some craniofacial structures.[3,20,21] An alternative contemporary hypothesis suggests that a soft tissue stretching mechanism elicits a morphogenetic response leading to the same results.[22]

However, considerable controversy exists about this form-function relationship and throughout orthodontic history numerous reports have denied this association.[23-29]

The conflicting opinions can be summarized as follows: In one school of thought mouth-

Abstract

Experimental evidence suggests that altered muscular function can influence craniofacial morphology. The switch from a nasal to an oronasal breathing pattern induces functional adaptations that include an increase in total anterior face height and vertical development of the lower anterior face. While some animal studies have suggested predictable growth patterns may occur, studies in human subjects have been much more controversial. Therefore, individual variations in response should be expected from the alteration of a long face syndrome patient's breathing mode.

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Key Words

Airway • Lower anterior face height • Long face syndrome

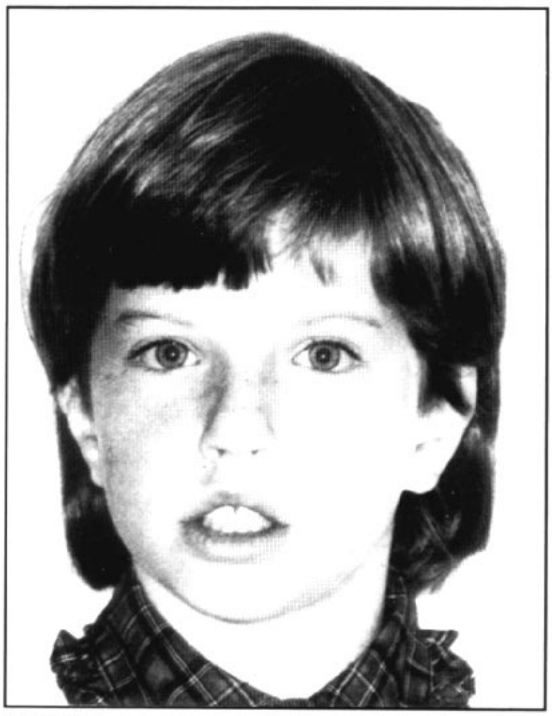

Figure 1
Typical facial appearance of the "adenoid facies." Note the long narrow face, narrow nose and nasal apertures, short upper lip and mouth-open posture.[53]

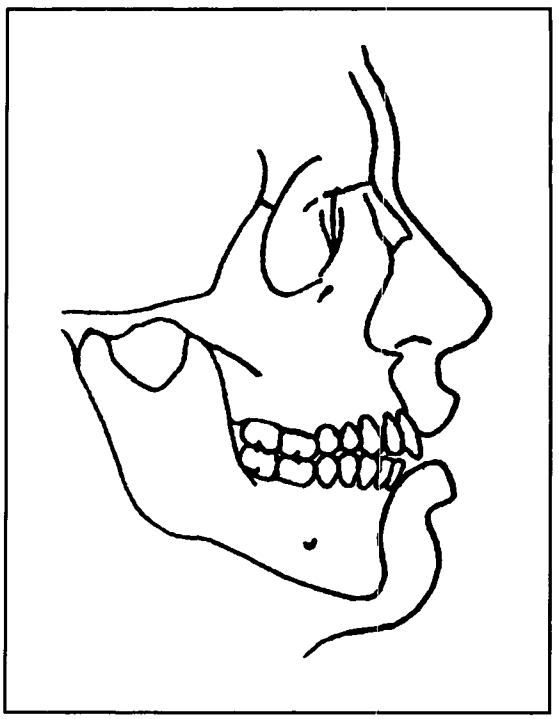

Figure 2
Typical skeletal appearance of LFS patient. Note the increase in lower anterior face height, gonial angle and mandibular plane angle. The skeletal pattern and facial physionomy resemble that of the adenoid face.

