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DENTOFACIAL DEVELOPMENT IN CHILDREN WITH CHRONIC NASAL RESPIRATORY OBSTRUCTION -- A CEPHALOMETRIC STUDY

by

TAI-YANG HSI B.D.S.

A Thesis Submitted to the Faculty of the Graduate School of Loyola University of Chicago in Partial Fulfillment of the Requirements for the Degree of

Master of Science

December

1989

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VITA

Tai Yang Hsi was born in Taipei, Taiwan, Republic of China, on November 11, 1950.

After graduation from high school, he enter Taipei Medical College, School of Dentistry and received his degree of Bachelor of Dental Science in June 1975.

Upon graduating from Dental school, he served in the Army as reserved Dental Officer for two years.

After discharging from the Army, he went to LuKu Health Center, the so-called "No formal Health Provider - Village, as dentist for one year and then back to his hometown Taipei to open his own dental office.

During those years of practicing as general practitioner, he has been the Broad of Director of Taipei Dental Association for four years, also been the Delegate of National Dental Association for two years devoting himself to dental public health and education.

In September 1985, he entered the School of Public Health of Columbia University in New York city and major in dental public health for one year.

In July 1989, he went to Loyola University, School of Dentistry as a post-graduate student in the Orthodontic Department and also enrolled in the graduate program of Oral Biology.

He received a certificate of specialty in Orthodontics in December 1988.

DEDICATION

To my wife, my mother-in-law, and my brother

whose supports to my family and the office

plus encouragement

made my study overseas possible.

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Chapter I

INTRODUCTION

Since the first study[1] showed a relationship between nasal airway obstruction and the development of facial growth patterns, many attempts have been made to establish a relationship between nasal airway obstruction and dentofacial abnormalities. The relation between nasorespiratory function and craniofacial morphology has a long and contentious history in orthodontics. It was based on the premise that restricted nasal airway function leads to "mouth breathing," which in turn results in a lowered tongue position and depressed mandibular posture[2]. If this altered posture was sufficiently prolonged during active growth, the result may be a narrowed maxillary dental arch, an increased lower facial height, an increased mandibular angle and an incompetent lip morphology. These features were often called long face syndrome or "adenoid face". Ricketts[3] described this condition as "Respiratory obstruction syndrome"

There has been some disagreement between groups : who exclusively support the functional matrix theory, that is, that function dictates form, and others who believe that facial structure was governed strictly by heredity.

The differing views on the relation between mouth breathing and a specific type of facial structure and

malocclusion fell into the following main groups:

1. Mouth breathing gave rise to a specific type of facial structure and malocclusion.

2. No relation exists between these phenomena.

3. Mouth breathing was a secondary phenomena to a specific hereditary pattern of facial structure.

Ranly[4] proposed a composite view. She stated that the chondrocranium was influenced by both intrinsic genetic and local environmental factors. These theories were relevant to the
controversy regarding the effects of altered respiration on facial structures.

Though there were some controversial points of view[5 6 7 8 9], a number of studies confirmed a relationship between nasopharyngeal airway obstruction and abnormal craniofacial development.[10 11 12 13 14 15 16 17 18]

Several articles suggested a direct cause-and-effect relationship between nasal airway obstruction and altered dentofacial morphology. Further well-controlled studies designed to quantify the relative amounts of oral versus nasal respiration were necessary before airway obstruction could be implicated as a significant etiologic factor in the development of any specific dentofacial deformity.

Within the field of orthodontics it has recently become apparent that nasal respiratory function played a significant role in the development of the face and occlusion. For this

reason, it was important to be able to determine whether or not there exists a reduced capacity for nasal breathing.

The purpose of this study was to identify the effect of chronic nasopharyngeal obstruction on the growth of facial pattern in children between ages three and seven years old.

Chapter II

REVIEW OF LITERATURE

(A). ANATOMY, GROWTH, AND PHYSIOLOGY

ANATOMY

The nasopharynx was a musculomembranous tube serving as a portal between the nasal chamber anteriorly and the oral pharynx inferiorly. The roof and posterior wall made a continuous curve downward upon the body of the sphenoid bone, the basilar part of the occipital bone, the arch of atlas and the body of axis. Its primary biologic function was to provide a passageway for air from the nasal chamber to the oral pharynx and ultimately to the lungs.(fig.1)

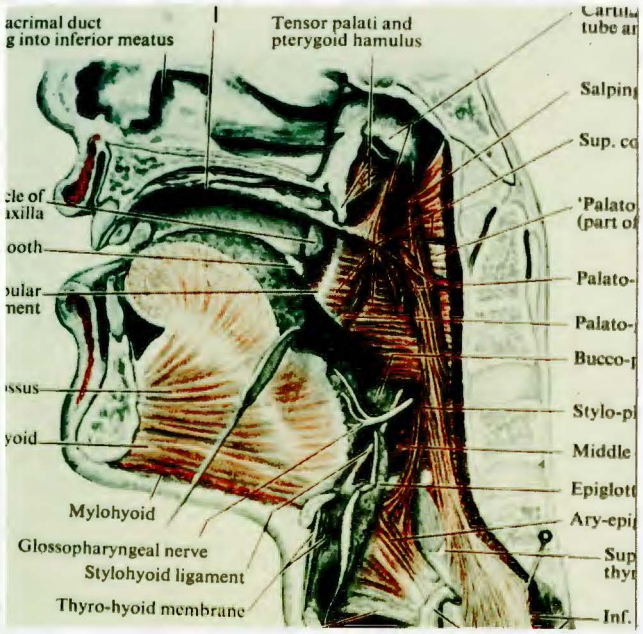


Fig. 1 Anatomy of upper airway

The nasopharynx also provided space on its posterior and superior walls for lymphoid tissue in the form of the nasopharyngeal tonsil where was part of Waldeyer's tonsillar ring. If this lymphoid tissue became hypertrophied so that it precipitated clinical symptoms, it was denoted as vegetation of the adenoid.

When the adenoid tissue was visible on the mid-sagittal radiograph, the major portion of the nasopharyngeal cavity may have appeared to be filled with lymphoid tissue as a convex soft tissue prominence. Its anterior inferior border approached the superior aspect of soft palate and inferior turbinate to varying degrees and passed posteriorly to blend into the posterior pharyngeal wall. The attachment to the posterior pharyngeal wall usually extended inferiorly to slightly below the level of the anterior tubercle of the atlas. Anteriorly, it occluded with inferior turbinate and the posterior superior aspect of the vomer bone.(fig.2)

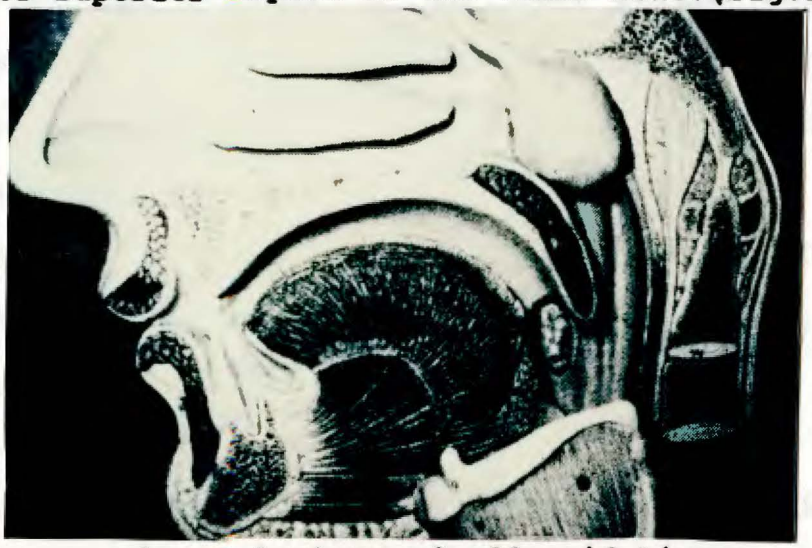


Fig. 2 Anatomy of addenoid tissue

The enlargement of the adenoid pad may have led to partial or total blockage of the nasopharyngeal passage making nasal respiration either inefficient or impossible.

GROWTH

The shape and size of the nasopharyngeal cavity can be defined in terms of depth and height in the median sagittal plane and width in the frontal plane. According to Brodie[19], King[7], Handelman & Osbornes' study[20], the total depth of the nasopharynx was established in the first or second year of life. King further stated that the increase of the depth of the nasopharynx was by the growth at the spheno-occipital junction. Ricketts[21] and Bergland[22] demonstrated that the more obtuse the cranial base, the greater the depth.

In contrast to the early stabilization of depth, King[7] demonstrated continued increase in nasopharyngeal height until maturity by the descent of the hard palate and cervical vertebrae from the cranium. Bergland[22] demonstrated a thirty-eight percent increase in nasopharyngeal height from six years of age to maturity.

Subtelny[23] demonstrated the width of the nasopharynx may be established early in life. The volume of the bony nasopharynx increased from six years to maturity by 80 percent in Bergland's[22] skull material. This increase was primarily due to changes in height and width, while depth remained stable.(Fig. 3)

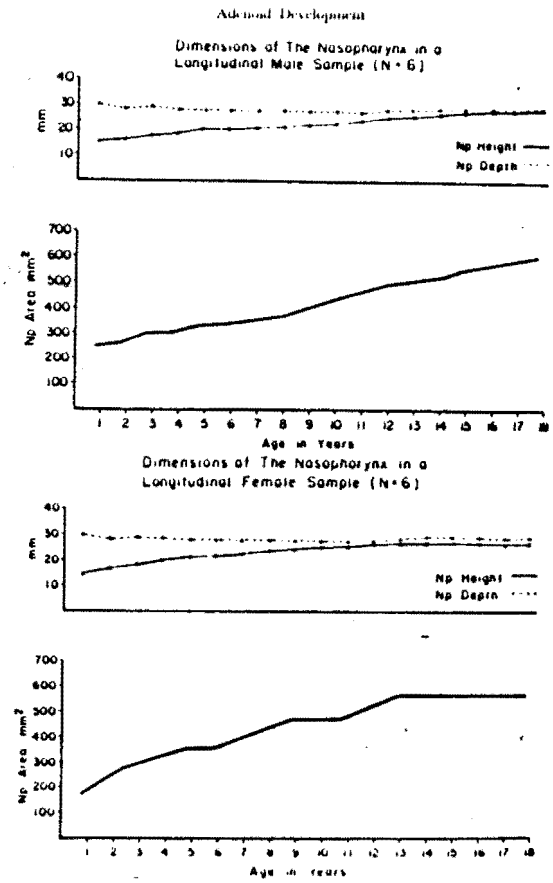


Fig. 3 Growth of Nasopharynx in height and depth Subtely[23]

In Handelman and Osborne's study[20] of the growth of the nasopharynx and adenoid development using lateral head films in patients from one to eighteen years of age. Four skeletally defined lines are used to measure the airway area and adenoid area. The nasopharyngeal area was defined as a trapezoid.(Fig.4) The nasopharyngeal area was divided into an adenoid-pharyngeal wall and an airway areas which were measured using a polar planimeter. The trapezoid analysis

proved to be a useful technique for quantification of nasopharyngeal dimensions.

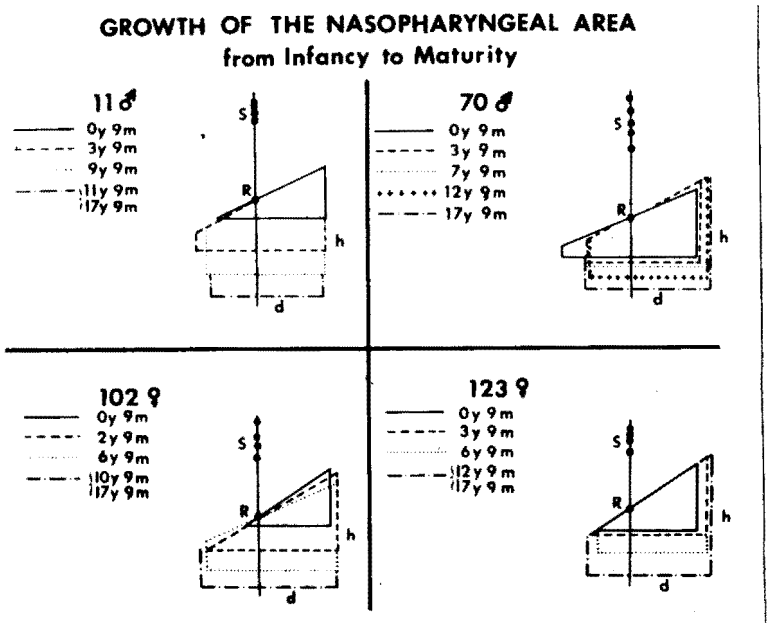


Fig.4 Growth of nasopharynx area

Handelman and Osborn[20]

Scammon[24] demonstrated that lymphatic tissue as interstinal lymphoid masses and thymus, shows rapid growth in infancy and early childhood, and continued to grow, though at a slower rate, until puberty with a gradual decline thereafter.(fig.5) Basing his observations on cadaver material, his graphs indicated that the peak of lymphatic growth was reached at about 10 to 11 years of age. Adenoid tissue, being lymphatic tissue, may follow this some path of growth.

Adenoid tissue was found to follow a definite growth cycle. It seemed to have a specific growth potential and it

was on this potential that the hypertrophic reactions to nasorespiratory infections and allergies may be superimposed.