breathing has been designated as an important etiologic factor for the production of the LFS. The contrary opinion holds that the typical features described are the expression of a hereditary pattern (somatotype) and that mouthbreathing is unrelated as an etiological factor or only a paraphenomenon in a narrow type of face and can at most be a precipitating (aggravating) factor.[24] (The sagittal dimension of the nasopharynx has been found indeed to be diminished in the LFS.[1])

The controversy still remains today[30] and as a consequence, considerable debate exists about the warranty of taking preventive measures to secure a patent airway[31-33] and about the treatment of impaired nasal function as an inherent part of the orthodontic therapy program.[8,34-42]

Clinical evidence

Most clinical studies dealing with the subject compare the craniofacial morphology of a mouthbreathing sample with that of nosebreathers.[4,12,15,22a,29,47,49,50] However, cross sectional studies, by their very methodology, cannot prove any cause-and-effect relationship between the factors involved. At most, they can show possible associations between certain skeletal features and the prevalence of nasopharyngeal obstruction. As a consequence, they cannot give credibility to one or the other hypothesis. A longitudinal study in controlled circumstances between an experimental group in which an environmental factor (nasal airway capacity) is altered and a matched control group can shed more light on the etiology of mouthbreathing.

In that respect, Linder-Aronson's follow-up studies[20,43] may be called unique. The adenoidectomy group of his earlier investigation[3] has been reexamined one year[20] and five years [43,43a] postoperatively. The experimental group that switched to nosebreathing was matched with a control nosebreathing group, also from his previous study. Mode of respiration was assessed by visual examination and nasal airflow measurement.[43a]

The greatest changes occurred in the incisor region during the first postoperative year and in the other variables (inclination of mandibular and nasal lines relative to sella-nasion, sagittal depth of the bony nasopharynx (pm-ba) and upper molar width) between the second and the fifth years. Significant group mean differences found initially were diminished and point to a normalization of the dentition after adenoidectomy. The author hypothesizes that the changes observed are due to an altered tongue position and a change in activity of the orofacial and pharyngeal muscles.[20] However, the measured changes, although statistically significant, are small numerically and their biological significance has been questioned.[32]

In the hypothetical assumption that the variables in a mouthbreather would not change at all during the following five-year period, a correction of the oral breathing pattern would result in an increase of 2.2 millimeters of intermolar width and a decrease of four degrees in mandibular plane angle over that time span.

The same longitudinal experimental and con-

trol samples were used to assess mandibular growth direction (MGD) during the five postoperative years.[43a] Relative to the control group, a significantly more horizontal MGD was found for female but not for male patients. Another significant finding was increased variability in the postoperative mandibular growth patterns, indicating variation in individual susceptibility to environmental functional factors. Because no data were available on MGD during the preadenoidectomy period, no scientific proof of an effective longitudinal change in MGD could be estalished.

McNamara,[45] also reported an effect on LAFH and mandibular plane angle after relief of nasal obstruction. However, this study did not use a control sample or any statistical methodology to assess the relevancy of the findings.

In summary, it can be stated that the mode of breathing has limited impact on the dentition and skeletal morphology. However, individual variation is present. The observed average changes are so small they hardly lend credibility to the hypothesis that an extreme skeletal pattern as observed in the LFS would be due in large part to an impairment of the nasal airway.

In search of objective criteria

The different sample selection criteria and diagnostic methodology used in clinical research may also contribute to the conflicting results published. Additionally, it makes a reliable interpretation of the data difficult. Some studies only differentiated their samples on the presence of adenoid vegetation[9,46,47] or the occurrence of allergy[4,5,48] without an attempt to assess the mode of breathing. Others used questionnaires,[29,48,49] a wisp of down or cotton[49,50] or vapor condensation on a tongue depressor[49] as a diagnostic tool. Visual examination of the patient's mode of breathing[3,6,7,11,20,51,52] is still an often used method. It is obvious that the diagnosis of oral breathing is in need of a more scientific (quantitative) approach.