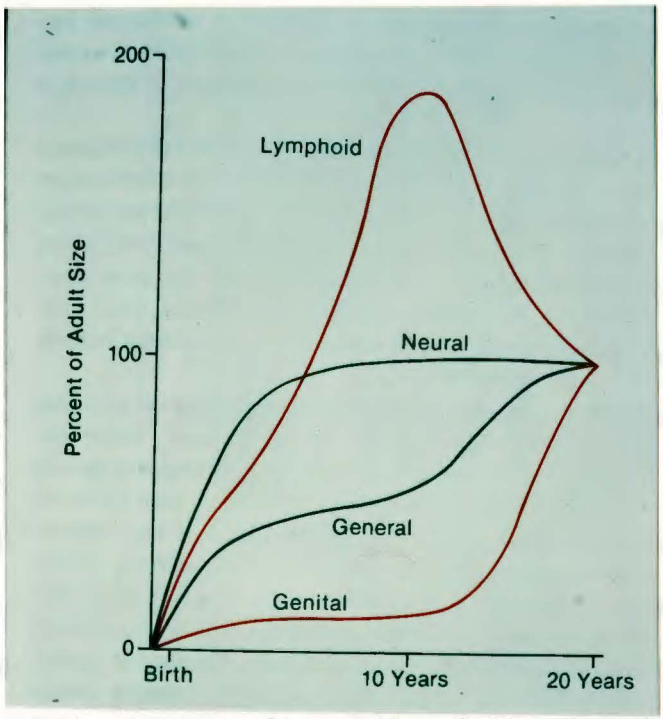


Fig.5 Scammon's curve of growth of the lymphoid tissue

Subtelny and Baker's radiographic study[25] indicated that the adenoids attained its maximum bulk between the ages of nine and fifteen years, and showed subsequent atrophy. They also point out that at age four to six the growth of the adenoids and the contiguous nasopharynx were largely related to each other in a delicate balance if the airway was to be maintained. The adenoids usually peaked in their growth prior to the adolescent spurt of the skeleton. If they increased in mass faster than the nasopharynx increased in size, proper nasorespiratory function was impeded and mouthbreathing may have developed. They concluded that the adenoids led to

mouthbreathing primarily in children with a small nasopharynx.

Johannesson[26] believed the roentgenographic evaluation of adenoid size was reliable and used it to investigate the nasopharyngeal tonsil in children of different ages.

Only minor changes in size were observed between the ages of 2 to 15 years. The means for these age groups ranged from 12.0 to 14.3 mm. It was reported that the increase of the size of adenoid occurred during the first two years of life and thereafter remained unchanged.

Generally, most subjects demonstrated minimal adenoid tissue at one year of age, adenoid hypertrophy evident by two years,a maximum amount of adenoid tissue during the early school years.(Fig.6,7,8,9,10)

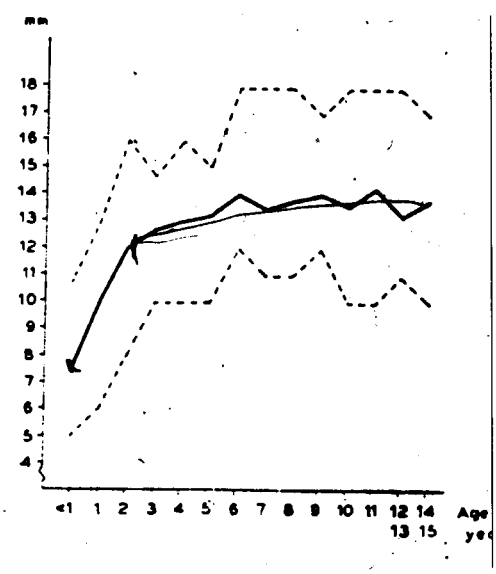


Fig. 2. Graph depicts the greatest width of the soft tissue in the nasopharyngeal roof in relation to age. Each point of the solid curve is the mean of measurements made in 10 children. The ranges are indicated by broken curves.

Fig.6 Curve of growth of adenoid tissue

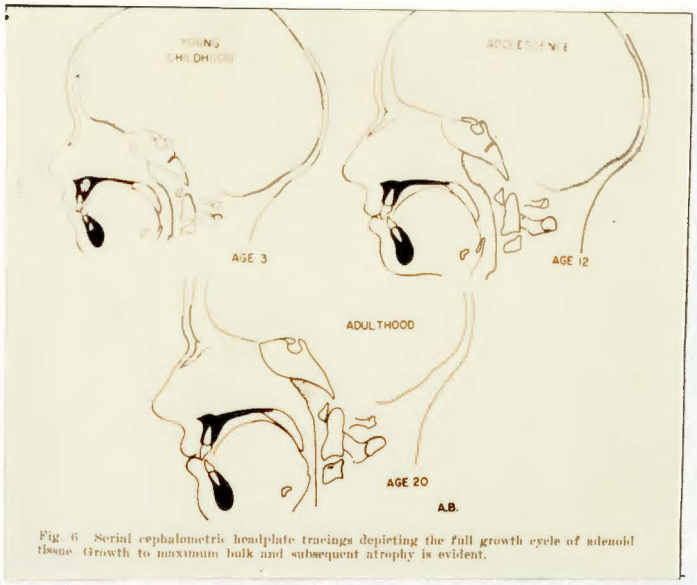


Fig. 6 Serial cephalometric headplate tracings depicting the full growth cycle of adenoid tissue. Growth to maximum bulk and subsequent atrophy is evident.

Fig.7 Growth cycle of adenoid tissue

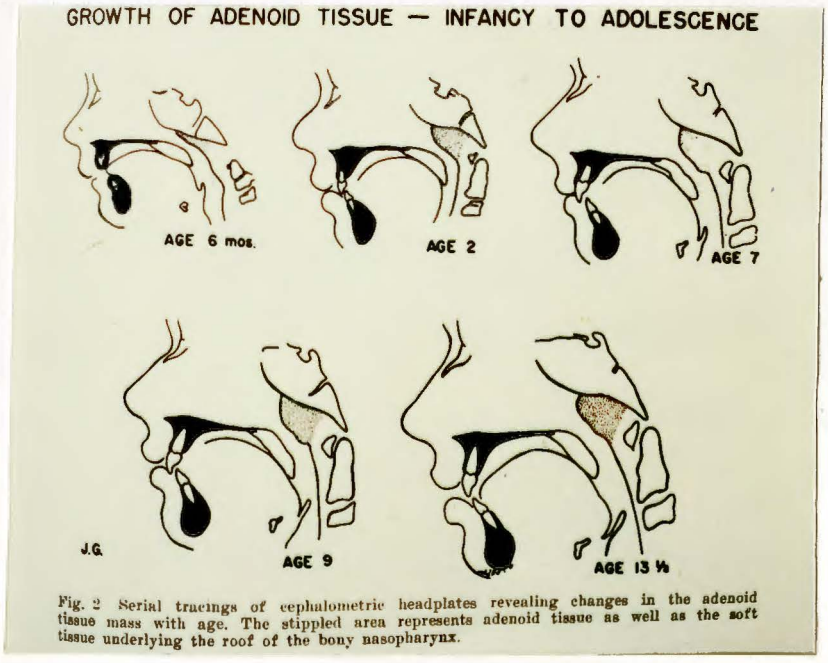


Fig. 2 Serial tracings of cephalometric headplates revealing changes in the adenoid tissue mass with age. The stippled area represents adenoid tissue as well as the soft tissue underlying the roof of the bony nasopharynx.

Fig.8 Growth of adenoid tissue, from infancy to adolescence

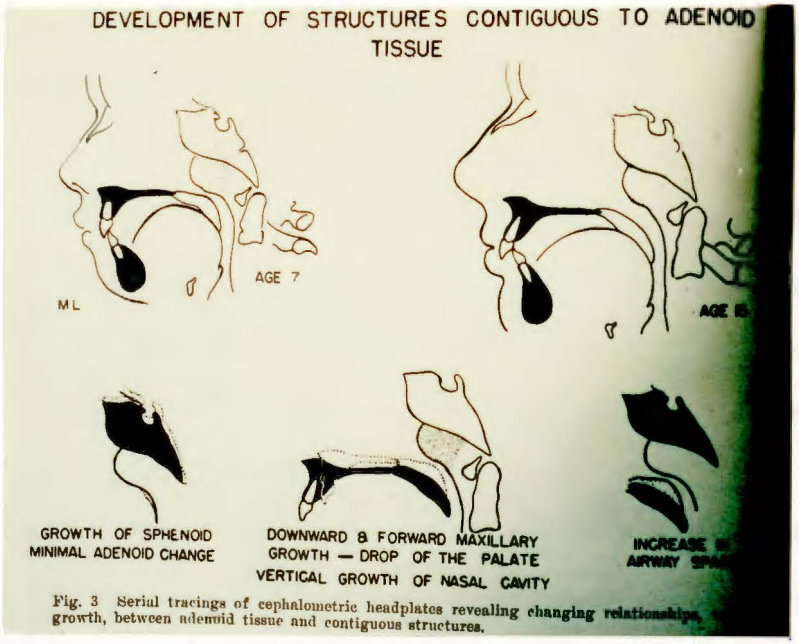


Fig. 3 Serial tracings of cephalometric headplates revealing changing relationships, growth, between adenoid tissue and contiguous structures.

Fig.9 Development of structures contigous to adenoid tissue

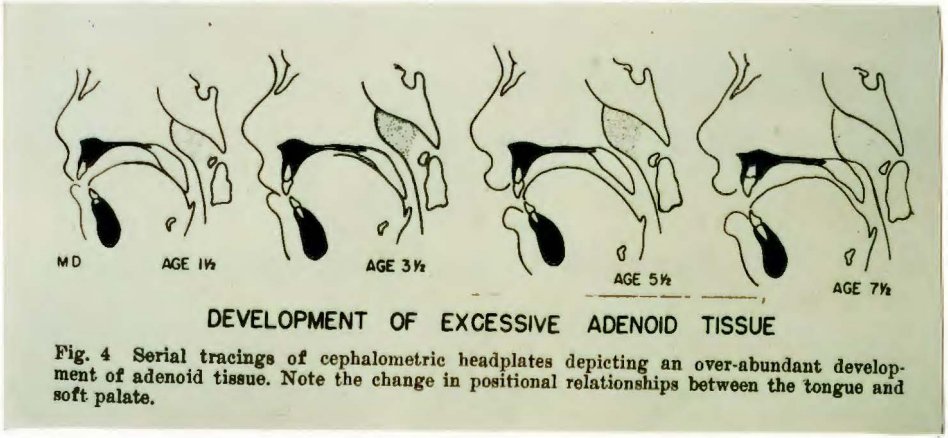


Fig. 4 Serial tracings of cephalometric headplates depicting an over-abundant development of adenoid tissue. Note the change in positional relationships between the tongue and soft palate.

Fig.10 Development of excessive adenoid tissue

PHYSIOLOGY

Miller,et al[27] tried to test the traditional concept that newborn infants were unable to breathe through the mouth and were thus obligatory nasal breathers.

The conditions under which oral breathing could occur and the contribution of oral ventilation to total ventilation were studied in 30 healthy term infants (aged 1 to 3 days). Nasal and oral airflow were measured using two resistance-matched pneumotachometers. The findings were as follows:

1. Spontaneous oronasal ventilation occurred during sleep.
2. Oronasal ventilation was also observed after crying.
3. Oral airway may be used effectively by infants in response to complete nasal occlusion.

These findings considerably alter the previous concept of the newborn infant as an obligatory nasal respiration.

Rodenstein & Stanescn[28] investigated the ability of the soft palate to direct airflow during breathing. They found the soft palate closed the oropharyngeal isthmus during quiet breathing(resulting in pure nasal breathing) and closed the nasopharynx during FVC effort (Forced Vital Capacity), which resulted in mouth breathing. During oronasal breathing, the soft palate was positioned between the tongue and the posterior pharyngeal wall.

(B). ETIOLOGY, SYMPTOMS OF AIRWAY OBSTRUCTION

Nasal obstruction that led to an alteration in mode of breathing can be caused by a variety of factors such as allergic rhinitis, adenoid hypertrophy, nasal polyps, congenital nasal deformities, neoplasms,and recurrent upper respiratory infections.[29] Perennial allergic rhinitis with accompanying nasal edema was the most common cause of nasal obstruction in children.

Weimert[30],an Ear,Nose,Throat Specialist, emphasized the function of the nose and role of the nares. The most critical area to the nose with regard to obstruction was the laminae valve area,located just inside the nares anteriorly. This is the smallest cross-sectional area of the nose. Relatively minor changes in nasal architecture in this area resulted in a significant increase in nasal airway resistance. It was the inferior turbinate responsible for airway obstruction. When there was inferior turbinate hypertrophy, choanal atresia, vasomotor rhinitis and polyps were other frequent etiology of nasopharyngeal airway obstruction.

Adenoids have long been regarded as one of the chief causes of mouth breathing, and this hypothesis recurs in many textbooks. [1] Several authors have stressed the importance of adenoids as the primary cause of mouth breathing.[1 31] The relative size of the nasopharynx as a cause of mouth breathing has also been cited.[32 33 34 35] Linder-Aronson[13] found that the

adenoids led to mouth breathing primarily in children with a small nasopharynx.

Adenoidal hypertrophy was the most common source of chronic airway obstruction in patients screened by the orthodontist.[36] It was accompanied by a description of a particular facial expression, which was typical of individuals with adenoids, i.e. the adenoid facies(Fig.11,12). Individuals that exhibited this facies were characterized by enlarged tonsils and had most or all of the following characteristics in common: the mouth stays open,a long narrow face with increased anterior vertical facial height in the lower third of the dentofacial skeleton, a flattened nose, small and underdeveloped nostrils, a short hypotonic upper lip, a thick and exerted hypertonic lower lip. The bite was also stated to be of a special type.[37]

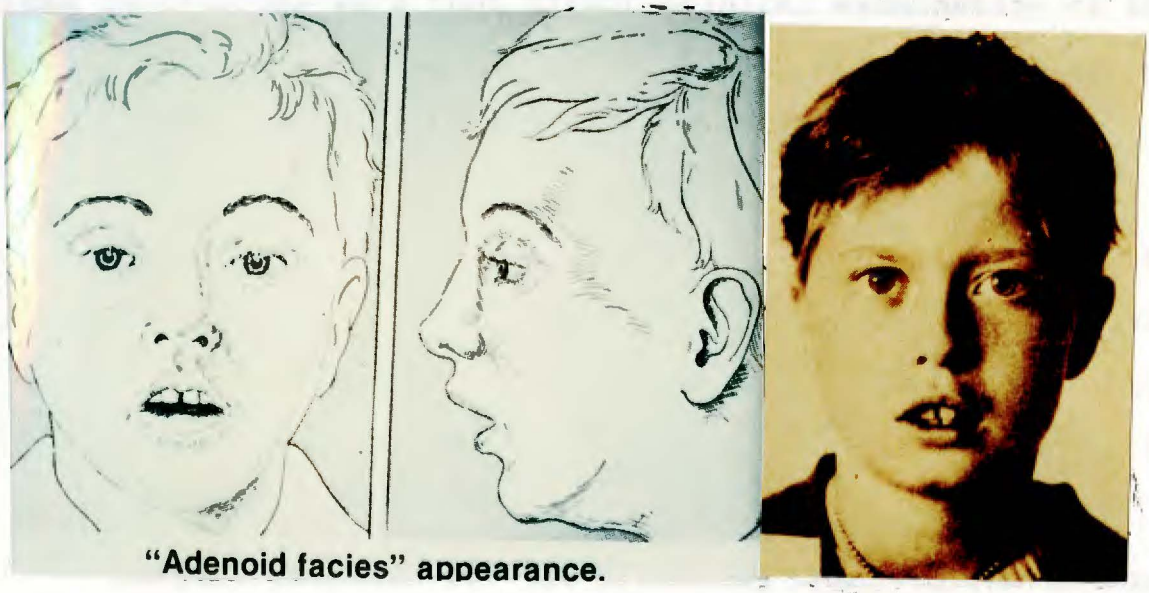


Fig.11 12 Adenoid facies

The "Allergic shiners" described by Weimert[38] were darkened areas below the eyes that were seen in people with allergies or in any patient with significant nasal obstruction. They are caused by venous congestion due to swelling in the nasal tissues.

(C). Airway Obstruction related to mouth-breath

Dr.Weimert[38], evaluated his young otolaryngeal patients and found that patients who were observed to mouth-breathe "all of the time" had an 85-percent incidence of demonstrable airway compromise.

There have been a number of studies correlating airway obstruction symptoms with various diagnostic techniques and the conclusions were that direct clinical examination of the nasal chambers using anterior and posterior rhinoscopy correlated best with patient symptomatology.

Galen Quinn[39] stated a practical clinical approach to identifying and evaluating nose breathing capabilities.It was whether or not the individual could comfortably inspire air through both nasal cavities without effort. Resistance in inspiration was greater in the child than in the adult.

Patient position for the breathing test was shown. Nose breathing capability was first tested by gently closing the lips together with light pressure of thumb and middle fingers

for 2 to 5 minutes(Fig.13). It was important that the patient not be informed of the purpose of this act.

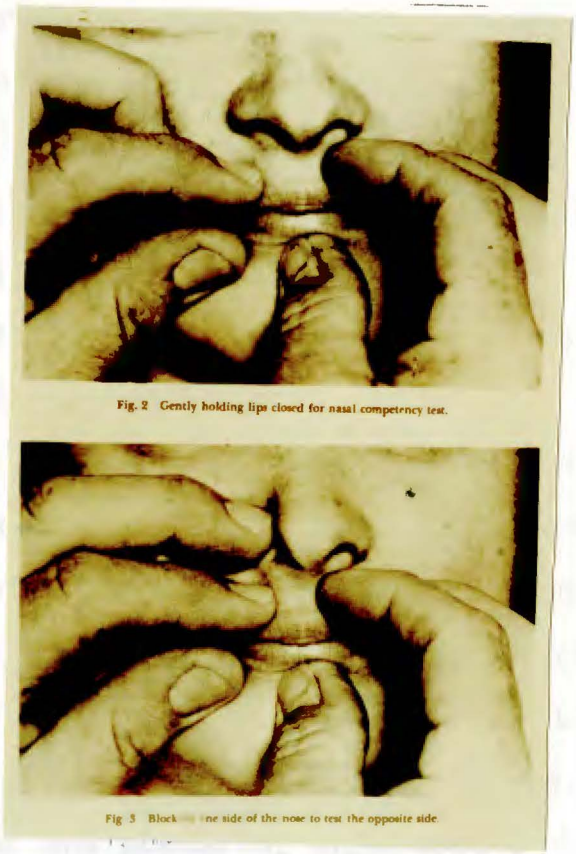


Fig. 2 Gently holding lips closed for nasal competency test.

Fig. 3 Block one side of the nose to test the opposite side.

Fig.13 Clinic test for nasal airway obstruction

In a multi-dimensional study, Linder-Aroson[13] evaluated the relationship between adenoids and mode of breathing. Experimental and control groups were evaluated biometrically, rhino-manometrically,and cephalometrically.

The results showed that the size of adenoid and the nasal airflow resistance was essentially determined by the relationship between size of adenoid and the size of

nasopharynx. The nasopharyngeal airway was important for the mode of breathing and large adenoids lead to mouth breathing primarily in children with a small nasopharynx. In these children, adenoidectomy was indicated as a means of promoting a change to nasal breathing.

Hibbert and Tweedie[40] investigated the relationship between preoperative signs and symptoms and the actual size of the adenoid found at the time of operation in a group of children listed for adenoidectomy.

A series of 80 children was the sample of the this study. The day before the operation the parents of the children were interviewed and questioned as to the presence of nasal obstruction with mouth breathing, snoring, rhinorrhoea, cough, headache and hyponasal speech. The children were then examined and assessed for evidence of mouth breathing. They were examined by anterior and posterior rhinoscopy.

The following day an adenoidectomy and bilateral antral lavage were performed. The removed adenoid was washed in saline, dried with gauze and weighed, and its volume was also measured.

The result of this study showed that in children under 7 the signs and symptoms usually attributed to adenoid hypertrophy have no statistical significance in the prediction of the size of adenoid.In children aged 7 and over, a history of snoring or clinical evidence of mouth breathing was related

to the weight of the adenoid and statistically significant at the 5% level.This would also suggest that in the younger age group adenoidectomy has little place in the management of most cases of nasal obstruction,nasal discharge and snoring.

Crepeau, et al[41] did a study on the radiographic evaluation of the symptom-producing adenoid. Adenoid hypertrophy had several variable symptoms. In this study, symptoms were divided into minor and major. A lateral radiograph of the nasopharynx was performed in 114 patients to study the superior and anterior adenoid diameters(Fig.14). A correlation was made between the various clinical groups and the adenoid measurements. Their result support Hibbert's[40] finding that the anterior adenoid width was a better indicator of the symptom-producing adenoid than adenoid mass measurements with their loosely defined norms. A through history and physical examination remained paramount in the diagnosis and management of adenoid hypertrophy.

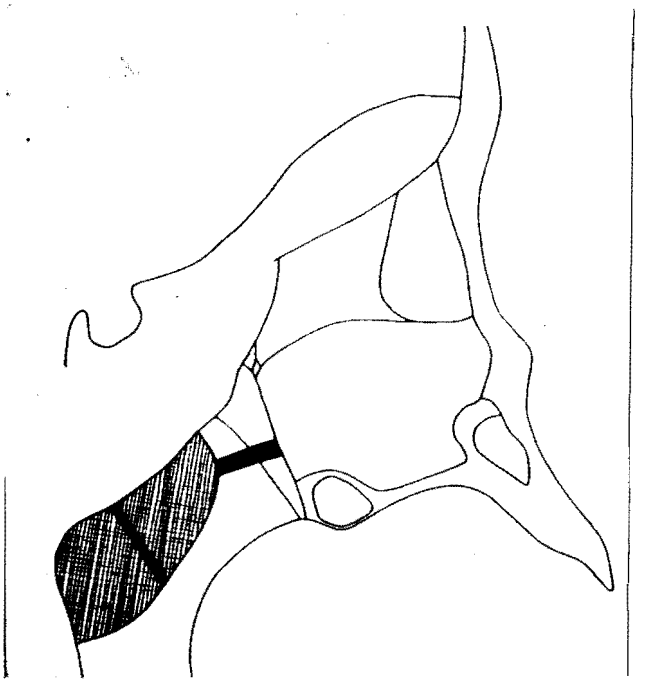


Fig.14 Antroadenoid and superioinferior diameter on lateral radiograph

(D). Response Chain (Tongue, Neuromuscular function, Mandible posture and head position)

Hannuksela[42] and Shapiro and Shapiro[43] have demonstrated that children with allergic hypertrophy of the faucial tonsils, adenoidal pad, and later, the inferior turbinates would develop the long-face syndrome. Conversely, the child with a normal upper airway was much less likely to develop this syndrome.

The question whether adenoids were associated with a special facial type was also evaluated by Linder-Aronson[12]. In

that study, photographs were observed by two observers independently and it was found that 75% of all of the children who underwent adenoidectomy were classified as having adenoid facies. Furthermore, adenoid facies was judged to be present in about only 4% of the controls. It followed that in a screening based on facial type alone, many cases requiring adenoidectomy would be missed at the same time as some cases would not need surgery. The facial characteristics of the group of children who underwent adenoidectomy showed a large facial height, high mandibular plane angle, small sagittal nasopharyngeal depth and small width/height facial ration. He concluded that adenoids occur in children of various facial types and obstructed nose breathing due to adenoids appeared to be most common among children with a leptoprosopic type of face and a small nasopharynx.

The upper airway may play a primary role in the generation of a secondary tongue dysfunction.[44] A close interaction between airway and tongue dysfunction may present many different aspects that enable a variety of clinical situations to occur. These differences in the morphogenic effect of a few basic and common etiologic factors may have been related to the timing at which an anatomic discrepancy occurs during growth.

The forward pressure from the alteration of proprioception of inflamed upper airways caused protraction

of the tongue. By acting during growth, these factors may change the growth pattern of the bony architecture to which the neuromusculatures to tongue were related.

Considerable hypertrophy of the tonsils and adenoids may push forward a normal tongue and transform it into a pathogenic factor acting to create a skeletal discrepancy. A simple volumetric correction of the hypertrophied tissues, when effected early, may be sufficient to deactivate the pathogenicity of the tongue and normalize the growth patterns of the face.

Thus, the pathogenicity of any given tongue was related to the status of the airways at a given time. Therefore, when abnormal growth and development at the level of the stomatognathic system was recognized at an early stage, and was related to a large tongue with upper airway obstruction.

Vargervik, et al[45] evaluated monkeys to test whether specific recordable changes in the neuromuscular system could be associated with specific alterations in soft and hard tissue morphology in the craniofacial region.

The neuromuscular changes were triggered by complete nasal airway obstruction and the need for an oral airway. Statistically, significant morphologic effects of the induced changes were documented in several of the measured variables after the 2-year experimental period.

They concluded that the changes in neuromuscular

recruitment patterns, which were necessary to establish and maintain an oral airway, resulted in altered soft-tissue and skeletal morphology. The extent of the skeletal changes appeared to depend on the degree of soft-tissue alterations. The degree of morphologic change, therefore, does not depend on the amount of air that flows through the mouth or nose, as has been stated by some authors. Rather, it depended on the nature of the neuromuscular and soft-tissue adaptations.

The other findings were as follows:

1. The anterior face height increased more in the experimental animals than in the control animals,
2. The occlusal and mandibular plane angles measured to the sella - nasion line increased,
3. The anterior crossbites and malposition of teeth occurred.

The experimental use of silastic plugs to create nasal obstruction in the rhesus monkey has clearly demonstrated that nasal obstruction with open-mouth posturing recruits accessory respiratory muscles around the mouth and jaws and led to the same clinical facial deformity and malocclusion.

Harvold[10] has produced increased anterior face height, narrowing of the maxilla, steeper mandibular phase angles, narrower thinly pointed tongues and larger gonial angles in monkeys by obstruction of air flow with nasal plug. He concluded that specific changes in jaw positioning could cause corresponding bone remodeling, but this should not be

correlated with a particular type of malocclusion.

Another animal experiment[11] determined if lowered tongue position caused by mouthbreathing can affect the craniofacial morphology. The lowered tongue position was induced by tactile stimulation to tongue from an acrylic block positioned in the palatal vaults of three groups of monkeys.

In group I all the experimental monkeys with the insert in the posterior part of the palate developed an open bite and significant changes in the dental arch. In group II and III with the insert in the anterior part of the palate, all animals manifested malocclusion and significant changes in the dental arches . The face height increased significantly in all experimental animals.

This study showed that any consistent changes affecting the relative tonus in the muscle groups suspending the mandible influences the extrusion of the teeth and the establishment of face height.

The findings of Drs.' Vargervik and Harvold animal study suggested that the position of the chin and the inclination of the mandibular plane were controlled by the balance between the hyoid and the orofacial muscles.[21] The morphology of the ramus appeared to be primarily controlled by the masticatory muscles. They also concluded that the changes in neuromuscular recruitment patterns were necessary to establish and maintain an oral airway and resulted in altered soft-tissue and skeletal morphology. The extend of the skeletal changes

appeared to depend on the degree of soft-tissue alteration present.