The assessment of nasopharyngeal capacity faces the same methodologic problems. The nasal cavity or nasopharyngeal area is routinely visualized by anterior or posterior rhinoscopy respectively.[53] This evaluation only mentions the subjective impression of airway impairment[3,52,54] or expresses it on an index scale basis.[54] Moreover, posterior rhinoscopy may be difficult to perform in children.[55]

Goldman and Brachman,[56] Khoo and Nalpon[57] and Captitonio and Kirkpatric[58] recommended roentgenographic study of the nasopharynx as an aid in determining the size, shape and position of enlarged adenoids and the amount of obstruction caused. The same method was also advocated and/or used by Ricketts,[58] Subtelny,[8,59] Hibbert,[60] Linder-Aronson[3,54,61] and Preston.[62]

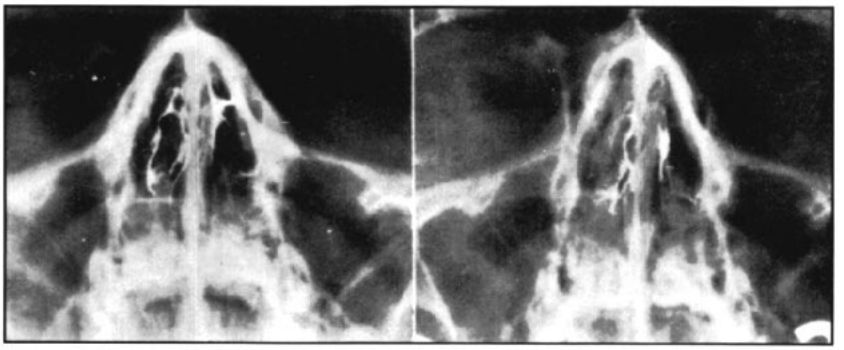

Figure 3

Figure 3
Roentgenogram of a normal subject during deep inspiration through the right nostril. Left: apnea, right: maximal inspiration. Almost total obliteration of the nasal lumen during deep inspiration is shown. The nasal valve is outlined with radioopaque tantalum dust. (From: Bridger, G.P.: Physiology of the nasal valve. Arch. Otolaryng. (with permission), 92:543-53, 1970.)

In some studies[8,11,56,57,59,63] the apparent radiographic degree of obstruction was estimated visually. In more recent publications, an attempt was made to quantitate the adenoid mass and pharyngeal space by linear measurement[3,55,58,62,64,65] and/or surface calculation[3,47,54,61,65,66] as a complement to the clinical examination. The finding of a significant relationship between the results of this cephalometric appraisal of airway patency and those obtained by posterior rhinoscopy and nasal airflow measurements has been used as evidence for the diagnostic validity of the cephalometric method.[54]

This statement, however, has met with severe criticism.[30,32,67] First of all there is the geometric limitation that a cephalogram produces only a two-dimensional representation of the space involved. Moreover, the nasal airway is a convoluted and anatomically irregular structure[68] and by superimposition and lack of soft-tissue detail this is obscured on traditional lateral or frontal cephalograms.[32] The limiting factor, that determines the capacity for airflow is the minimal cross-sectional area of the passage.[69] This narrowest portion can occur at any point along the nasopharyngeal trajectory and can only be accurately visualized by computed tomography.[68]

Presently, some attention is focused on the anterior portion of the nose at the nares ("the nasal valve area") which would be a significant flow limiting factor by acting as a Starling resistor.[70] At a certain point of inspiratory effort, the alar cartilages collapse and an increased inspiratory effort will not increase the nasal airflow (Fig. 3). Dilating the nares of a patient gives nasal resistance values that are reduced by approximately 40 percent to 45 percent.[32,71] The physiological and clinical importance of this is obvious. The nasal valve may be a factor much more important for nasal airway patency than apparent adenoid obstruction on a radiograph. Its evaluation should be included in every assess-

ment of nasal respiratory obstruction.[7]

Attempts have been made to quantify respiratory airflow objectively. A historical review of the methods developed is beyond the scope of this work. The reader is referred to the publication of Foxen et al.[72] for this purpose.

The method most frequently used in contemporary orthodontic research describes nasal patency in terms of nasal airflow at a certain pressure or by a nasal resistance value.[3,55,73-75] The nasal respiratory resistance (NRR) is calculated from the parameters of differential oral-nasal pressure and nasal expiratory airflow using a modification of Ohm's law.[69] However, this value may give no reliable indication of the breathing mode of the patient.[32] A value of 4.5 centimeters H_2O/l/sec and has been found to be associated with mouthbreathing in 77 percent of the cases.[76] This has been proposed as a critical value for airway impairment[67] even though variation can be considerable.[77] Additionally, some experimental studies[78,79,80] suggest the switching point from nasal to oronasal breathing is not only determined by a mechanical factor as nasal resistance, but that it is also influenced by psychological factors like perception of effort in breathing[78] or "nasal comfort".[80]

Recent SNORT studies (see below) also indicate that nasal resistance is not a good predictor of the percentage nasality of respiratory mode.[81,82] As a consequence, and as already commented upon by Vig,[32] it is not surprising to find the statistical correlations between visually determined mouthbreathing and NRR-values to be inconsistent throughout the experimental literature. (For a recent example, see the study of Cheng et al.[82a])

As the NRR assessment still has considerable limitations for describing a patient's mode of breathing, alternative methods have been developed.[85,86,87] Gurley and Vig[88] reported a technique with sufficiently small method error, which for the first time enabled the direct and simultaneous measurement of inspired and expired air, both orally and nasally. The technique is called Simultaneous Nasal and Oral Respirometric Technique (SNORT).[82,88] SNORT permits the objective quantification of the ratio of oral to nasal airflow and permits a numerical determination of both normal and pathological states of breathing mode.[32] This improvement in methodology may lead to more valid clinical reports that prove or disprove an association between oronasal respiratory mode and skeletal morphology.

Even within this methodology, inconsistent results may be expected if no further standardization of the experimental sample is attempted because different clinical variations of mouthbreathing have been described.[89] In other words, the altered reflex behavior to cope with a threatened airway is not completely identical in all subjects. As a consequence, the periosteal matrix may act differently on the dental and skeletal structures leading to different dentofacial results. An identical percentage of nasality in an oronasal breathing pattern does not warrant an identical postural response.

In search of experimental evidence

Based on an increasing quantity of experimental evidence, the prevailing belief today is that bone growth is strongly influenced by the soft tissues.[2] This "functional paradigm"[90] encompasses interpretations for the experimental data observed as Wolff's Law[91] and the Functional Matrix Hypothesis.[92,93,94] From within this paradigm the expectation would be that oral breathing, as an environmental impact and as an application of the above stated theories, would induce skeletal changes in the growing individual. Indeed, different but not necessarily conflicting theories have been proposed to explain the clinical associations found between mouthbreathing and some facial characteristics. One assumption is that mouthbreathing induces an altered pattern of muscle recruitment which in turn elicits skeletal change. The second theory, called the Soft Tissue Stretching Hypothesis, puts emphasis on the passive traction of the soft tissues as an indicator of bone growth. Both explanations will be investigated upon their experimental evidence and clinical validity.