The nose and nasopharynx were the primary airway. Under normal circumstances, nasal breathing did not require recruitment of accessory respiratory muscles. When mouth breathing was forced by obstructions in the nasal airway or by increased oxygen demands, accessory respiratory muscles were recruited. These included craniofacial muscles involved in formation of an oral airway. They may include neck muscles that extend the head and neck. If the mouth-breathing was temporary, such as during catching a cold or during exercise, the neuromuscular change would fluctuate and would not produce dental or skeletal changes. If mouth-breathing persisted and became a habitual pattern during those periods of normal whole-body growth, the associated changes in the position and shape of the tongue with lowering of the mandible may have certain effects on dentoalveolar and skeletal morphology. It was that the child's neuromuscular adjustments to and impaired nasal airway were the determining factors in the effects on developing facial and dental structures.

Changes in mandibular morphology will only occur when lowering of the mandible was sufficiently consistent. Downward displacement of the maxilla and excessive extrusion of teeth may or may not have occurred in response to a lowered mandibular posture. The maxillary response was mainly determined by tongue posture and movements. Lower face height

was measured with the teeth in occlusion and increased significantly when a downward displacement of the maxilla or excessive molar extrusion occurred. Increased molar extrusion would be expected to occur most rapidly during eruption of the first and second molars.

Chronic mouth-breathing called forth the recruitment of perioral and suprahyoid muscles[46]. The increased tonicity and rhythmicity of these muscle groups often produced a negative effect on dentofacial form and function. Often, the long-face syndrome developed as a result.

Children with a genetic proclivity for dolichocephalic dentofacial development were at higher risk, as were children with neuromuscular dysfunctions[46]. Allergic hypertrophy of the tonsils, adenoid pad, and inferior turbinate, when combined with neuromuscular dysfunction and a genetic predisposition for the dolichocephalic face, placed that child in the highest risk group of all.

The causal relationship between adenoid vegetation associated with mouth breathing and increased lower facial height may be due to a rotation downward and backwards of the mandibular symphysis.[47]

The head posture was investigated by Linder-Aronson in 16 patients who had undergone adenoidectomy due to difficulties in nose breathing. A comparison was made with a similar number of controls in the same age group without impeded nose breathing. Inclination of SN line was measured

relative to a vertical reference line included in the lateral skull radiographs. A small value of the SN/vert. angle expressed extended head posture. Measurement was made initially and 1 month after adenoidectomy.

A significant difference was noted. In order to increase the respiratory passage, the head was extended forward with an increase in lower facial height and a resultant increased retrusive pressure from the facial musculature on the underlying skeleton.

Bosma[48] has stated that one important function of head posture was to maintain an adequate naso-oro-pharyngeal airway, In patients with morphologic disturbances which impede and adequate airflow one can expect to find an extended head posture. The Pierre Robin syndrome was an example of such a morphological disturbance.

Solow and Greve[61] studied head posture and its relation to nasal respiratory resistance.It confirmed the results of Linder-Aronson's work. They examined 24 children ages 4 to 12 years before and after adenoidectomy. Cephalometric recordings of the natural head position and rhinomanometric readings of nasal resistance were obtained for each child.Before adenoidectomy, a large craniocervical angulation was seen in relation to large nasal respiratory resistance and narrow airway. After adenoidectomy,reduction of craniocervical

angulation resulted in children who had received adenoidectomy and nasal resistance was reduced.

The findings confirm predictions of soft tissue stretch hypothesis and provide an explanation for the reversibility of craniofacial morphology previously observed.

Bibby[49] stated that in mouth breathers one might have expected a different head posture to be adapted to facilitate breathing especially where the mouth breathing was due to an obstructed nasopharynx.

Individual Variance

In one of Vargervik's animal studies[45], silicon plugs were formed to fit the individual nares to obstruct inspiration but allowed some air to escape during expiration. The changes observed in the middle and front of the tongue showed considerable variation in tongue adaptations. This was reflected in the individual animal's optimal adjustment to the experimental condition present. This study demonstrated a wide individual variation in response to an identical stimulus.

For this reason Dr.Meredith[46] suggested that a detailed history and physical examination should be complemented by serial cephalometric x-ray studies, PA tomograms of the nasal vault ,rhinomanometric studies and, in selected cases, sleep-laboratory studies.

(E).CEPHALOMETRIC STUDIES

The nasal passages and nasopharyngeal airway can be clinically assessed by the ear, nose, and throat specialist using anterior and posterior rhinoscopy. The sagittal depth of the nasopharynx can also be evaluated on lateral skull radiographs. There were differing opinions, however, concerning the accuracy of this method in view of the fact that these radiographs can reflect the nasopharynx in only two dimensions. A number of authors on the other hand, have found this type of radiographic examination to be practical, having satisfactory results in children of all ages.

An investigation was carried out by Linder-Aronson[50] in an attempt to clarify the value of lateral skull and frontal radiographs as a means of evaluating nasal respiratory function. The following factors were selected for evaluation:

1. The relationship between the size of the adenoids as measured on lateral skull radiographs and judged clinically following posterior rhinoscope examination.

2. The relationship between the size of the adenoids as measured on lateral skull radiographs and nasal airflow measured in liters per minute.

3. The relationship between the size of the nasal airway as measured on frontal radiographs and nasal airflow measured in liters per minute.

4. The degree of nasal obstruction as judged on visual

examination of frontal radiographs compared with the nasal airflow measured in liters per minute.

Subsequent correlation analysis gave the following results:

1. A significant relationship between the size of the adenoids as measured on lateral skull radiographs and assessed clinically.
2. A negative relationship between the size of the adenoids as measured on lateral skull radiographs and the nasal airflow.
3. A significant relationship between the capacity of the nasal airway as measured on frontal radiographs and the nasal airflow.
4. A reasonable assessment of the nasal airflow by subjective evaluation of airway capacity from frontal radiographs.

He made the conclusion that lateral and frontal skull radiographs provided a satisfactory means of evaluating the dimensions of the nasopharynx and the capacity of the nasal airway, respectively.

Bresolin,et al[51] completed a cephalometric investigation of thirty allergic children, aged 6 to 12 years who had moderate-to-severe nasal mucosal edema on physical examination and who appeared to breathe predominantly through the mouth. They compared them to 15 children without allergy who had normal findings from nasal examination and who appeared to

breathe predominantly through the nose. The facial characteristics of children who were mouth breathers were as follows:

1. They had longer faces.
2. The faces were more retrognathic in lateral profile.
3. The mandibles had more obtuse gonial angles.
4. The palates were higher and narrower.
5. They were more likely to have posterior dental crossbites than children who breathed through the nose.

In Trask's study[29] they analyzed the effect of perennial allergic rhinitis on dental and facial skeletal characteristics. Twenty-five allergic children who were apparent mouth breathers, their 25 siblings who did not have the disease and were apparent nose breathers, and 14 nasal breathing control subjects were used in this study in an attempt to differentiate the facial characteristics most strongly determined by heredity from the facial structures more vulnerable to environmental influence-specifically, mode of breathing. A control group of nasal breathers was used to determine whether the sibling pairs had genetic predispositions to specific facial and skeletal characteristics.

Overall, the allergic children had longer, more retrusive faces than controls.These results confirm earlier reports that allergic rhinitis may be associated with mouth breathing and

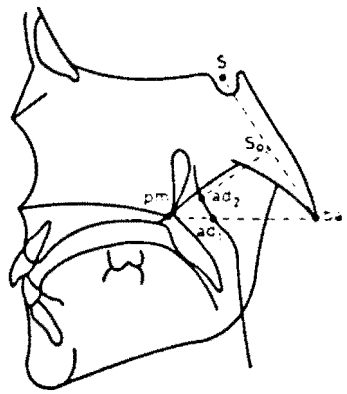
altered facial growth.

Linder-Aronson and Henriksson[50] compared the anteroposterior nasopharyngeal dimensions cephalometrically of 6 to 12 year old mouth breathers to nose breathers. The purpose of the study was to calculate the average anteroposterior size of the nasopharyngeal airway in children of this age group in order to obtain cephalometric standards. From these standards, it is possible to judge the extent by which mouth breathing may be obstructed.

Lateral radiographs were taken and evaluated by two independent examiners. Measurements were made to assess airway dimension and a test was used for calculating statistical differences between the groups.(Fig.15) The result showed that variable A1 and A2 gave a good indication of the anteroposterior size of the nasopharyngeal airway. This gave a more reliable indication of the need for an otologic examination. The standard values obtained in this study showed that an otologic examination of the nasopharyngeal space was to be recommended if the measured distance pm-ad1 or pm-ad2 was less than the present mean minus 1 SD for mouth breathers in the appropriate age group.(Fig.16,17,18).

The results also showed that when planning orthodontic therapy, in which it was desirable to assess the ability of the patient to breathe through the nose, a clinical record of the mode of breathing can be supplemented with

radiocephalometric data on the anteroposterior size of the nasopharyngeal airway. Furthermore, they found that the contour of the posterior nasopharyngeal wall could be satisfactorily assessed on lateral skull radiographs of children.



Reference points. pm = pterygomaxillary; s = sella turcica; ba = basion; s_{os} = the mid-point on the line joining s and ba; ad_1 = the intersection of the posterior nasopharyngeal wall and the line pm–ba; ad_2 = the intersection of the posterior nasopharyngeal wall and the line pm–s_{os}.

Fig.15 Airway dimension measurement by Linder-Aronson/Henrikson

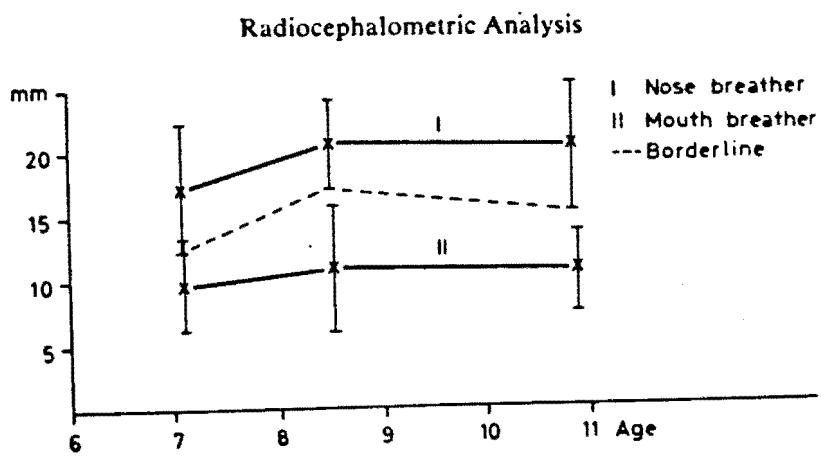


Diagram of means and SD for variable A1 (the distance pm–ad mouth breathers and nose breathers aged 6–12 years.

Fig.16 Diagram of airway dimension measurement by Linder-Aronson/Henrikson

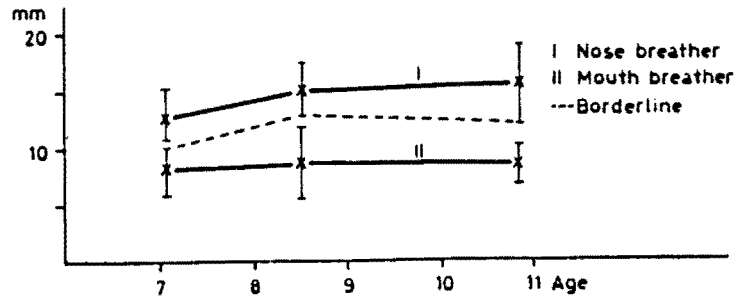


Diagram of means and SD for variable A2 (the distance pm–ad$_2$) for mouth breathers and nose breathers aged 6–12 years.

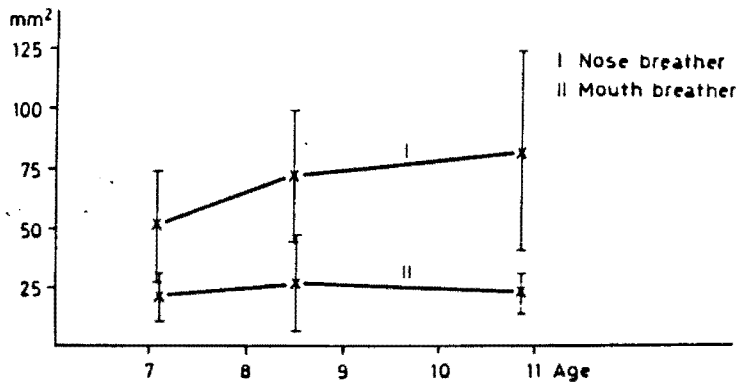


Diagram of means and SD for variable A3 (the area pm–ad$_1$–ad$_2$–pm) for mouth breathers and nose breathers aged 6–12 years.

Fig.17,18 Diagram of airway dimension measurement by Linder-Aronson/Henrikson

Radiographs of the nasopharynx were sometimes misinterpreted because of poor technical quality. A simple method of interpretations suggested by Cohen and Konak[52] was based upon measuring the airway immediately behind the upper part of the soft palate(Fig.19).If it was narrower than the width of the soft palate it was considered as markedly obstructed. When narrower than half of the soft palate, it was severely obstructed. When it was the same width as the soft palate, it is not narrowed.