Muscle function

A definite advantage of animal research is that the experimental circumstances can be much better controlled, pairing between treatment and control groups is easier and conditions can be used that would cause ethical problems when performed on humans. The primate experiments performed by Harvold et al.,[21,95,96] Miller et al.[97,98] and Vargervik[99] are examples of this. Rhesus monkeys were paired by age, sex and similar craniofacial measurements. Half the animals received silicone plugs bilaterally, completely blocking the airway during inhalation. Radiographs, EMG records and plaster models were taken at regular intervals during a two-year experimental period and also at one year following removal of the plugs. The results showed a marked alteration in craniofacial muscle recruitment over the whole experimental period and even thereafter. Although the behavioral response was individualized, mandibular position was lowered in almost all experimental animals. The malocclusions and skeletal distortions found were not uniform, e.g. open-

bite was not a constant finding but increased facial height and gonial angle were. The skeletal morphology demonstrated more the secondary characteristics of growth that result because the mandible is held at a lower position, allowing further growth and tooth eruption.[98]

These experimental data corroborate the hypothesis that a possibly morphogenetic impact such as mouthbreathing would indeed effect an increase in face height. However, one must be cautious to extrapolate the data to humans. There is a difference between the anatomy of the oropharyngeal passage, temporomandibular joint and location of jaw muscles in monkey and man.[97] So, postural responses and muscle recruitment are not necessarily the same. In this experimental set-up there is also a definite difference in amount of environmental impact with what is applicable to a clinical situation. The monkeys were adapting to a continuously total nasal obstruction, a condition that is exceptional in human beings. Because its long soft palate touches the epiglottis, it is practically impossible for the animal to breathe through the nose,[96,98] making the need for excessive muscle recruitment obvious. Moreover, a two-year period of nasal obstruction means a time-span of 46 percent of the monkeys' total growth period.[99a] Extrapolated to the human race according to Asdell's data,[99a] this would mean a period of total nasal blockage of about 8.2 years, which is irrelevant to the human situation. All these factors may explain why human clinical follow-up studies have revealed some morphologic changes, but have failed to show the same dramatic environmental impact as the experimental conditions showed.

Soft tissue stretch

The first systematic study of the relationship between craniofacial morphology and posture of the head and cervical column was carried out by Solow.[100] An association was found between craniocervical angulation, cervical inclination and some facial characteristics. One extreme postural type characterized by extension of head and neck displays craniofacial characteristics that are typically attributed to the patient with impaired nasal function (see above). Moreover, the same type of extension has been discussed in a previous publication[1] as a postural reflex mechanism induced by obstruction of the airway. The combination of these two observations led Solow and Kreiborg[22] to propose a hypothesis to account for the association between head posture, craniofacial morphology and airway obstruction. This has become known as the Soft Tissue Stretching Hypothesis. According to this theory, the observed morphological associations might be due to a dorsal and a caudal restraint on facial development during long-term extension of the head in the relation to the cervical column. The chain of reactions in which each element can be the triggering event is illustrated by Fig. 4.

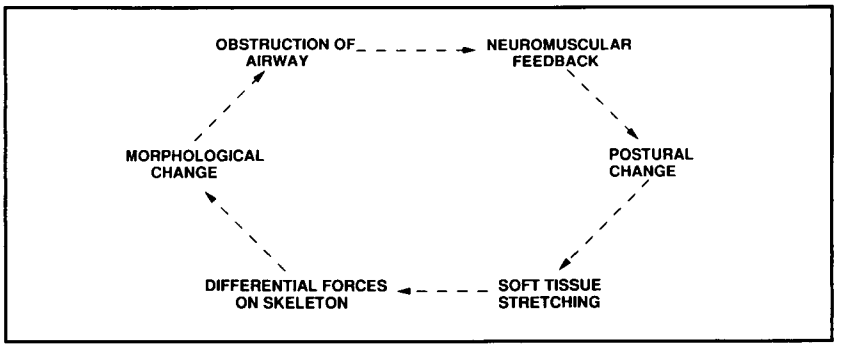

Figure 4

Figure 4
The soft tissue stretching hypothesis diagrammatically represented as a self-perpetuating cycle of events leading to morphologic changes in the craniofacial complex. (From: Solow, B., Kreiborg, S.: Soft tissue stretching: a possible control factor in craniofacial morphogenesis. Scand. J. Dent. Res. (with permission), 505-07, 1977.)