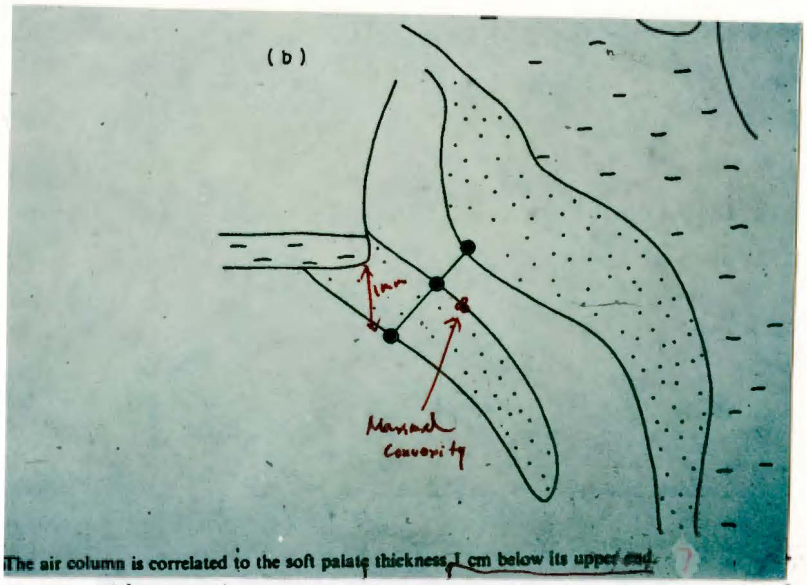


Fig.19 Airway dimension measurement by Cohn and Kónak[52]

In this study, he also showed six other methods of nasopharyngeal airway evaluation.(Fig.20) All methods showed good correlation and the present method was easy to use and has proven to be useful even in radiographs which other methods fail to interpret. This study also stressed the importance of evaluating the airway instead of the adenoidal thickness.

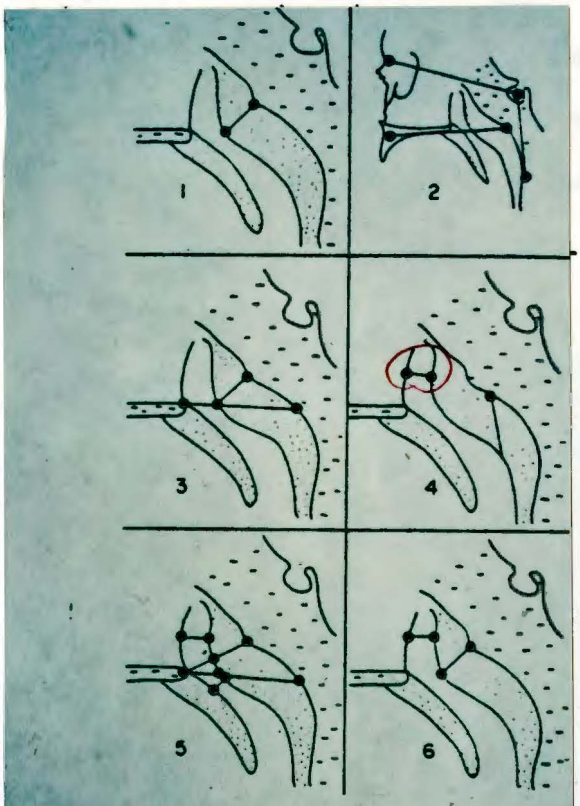


Figure 1 Graphical synopsis of 6 methods cited for measuring the size of adenoids: Johannesson,[1] Maran,[2] Fujioha, [3] Hibbert, [4] Sørensen[5] & Crepeau[6].

Fig.20 Graphic synopsis of six methods for measuring the size of adenoid

Hibbert and Whitehouse[34] evaluated the accuracy of radiology in the assessment of both adenoidal size and the size of the nasopharyngeal airway.

Seventy-six consecutive children who subsequently underwent adenoidectomy were reviewed. A lateral radiograph of the postnasal space was taken on the day before surgery.The area of the adenoid shadow on the radiograph was traced onto graph paper.(Fig.21). It has been observed that the posterior

wall of the maxillary antrum was in close approximation to the plane of the posterior choana. A line drawn at right angles to the adenoid shadow will intersect the line of the posterior wall of the antrum. The shortest line between these 2 points was considered to be the width of the nasopharyngeal airway.

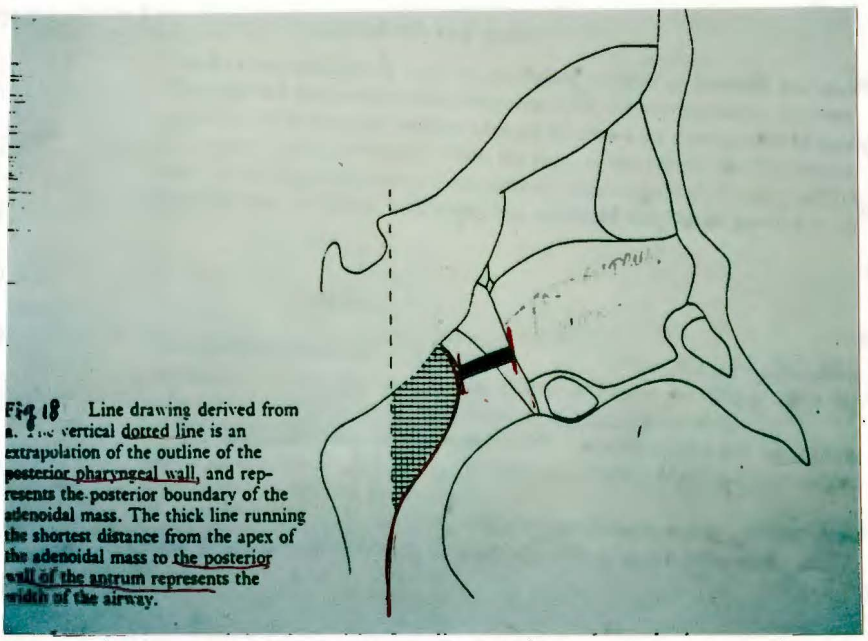


Fig.21 Airway measurement by Hibbert and whitehouse[34]

The adenoids were removed by a standard technique and they were washed, dried weighed, and their volume was measured by displacement.

The study showed that radiograms were an accurate method of assessing the size of the adenoid mass, in contrast to preoperative signs and symptoms which were poor predictors of adenoid weight. This study also indicated that it was the size

of the adenoid rather than the size of the nasopharynx which was important of the impairment of the airway.

Hibbert and Stell[53] compared the adenoid of two groups of children: those selected for adenoidectomy and those who presented as normal control group.

The method to evaluate the size of adenoid and nasopharyngeal airway was previously described.

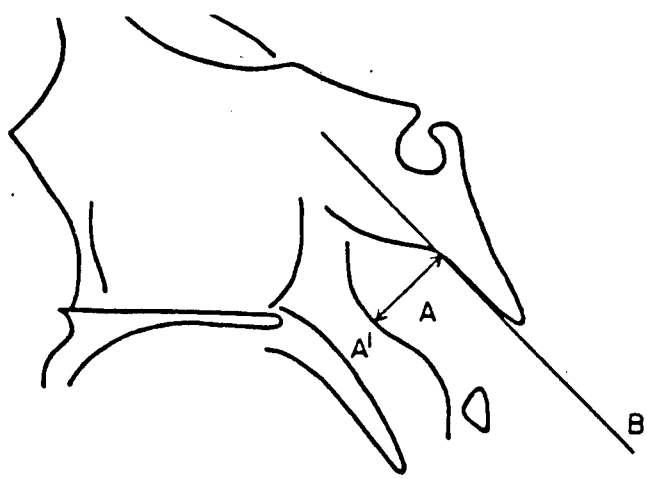
This study showed that in a series of children selected for adenoidectomy the radiographic area of the adenoid did not differ significantly from that in normal children. That meant the adenoid in children selected for adenoidectomy was no larger than in normal children. However, the children selected for adenoidectomy have a significantly smaller nasopharyngeal airway than the same measurement in normal children.

In this series of normal children studied it was shown that the radiographic area of the adenoid does not increase with age, though the nasopharyngeal airway does. The increase of the nasopharyngeal airway must therefore be due to an increase in the anterior-posterior dimension of the nasopharynx as the child grows. It was suggested that in children below 70 months an airway of 2 mm or less can be considered abnormal and in children over 70 months an airway of 3 mm or less can be considered abnormal.

Fujioka, Young & Girdany[54] thought the absolute size of the adenoids and the size and shape of the nasopharyngeal

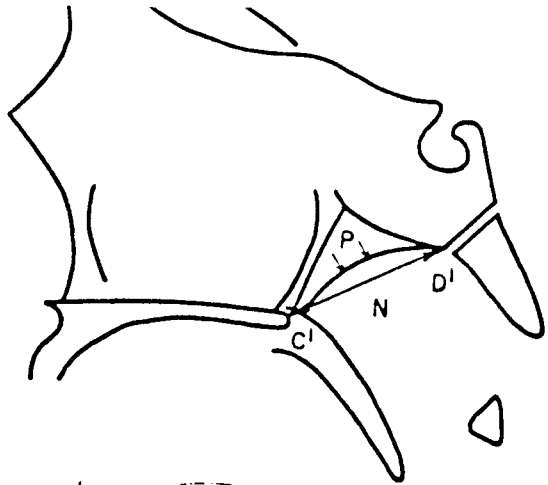
space were major factors that determine nasopharyngeal obstruction. They described an adenoidal-nasopharyngeal ratio (AN ratio) derived from linear measurements on lateral radiographs of the nasopharynx. The ratio of these two sizes can provide a simple arithmetic measure of nasopharyngeal obstruction.

Lateral radiographs of the nasopharynx of 1,398 children between ages 1 month and 16 years were reviewed and the AN ratio were calculated, tabulated, and statistically analyzed(Fig.22,23). The 143 lateral nasopharyngeal radiographs of 92 patients and their adenoidal size and nasopharyngeal air patency had been estimated visually by experienced observers and classified to the AN ratio (Fig.24).



Adenoidal measurements. "A" represents distance from A', point of maximal convexity, along inferior margin of adenoid shadow to line B, drawn along straight part of anterior margin of basiocciput. "A" is measured along line perpendicular from point A' to its intersection with B.

Fig.22 Adenoid measurement by Fujioka,Young and Girdany[54]



(Nasopharyngeal measurement) N' is distance between C', posterior superior edge of hard palate, and D', anteroinferior edge of sphenobasioccipital synchondrosis. When synchondrosis is not clearly visualized, point D' can be determined as site of crossing posteroinferior margin of lateral pterygoid plates P and floor of bony nasopharynx.

Fig.23 Nasopharynx measurement by Fujioka, Young and Girdany[54]

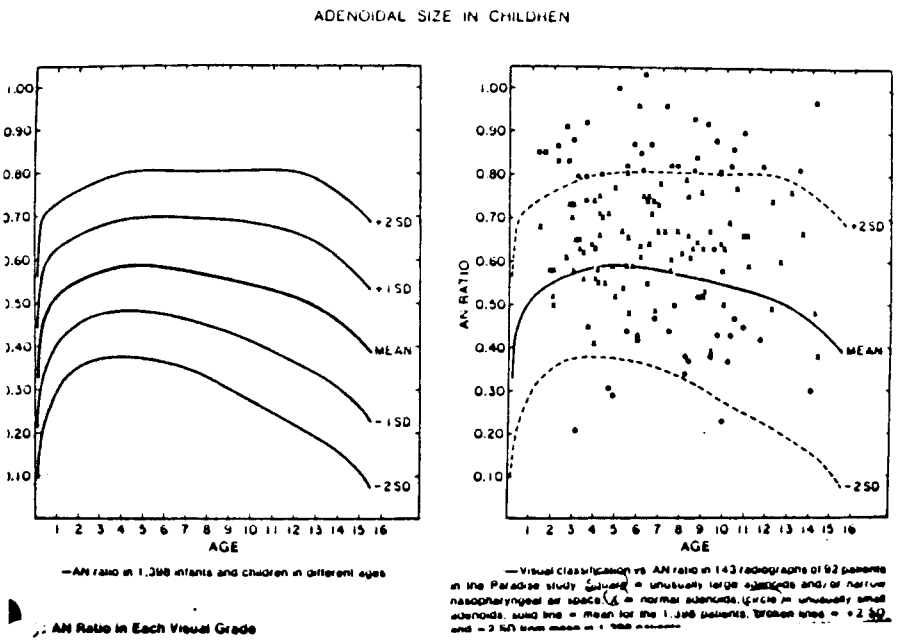


Fig.24 Adenoid/Nasopharynx ratio by Fujioka, Young and Girdany[54]

The results were as follows:

1. The frequency distribution of the AN ratios for each gender and in each age group followed the expected curvature of a normal distribution.There were no statistically significant differences on AN ratio for gender in any age group.
2. The assessment of visualized classification of the size of the adenoid and nasopharyngeal space was in general agreement with the statistical analysis.
3. For Practical purposes, a value of the AN ratio greater than 0.80 may be considered indicative of enlarged adenoids.

(F). Treatment, effect of adenoidectomy.

Tonsillectomy and adenoidectomy play a certain role in the treatment of certain infectious and inflammatory diseases of the upper airway.[55]

Linder-Aronson et,al[56] did a study on the mandibular growth direction following adenoidectomy. The adenoidectomy sample initially showed significantly longer lower face heights, steeper mandibular plane angles, and more retrognathic mandibles than the matched controls.
Analysis showed the following results:
1. During the 5 years after adenoidectomies, the girls had a more horizontal mandibular growth direction than did the

female controls.

2. A corresponding but not significant trend was found for the boys.

3. The growth directions were significantly more variable for both boys and girls after adenoidectomies than for controls during the five-year growth period.

4. The mean airflow through the nose increased for both sexes 1 year after adenoidectomy to values equal to the initial values for the control.

Respiratory function and its effects on craniofacial growth was evaluated by Linder-Aronson[47]. Longitudinal results of five years post adenoidectomy were presented to examine the effects on the dentition and facial skeleton with a change in the mode of breathing.

The sagittal depth of the bony nasopharynx, as measured from the pterygomaxillary point to basion, changed in children who became mouth breathers after removal of their adenoids as well as that in the control children. The greatest change occurred in the first year post-operatively in the group of children whose adenoids had been removed, During the following four years, the
increase in this group was similar to the controls.

The angle between the mandibular plane and the palatal plane changed due to the change of the mode of breathing. The change during the first year was not significant but by the

fifth year post-operatively, a significant change was noted. A correlation analysis between reductions in the ML/N angle and lower facial height was found to be significant at the .001 level.

Linder-Aronson[57] in the study of the effects of adenoidectomy on mode of breathing stated that the multiple regression analysis clearly supported the hypothesis that enlarged adenoids give rise directly or indirectly to mouth breathing and that in most cases the individual changes to nasal breathing after adenoidectomy. The multiple regression analysis also showed that the size of the nasopharynx was of importance in this respect.

He concluded that in any case, improved adeno-tonsillar function and lessened inferior-turbinate hypertrophy will improved the upper airway and further reduced the effect of a large tongue on the developing tissues or structures.

Linder-Aronson & Lindgren[58] stated that the narrow maxilla may be treated by surgical or orthodontic expansion. The mid-palatal suture split will decrease the higher to normal nasal resistance.

Guenthner,et al[59] studied the effect of Le Fort I maxillary impaction on nasal airway resistance. The nasal airway resistance was determined by means of a universal

active rhinomanometric technique. Contrary to the predicted negative effects of maxillary superior movement on nasal airway function, there was a statistically significant improvement in nasal airway resistance after maxillary superior movement.

When abnormal growth and development at the level of the stomatognathic system was recognized at an early stage, and is related to a large tongue with upper airway obstruction[44]. It may be wise to act medically or surgically to normalize the enlarged tissue mass of the tongue before its full development(at about age eight years).

Improved adenotonsillar function and lessened inferior-turbinate hypertrophy improved the upper airway and further reduce the effect of a large tongue on the developing tissues or structures of the oral cavity. Thus, the pathogenicity of any given tongue was related to the status of the airways at a given time. A simple volumetric correction of the hypertrophied tissues, when effected early, may be sufficient to deactivate the pathogenicity of the tongue and normalize the growth patterns of the face.

It was well established that the jaws were vulnerable to environmental factors that may have detrimental effects.[60] Hypertrophic tonsils causing forward tongue displacement may have similar effects. The tendency for self-correction of dental irregularities after removal of various detrimental

factors can be interpreted as a definite indication that a cause-and-effect relationship may exist.Similarly, elimination of nasal airway interferences followed by a change from oral to nasal respiration may result in improvement of certain aspects of facial and dental deviations.

Because of the individual variation response, adenotonsillectomy or other airway surgery should not be done in a very young child to prevent future unfavorable craniofacial development because this may never ensue.[60] Moreover, we believe that surgery should be considered only when the characteristic deviations are manifested. Hoverer. they stress that children who demonstrate features associated with open-mouth posture should receive appropriate airway treatment and facial growth management to prevent undesirable growth patterns from persisting and progressing.

Chapter III

MATERIAL AND METHOD

MATERIALS:

A. Experimental group:

1. There were nineteen subjects in this study. They were all referred to one Ear, Nose and Throat specialist by physicians on the basis of a history of persistent nasal respiratory obstruction which was confirmed by physical examination. Obstruction was still present after administration of vasoconstrictor spray. All of them had obstructive adenoids and were scheduled for adenoidectomy after the study records were obtained. The subjects' general medical histories, physical conditions and mouthbreathing situations were evaluated and understood employing the history form, examination form, and case form used in this Ear, Nose and Throat clinic.(Fig.25,26,27)

All subjects were caucasians. There were four subjects in three age group- two were males and two were females; three in four age group- two males and one female; one in five age group- one male; four in six age group- one male and three females; and seven in seven age group- three males and four females.(Fig.28)

2. The subjects of this research were referred to an ENT specialist from physicians and pediatricians to eliminate any bias for certain facial characteristics that might influence

the results. Because general dentists and pedodontist were aware of the association between airway obstruction and facial deformity, referral by them was excluded.

FAMILY HISTORY	Age	State of Health	If deceased, cause of death.	Age at death
Father				
Mother				
Spouse				
Brothers (B)				
Sisters (S)				
Children				
Sons (S)				
Daughters (D)				

Check diseases blood relatives have had. (If checked—state relationship)

- ☐ High Blood Pressure _____
- ☐ Heart Disease _____
- ☐ Stroke _____
- ☐ Kidney Disease _____
- ☐ Epilepsy (Convulsions) _____
- ☐ Jaundice _____
- ☐ Migraine _____
- ☐ Tendency to Bleed _____
- ☐ Diabetes _____
- ☐ Hay Fever _____
- ☐ Cancer _____
- ☐ Tuberculosis _____
- ☐ Nervousness _____
- ☐ Nervous Breakdown _____
- ☐ Ulcers _____
- ☐ Other _____

Please list any illnesses you have had and give the dates:

_____ Date _____
_____ Date _____
_____ Date _____

Please list previous surgeries you have had--type and date:

Type _____ Date _____
_____ _____

Please list any allergies or reactions you have had to medications, foods, cosmetics, plants, etc:

_____ _____
_____ _____

Please list any medications that you are taking including aspirin, laxatives, hormones, tranquilizers, cortisone, blood pressure pills, or other: _____ _____
_____ _____

Habits — How much per day or per week?

- Coffee _____ _____
- Tea _____ _____
- Tobacco _____ _____
- Alcohol _____ _____
- Beer _____ _____
- Wine _____ _____
- Whiskey _____ _____

REPORT OF MEDICAL HISTORY

Fig.25 History form used in the Ear,Nose and Throat clinic

Please check any of the following complaints which presently trouble you:

- ☐ Head colds
- ☐ Chest colds
- ☐ Sore throats
- ☐ Sinus trouble
- ☐ Nose bleeds
- ☐ Cough
- ☐ Hay Fever, Asthma
- ☐ Asthma
- ☐ Excessive perspiration
- ☐ Loss of weight
- ☐ Obesity
- ☐ Joint pains
- ☐ Blue moods
- ☐ Inability to concentrate
- ☐ Lack of self confidence
- ☐ Nervousness
- ☐ Sleeplessness
- ☐ Back trouble
- ☐ Abdominal pain
- ☐ Painful urination
- ☐ Shortness of breath
- ☐ Heart pain
- ☐ Skin trouble
- ☐ Visual difficulties
- ☐ Earache
- ☐ Discharge from ears
- ☐ Deafness
- ☐ Poor appetite
- ☐ Discomfort after meals
- ☐ Nausea-vomiting
- ☐ Constipation
- ☐ Diarrhea
- ☐ Speech difficulty
- ☐ Convulsions, fits
- ☐ Fainting spells
- ☐ Headaches
- ☐ Dizziness
- ☐ Thyroid disturbances
- ☐ Fever
- ☐ Anemia
- ☐ Rectal bleeding
- ☐ Frequent urination
- ☐ Easy fatigue
- ☐ Heart pounding
- ☐ Hives

Past medical illnesses--give approximate age at which you had any of the following illnesses:

- _____ German measles
- _____ Measles
- _____ Mumps
- _____ Chicken pox
- _____ Scarlet fever
- _____ Whooping cough
- _____ Diptheria
- _____ Typhoid fever
- _____ Asthma
- _____ Diabetes
- _____ Stomach trouble
- _____ Appendicitis
- _____ Hernia (rupture)
- _____ Colitis
- _____ Concussion
- _____ Kidney trouble
- _____ Albumin in urine
- _____ Sugar in urine
- _____ Malaria
- _____ Undulant fever
- _____ Hepatitis
- _____ Poliomyelitis
- _____ Influenza
- _____ Pneumonia
- _____ Pleurisy
- _____ Tuberculosis
- _____ Tumor or cancer
- _____ Radioactive exposure
- _____ Nervous breakdown
- _____ Epilepsy
- _____ Venereal disease
- _____ Jaundice
- _____ Dysentery
- _____ Tendency to bleed
- _____ Infectious mononucleosis
- _____ Rheumatic fever
- _____ St. Vitus dance
- _____ Tonsillitis
- _____ Discharge from ears
- _____ Mastoid infection
- _____ Sinus trouble
- _____ Heart trouble
- _____ High blood pressure
- _____ Hay fever

Are you subject to distressing periods of mental depression? _____

Have you ever been treated for a nervous or mental disorder? _____

Do you consider yourself more nervous than the average person? _____

Have you any apprehension in regard to your health? _____

Have you had psychotherapy? _____

Have you lived with anyone with tuberculosis? _____

List any states and countries in which you have lived _____

Women check:

Menstruation: Age at onset _____ Periods regular every _____ days Irregular _____ Duration _____ Days

Amount: Small _____ Medium _____ Profuse _____ Pain _____ days Character of pain _____ Cramping _____ Dull

Backache _____ Go to bed _____ Stay home _____ Vaginal discharge? _____ Color _____

Present or past treatment of menstrual disorders? If so, what _____

_____

Pregnancies: Dates _____

Complications _____

Please state the reason why you require medical care and include any additional information that would be helpful in the diagnosis or management of this problem. _____

_____

REPORT OF MEDICAL HISTORY

Page 2

Fig.26 Examination form used in the Ear,Nose and Throat clinic

NAME: _____ Date: _____

PRESENT ILLNESS and SYMPTOMS: _____

ALLERGIES or SENSITIVITIES: _____ None Known: ____

RINNE

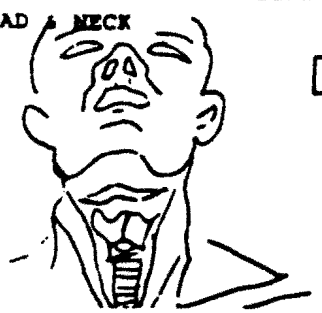
	Normal	No Exam
R	☐	☐
L		

RINNE

NOSE

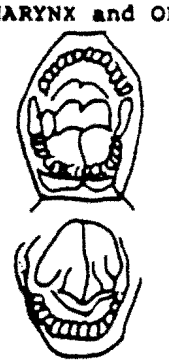
NASOPHARYNX

HEAD & NECK



PHARYNX and ORAL CAVITY

	Normal	No Exam
	☐	☐



LARYNX

	☐	☐

AUDIO ☐ ☐

IMPRESSION:

PLAN:

Richard F. Bulger, M.D.
James E. Rejowski, M.D.

Next Visit _____

Fig.27 Case form used in the Ear, Nose and Throat clinic

3. Subjects were given a vasoconstricting spray to differ between mucosal vs adenoid blockage in Ear, Nose and Throat examination. It could be assumed that the subjects had persistent and obligatory oral respiration rather than temporary and transitory mouthbreathing histories because nearly all subjects had adenoids and were scheduled for adenoidectomy soon afterwards. From parents' anamnestic information and the lateral head X-Ray films, it was also confirmed that most subjects were suffering from mouthbreathing due to adenoids.

B. Control group:

Nineteen lateral head X-Ray films from the Broadbent-Brush Growth Study Center in Case Western Reserve University were chosen to match the experimental group in race, age, and sex. Due to the fact that adenoidectomy was very prevalent, even as a routine surgery for young children when the Broadbent-Brush Growth Study was being conducted in the nineteen thirties, all the subjects in the control group were chosen by their medical histories of either those who had their adenoids removed very early at ages of three or four; or those who had never had adenoidectomy. For those who had early adenoidectomy it is presumed they did not have problems of chronic nasal respiratory obstruction later on as well as those who had never had adenoidectomy. Subjects having adenoidectomies after six or seven years of age were excluded from this control group due to the consideration that they

might have had adenoidal obstruction and mouthbreathing problems during their early ages but waited until later to have adenoidectomy.

Experimental group

Source: Samples were referred to one E.N.T specialist, and then referred to Orthodontic department for taking lateral head X-Ray plate.

Number: 19 Race: Caucasian

		Male	Female
Age	3	2	2
	4	2	1
	5	1	0
	6	1	3
	7	3	4

History: 1, Persistent nasal respiratory obstruction.
2, Obstructive adenoids and scheduled for adenoidectomy.

Fig. 28 Sample of the experimental group

Control Group

Source: Lateral head X-Ray films from the Broadbent-Brush Growth study Center in Case Western Reserve U.

Number: 19 Race: Caucasian

		Male	Female
Age	3	2	2
	4	2	1
	5	1	0
	6	1	3
	7	3	4

History: 1, Early removed adenoids at age three or four.
2, Never had adenoid removed.

Fig.29 Sample of the control group

METHODS:

A. Experimental group:

1. Cephalometric radiographs for all subjects were taken on a standard cephalometer in the Orthodontic Department.(Fig.30,31) The sagittal plane of the head was five feet from the X-Ray source and 15 centimeter to the X-Ray film cassette. The X-Ray machine was set at seventy-seven KVP, 1/6 second, and 4.5 milliamperage. The radiographs were taken with the subjects' heads in upright natural position, their teeth in centric occlusion and their lips at rest.