Of course, the hypothesis described is only speculative and will have to be subjected to experimental testing. As to the effect of stretching, it has been demonstrated that periosteal tension is able to induce bone deposition and consequent morphological change.[101] Moreover, bipedalism experiments by Moss[102] and Lisowski et al.[103] clearly demonstrate a morphogenetic effect of altered head posture. However, the environmental impact induced was so great that the data are hardly comparable to a clinical situation. The question is whether an increase of six degrees in craniofacial angle, as encountered in humans with total nasal obstruction,[111] is sufficient to induce measureable and physiologically relevant traction on the periosteum.

Archer and Vig[104] found differences in intraoral pressure with variable head positions but found no difference in labial pressures. So tongue position (by altered activity or by passive stretch?) may be of more importance in influencing the dental variables than extraoral stretching. Moreover, several studies indicate a link between head posture and altered muscular activity.[105-110] In light of the above, it is conceivable that if long-term alterations in head posture are accompanied by a long-term change in motoneural firing, this periosteal matrix may well alter craniofacial morphology. Consequently, although there is not yet hard evidence to reject the Soft Tissue Stretching Hypothesis, altered muscle recruitment seems to be a more plausible explanation for the skeletal changes found.

Discussion and conclusions

There is abundant experimental evidence that altered muscular function can influence craniofacial morphology. The morphogenetic impact

of an oral breathing pattern as demonstrated in the animal experiments can be explained within the context of Wolff's Law or the Functional Matrix Theory of Moss as the result of a reflexive neuromuscular adaptation to certain functional needs. Human clinical studies show controversial results, primarily because the experimental design does not allow for inference about any cause-and-effect relationship and/or because of methodologic difficulties in assessing and quantifying the patient's mode of respiration. Within this context, all clinical data should be interpreted with caution.

Some longitudinal studies support the hypothesis that an alteration in breathing pattern has a limited impact on craniofacial growth. In contrast, dramatic skeletal and facial differences out of the normal range have been described for the LFS.[2,13,44] As such, the question can be raised whether breathing mode is of sufficient importance to be regarded as a major etiological factor leading to the LFS physionomy.

A vertical growth pattern seems to be established very early in postnatal life (from at least the third year on) and, if untreated, remains relatively stable during the craniofacial growth period.[114] In addition, there is evidence in the literature that consistently shows a higher degree of vertical morphologic craniofacial similarity in monozygotic than in dizygotic twins.[115-119] Moreover, intrapair differences in size of facial skeleton increased with age four times more in dizygotic than monozygotic twins.[117] Under the assumption that the environmental factors have been different for adult monozygotic twins reared apart, intercanine and intermolar widths showed a significant intrapair resemblance at the .001 level of significance.[120]

As the shape of the dental arch obviously can be influenced by activity of the orofacial musculature, here too, a higher degree of similarity in EMG activity has been reported in monozygotic than in dizygotic twins.[112] Muscular pathologic changes at the microscopic level[112,113] have been reported for the LFS and, hypothetically, may be indicative of a more systemic or neurologic etiology of this growth pattern.[122,123] As a consequence, the possibility of a genetic predetermination towards a LFS-morphology cannot be ruled out. The currently prevailing belief is that the genome is rather expressed in the phenotypical traits of the soft tissues.[90] Skeletal morphology would be mostly the result of the growth and action of this functional matrix. However, this expression of the soft-tissue genome would remain during growth. Functional needs would superimpose to a certain extent, to mold the skeletal structures into their definitive form. This would account for a built-in different muscular morphology of the LFS-patient,[124,125] the possibility of some degree of adaptability to the altered functional needs that may occur during growth and a higher susceptibility of some patients to these neuromuscular modifications in comparison with others. At our present state of knowledge, the scientific evidence prompts us to moderate our overall clinical expectations and to expect individual variability in response when we therapeutically attempt to alter a LFS patient's breathing mode.

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