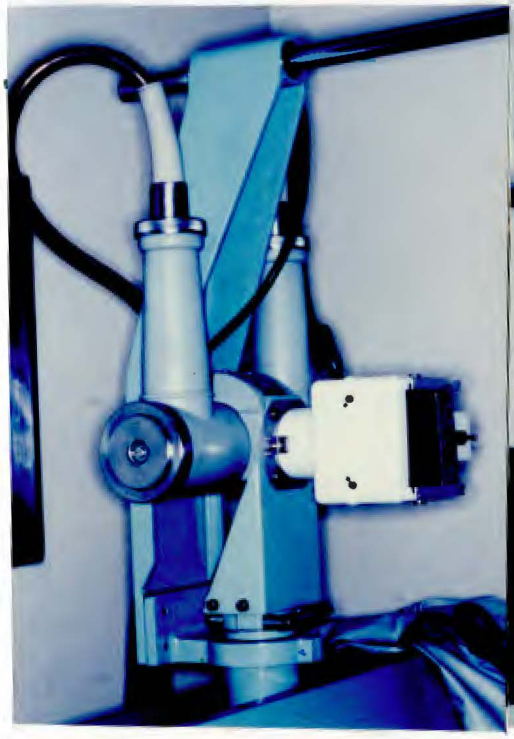


Fig. 30

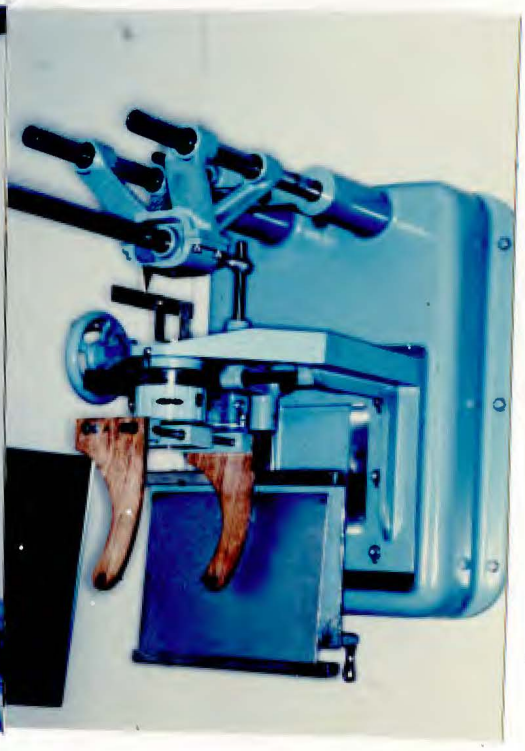


Fig. 31

Standard cephalometric machine

2. Tracings of the radiographs were made on 0.003-inch matte acetate paper with an 0.5-mm pencil. Soft-tissue outlines were excluded to eliminate measurement bias created by lip posture.

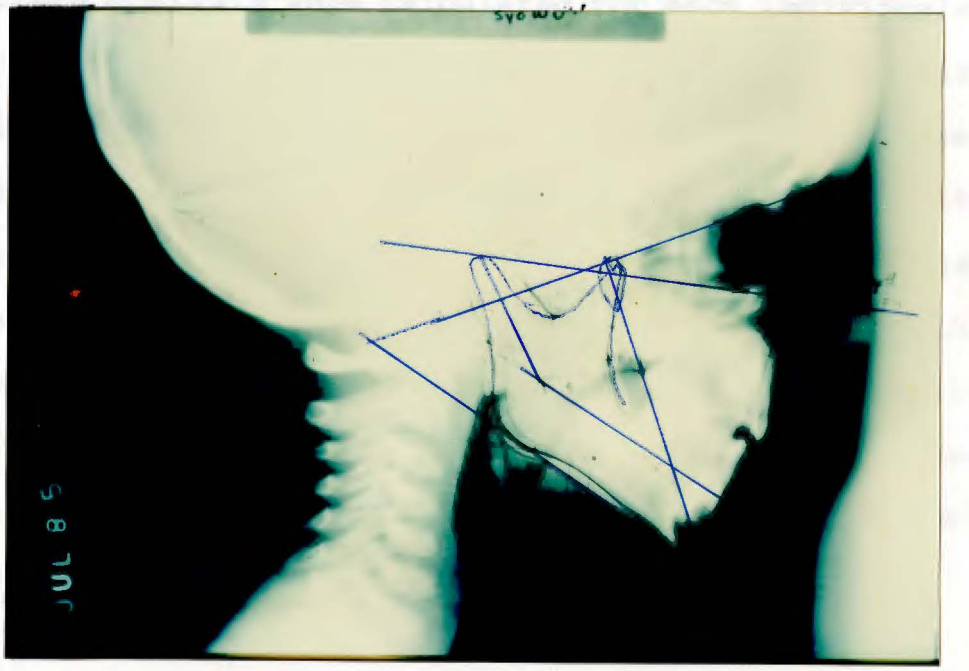


Fig. 32 Example of tracing of the head X-Ray plate

3. Skeletal landmarks (Fig.33) and Planes (Fig.34) necessary for Ricketts facial pattern analysis were identified, and selected by two orthodontists to produce five angular measurements i.e. facial axis, facial depth, mandibular plane angle, mandibular arc and lower facial height. (Fig.35) Those radiographic and skeletal landmarks needed for the Ricketts facial pattern analysis are illustrated. (Fig.36,37,38,39,40)

4. Those cephalometric measurements for the facial pattern analysis were calculated for each individuals' facial pattern according to Ricketts' facial pattern analysis method. (Fig.41,42,43) The norm of each of the above measurements for each age group were extrapolated from original Ricketts' norms due to the fact that the stature growth rate is almost constant from young age to puberty according to growth studies from the National Center for Health Statistics,1979. (Fig.44,45,46) The standard deviations of these five measurements were kept the same as in the Ricketts' facial pattern analysis.

DEFINITIONS OF ANATOMIC LANDMARKS

(USED IN RICKETTS FACIAL PATTERN ANALYSIS)

Points	Definition
Na	The suture between the frontal and nasal bones.
Or	The lowest point on the average of left and right infraorbital margin.
Pr	The highest point on the average of the left and right superior surface of the external auditory meatus.
Pg	The most anterior point on the mandible in the midline, determined by a tangent through nasion.
Gni	a point at the intersection of the facial and mandibular planes.
Me	The most inferior point on the symphyseal outline.
Ba	The most inferior posterior point on the anterior border of the foramen magnum.
Ptv	Intersection of inferior border of foramen rotundum with posterior wall of pterygomaxillary fossa.
Ans	The most anterior point on the maxilla at the level of the palate.
Pm	Point on the anterior border of the symphysis between B point and Pogonion where the curvature changes from concave to convex.
Sn	A point located at the center and most inferior aspect of the sigmoid notch of the ramus of the mandible.
Ra	The deepest point on the curve of the anterior border of the ramus.
Dc	The midpoint between the anterior and posterior border of the condyle intersected with Na-Ba line.
Xi	The geometrical center of the ramus. Lacated as shown on fig.___.

Fig.33 Definition of anatomical landmarks for Ricketts facial pattern analysis

DEFINITION OF CEPHALOMETRIC PLANES

(USED IN RICKETTS FACIAL PATTERN ANALYSIS)

Line	Description
Po-Or	Frnakfort Horizontal : a horizontal plane running through the right and left porion and orbitale.
Me-tangent	Mandibular plane : A line at the olwer border of the mandible tangent to the gonion angle and Me.
N-Pg	Facial plane : A line from nasion to pogonion.
N-Ba	Dividing line : between the face and the cranium.
Xi-Dc	Condylar axis : to describe the morphology of the mandible.
Xi-Pm	Corpus axis : to evaluate the morphology of the mandible.

Fig. 34

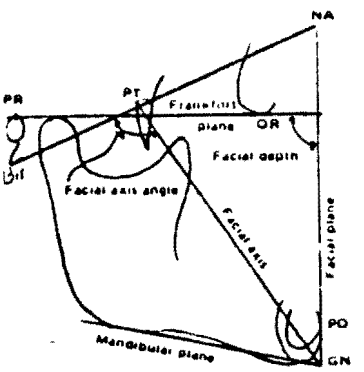
DEFINITION OF CEPHALOMETRIC MEASUREMENTS

(USED IN RICKETTS FACIAL PATTERN ANALYSIS)

Measurement	Description
Facial axis	The angle between the Facial axis and Basion-Nasion plane.
Facial depth	The angle between the Frankfort plane and the facial plane N-Po.
Mandibular plane angle	The angle betwen the Mandibular plane and the Frankfort plane.
Lower facial Height	The angle from Ans to Xi To Pm.
Mandibular arc	The angle between the Condylar axis and the Corpus axis.

Fig.35

FIELD V – CRANIO-FACIAL RELATION



32. FACIAL DEPTH: The angle between the facial plane and Frankfort plane. Downs facial angle.

CLINICAL NORM: 87° at age 9. Increases 0.33° per year.

CLINICAL DEVIATION: ±3°

INTERPRETATION: Locates the chin horizontally. Determines if the skeletal Class II or Class III is due to the mandible.

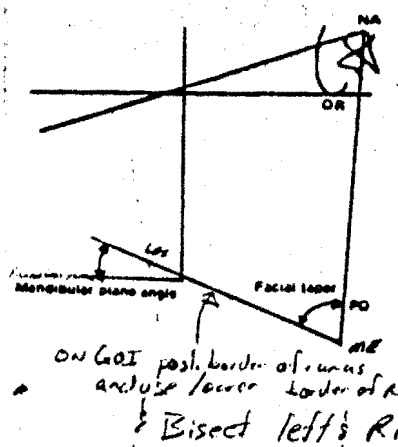
34. FACIAL AXIS: The angle between the facial axis and Basion Nasion.

CLINICAL NORM: 90°

CLINICAL DEVIATION: 3.5°

INTERPRETATION: The direction of growth of the chin and the molars. Expresses the ratio of facial height to depth.

M-7



35. FACIAL TAPER: The mandibular plane measured to the facial plane.

CLINICAL NORM: 68°

CLINICAL DEVIATION: 3.5°

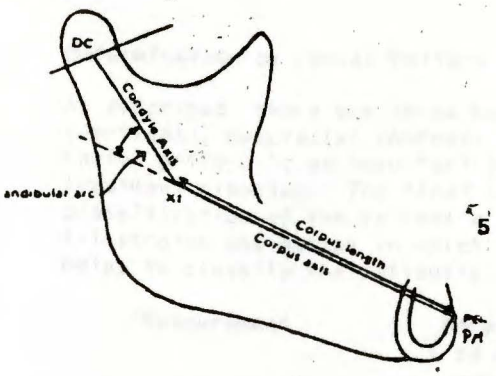
39. MANDIBULAR PLANE ANGLE: Measured to Frankfort horizontal.

CLINICAL NORM: 26° at age 9. Decreases 0.3° per year.

CLINICAL DEVIATION: 4.5°

INTERPRETATION: "High" mandibular plane implies that skeletal open bite is due to the mandible. "Low": mandibular plane implies skeletal deep bite is due to the mandible.

Fig.36, 37, Definition, clinic norm and clinic deviation of facial axis, facial depth and mandibular plane angl used in Ricketts facial pattern analysis



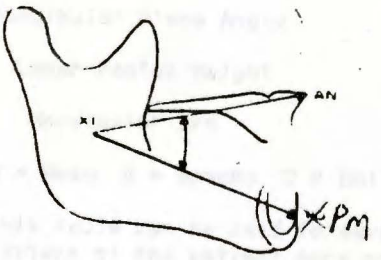
(50) **MANDIBULAR ARC:** The angle between the corpuse and condyle axes.

CLINICAL NORM: 26° at age 8½. Increases 0.5° per year.

CLINICAL DEVIATION: 4°

INTERPRETATION: High angles are square mandibles – deep bites, sometimes prognathic patterns. Low angles tend to open bites, retrognathic.

5



(15) **LOWER FACE HEIGHT:** The angle from anterior nasal spine to the center of the ramus (Xi) to Pogonion (Pm).

CLINICAL NORM: 47°. Stays constant with age.

CLINICAL DEVIATION: ±4.0°

INTERPRETATION: Describes the divergence of the oral cavity with growth. High values are "open bite" skeletally - low values "deep bite."

Fig.38,39 mandibular arc and lower facial height used in Ricketts facial pattern analysis

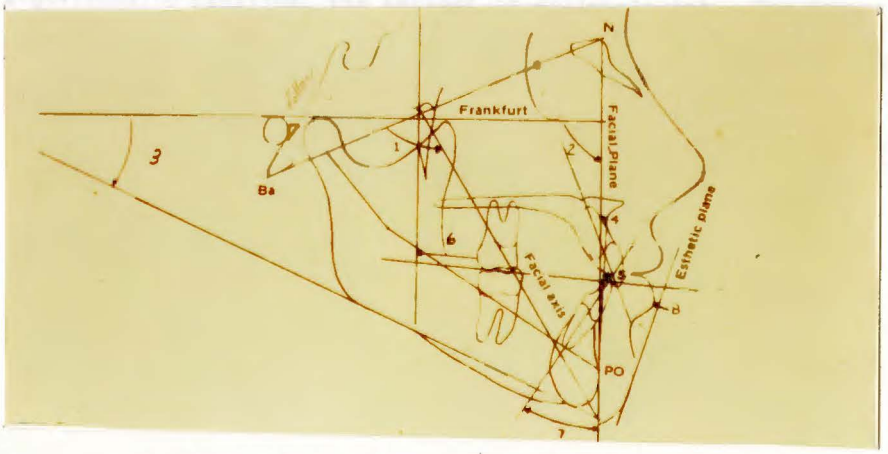


Fig.40 Tracing of the five measurements for facial pattern analysis

Determination of Facial Pattern

As described there are three basic facial patterns, dolichofacial (vertical), mesofacial (normal) and brachyfacial (horizontal). Facial pattern is an important factor in growth prediction and in treatment planning. The first step in craniofacial diagnosis is classification of the patient's facial type. The following diagram illustrates the manner in which the magnitude of these measurements helps to classify the patient's facial type.

Measurement	More than 1 cd below norm	Within 1 cd of norm	More than 1 cd above norm
Facial Axis	D	M	B
Facial Depth	D	M	B
Mandibular Plane Angle	B	M	D
Lower Facial Height	B	M	D
Mandibular Arc	D	M	B

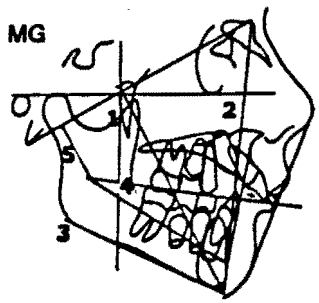
M = Meso B = Brachy D = Dolicho cd = Clinical Deviation

This table can be used to develop a scheme for describing the facial pattern of the patient more precisely.

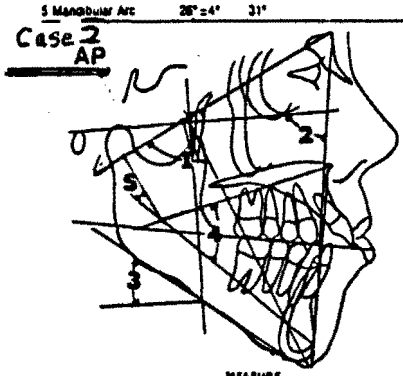
For each of the five facial classification measurements, the number of clinical deviations from the norm is calculated. All measurements which are more dolichofacial than the norm are given a minus sign. All measurements which are more brachyfacial are assigned a plus. The five signed clinical deviations are then averaged. The resultant number is called Vertical Description (amount of vertical growth). If Vertical Description is significantly negative, the patient is dolichofacial. The larger the negative number, the more dolichofacial the patient. Similarly, a high positive number indicated and extremely brachyfacial patient. A useful descriptive guideline for using Vertical Description appears below.

Facial Pattern	Severe Dolicho	Dolicho	Mild Dolicho	Meso	Brachy	Severe Brachy
Clinical Deviation	-2.0	-1.0	-0.5	0	0.5	1.0

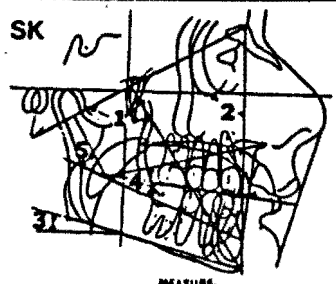
Fig.41 Determination of Ricketts facial pattern analysis



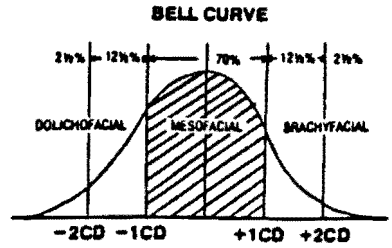
FACTOR	MEANS	MEASURE-MENT	DOLICHO	MESO	BRACHY
1 Facial Axis	90° ± 3°	88°		•	
2 Facial Angle	87° ± 3°	85°		•	
3 Mandibular Plane Angle	26° ± 4°	24°		•	
4 Lower Facial Height	47° ± 4°	44°		•	
5 Mandibular Arc	26° ± 4°	31°			•



FACTOR	MEANS	MEASURE-MENT	DOLICHO	MESO	BRACHY
1 Facial Axis	90° ± 3°	85°	•		
2 Facial Angle	87° ± 3°	82°	•		
3 Mandibular Plane Angle	26° ± 4°	37°	••		
4 Lower Facial Height	47° ± 4°	57°	••		
5 Mandibular Arc	26° ± 4°	19°	••		



FACTOR	MEANS	MEASURE-MENT	DOLICHO	MESO	BRACHY
1 Facial Axis	90° ± 3°	98°			••
2 Facial Angle	87° ± 3°	91°			•
3 Mandibular Plane Angle	26° ± 4°	13°			••
4 Lower Facial Height	47° ± 4°	35°			••
5 Mandibular Arc	26° ± 4°	36°			••



Together, these five angles determine whether the facial pattern is Meso-, Brachy-, or Dolichofacial. On a Bell curve, the middle section (representing one clinical (or standard) deviation on either side of the mean) is the range of Mesofacial patterns. Approximately 70% of the malocclusions that we treat fall in the Mesofacial range. Approximately 12½% fall on the Brachyfacial side and 12½% on the Dolichofacial side, one additional clinical deviation from the mean. This leaves approximately 2½% on each side, which are extreme Brachy- or extreme Dolichofacial, more than two clinical deviations from the mean.

Three different faces are presented to demonstrate how the five factors are used to describe the face.

1. MG is a Mesofacial pattern with a Brachyfacial mandible.
2. AP is a severe Dolichofacial pattern or vertical grower.
3. SK is an extreme Brachyfacial or horizontal growth pattern.

It is important to establish what the facial type is, because the reaction to treatment mechanics and the stability of the denture is dependent upon the analysis of the facial pattern. For example, Brachyfacial patterns show a resistance to mandibular rotation during treatment and can accept a more protrusive denture, whereas Dolichofacial patterns tend to open during treatment and require a more retracted denture in order to assure posttreatment stability. Thus, certain expectations from treatment may be modified with reference to facial type.

Fig.42 Example of determination of Ricketts facial pattern analysis

Facial pattern analysis calculation

Example:

Case 2, (AP)

	Factor	Mean	Measure-ment	Calculation
1	Facial Axis	90^0+-3^0	85^0	(85-90) ÷ 3 = 1.67 (-)*
2	Facial Angle	87^0+-3^0	82^0	(82-87) ÷ 3 = 1.67 (-)
3	Mandibular plane angle	26^0+-4^0	37^0	(37-26) ÷ 4 = 2.75 (-)
4	Lower facial Height	47^0+-4^0	57^0	(57-47) ÷ 4 = 2.5 (-)
5	Mandibular Arc	26^0+-4^0	19^0	(19-26) ÷ 4 = 1.75 (-)

Total = 10.34 (-)

Devided by 5

= 2.06 (-)

(On the Dolicofacial pattern)

* Each "-" means the measurement is on the Dolicofacial side.

Fig.43 Example of calculation of Ricketts facial pattern analysis

Fig.44 Life size growth chart from Nation Center for Health Statistics

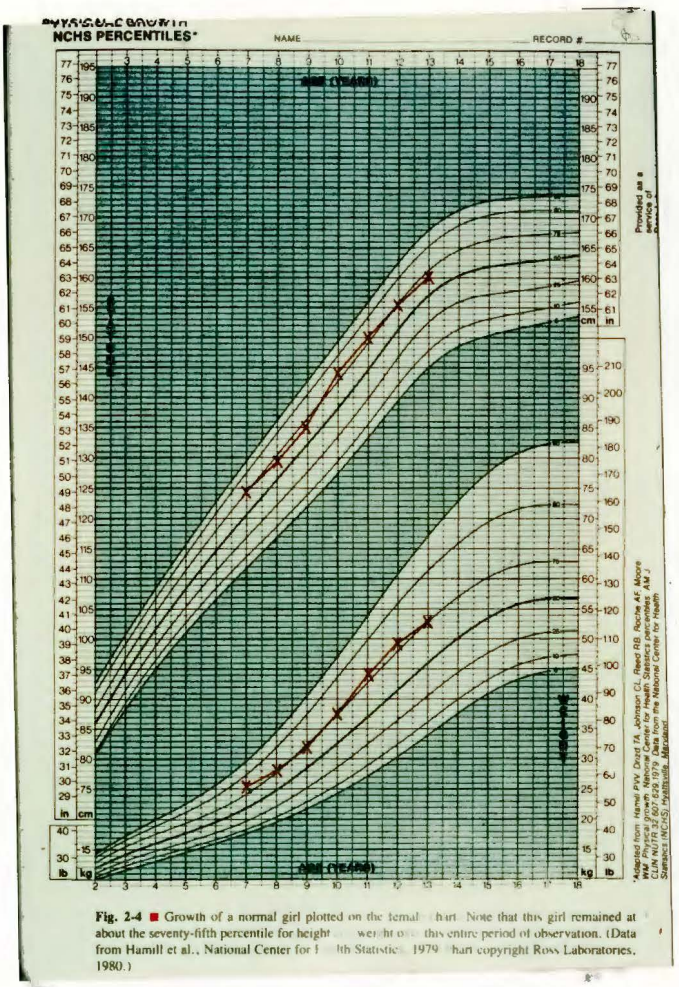


Fig. 2-4 ■ Growth of a normal girl plotted on the female chart. Note that this girl remained at about the seventy-fifth percentile for height and weight over this entire period of observation. (Data from Hamill et al., National Center for Health Statistics, 1979. Chart copyright Ross Laboratories, 1980.)

Fig.45 Growth chart,female, National Center for Health Statistics

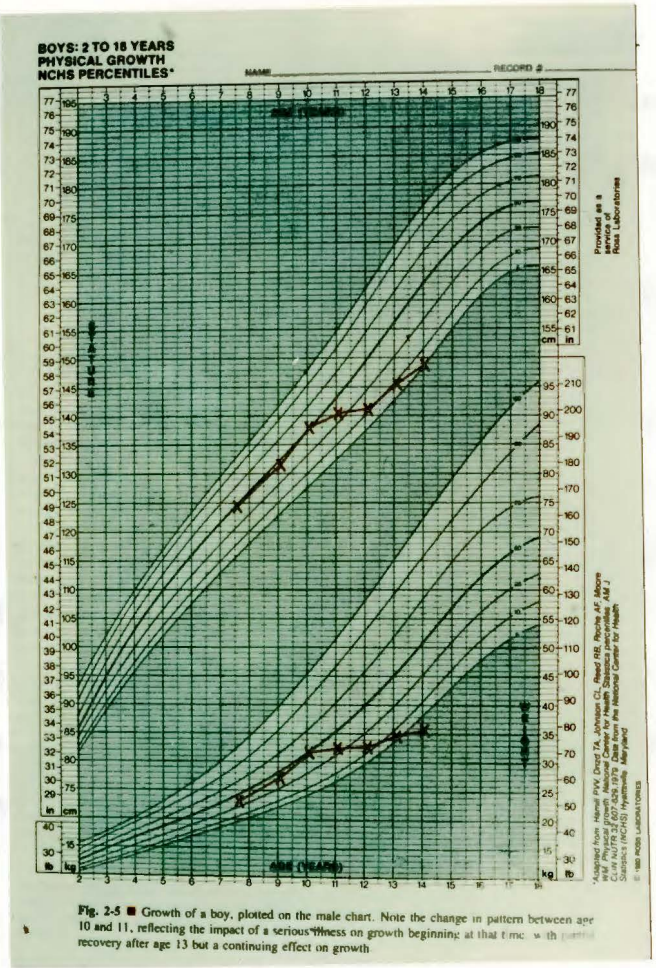


Fig. 2-5 ■ Growth of a boy, plotted on the male chart. Note the change in pattern between age 10 and 11, reflecting the impact of a serious illness on growth beginning at that time with partial recovery after age 13 but a continuing effect on growth.

Fig.46 Growth chart,male,National Center for Health Statistics

B. Control group:

1. By using a set of preformed templates supplied by the Broadbent-Brush Growth Center to cover on the original lateral head plates of the control group, the machine porions were converted to anatomical porions which would be used in the Ricketts' facial pattern analysis. Each different templet was fabricated according to the different types of ear rods and head fixer poles of the X-Ray machines used in different years during that Growth Study.

2. The facial patterns of all the tracings of the lateral head plates in the control group were also calculated following the same method as the experimental group.

3. The standard templates of the Broadbent-Brush Growth Study for each age group from three to seven were also traced and the facial patterns were calculated following the same method as above.

CHAPTER IV

RESULT

Statistical analysis:

This study used Ricketts' facial pattern analysis as a method to calculate the facial patterns of the experimental samples, the control samples and the Broadbent-Brush standard templates from age three to seven.

The means of the facial patterns of each age group for both the experimental and the control groups were calculated. The means of the facial patterns for all of the experimental group, the control group and the Broadbent-Brush standard templates group were also calculated.(Fig. 47 and 48)

Factor		FACIAL AXIS		FACIAL DEPTH		MAND. ANGLE		MAND. ARC		LOWER FACE HT.		FACIAL PATTERN	BOLTON FACE P.
AGE		NORM	S.D.	NORM	S.D.	NORM	S.D.	NORM	S.D.	NORM	S.D.		
		90°	±3.5	85°	± 3	28°	±4.5	23°	± 4	47°	± 4		
3,	# 1	86.5	- 1	84	-0.3	31	-.9	30.5	+1.9	45	+0.5	+0.1	
	# 2	86.5	- 1	82	- 1	27	+0.2	30	+1.8	41	-1.2	+0.2	
	# 3	90	0	88	+ 1	25	+0.7	31	+ 2	42	+1.3	+ 1	
N	# 4	90	0	83.5	-0.5	27	+0.2	30.5	+1.9	41.5	+1.4	+0.6	
	Mean	88.3	-0.5	84.4	-0.2	27.5	+0.1	30.5	+1.9	42.4	+1.2	+ 0.5	
4	Bltn	91	+0.3	82.5	-0.8	26	+0.4	29	+1.5	45	+0.5		+ 0.4
AGE		90	±3.5	85.3	± 3	27.7	±4.5	23.5	± 4	47	± 4		
	# 5	83	- 2	82	-1.1	31.5	-0.8	27	+0.9	47	0	-0.6	
4,	# 6	87.5	-0.7	81	-1.4	30	-0.5	25	+0.4	48	-0.3	-0.5	
	# 7	90	0	88	+ 1	19	+1.9	37	+3.5	41	+2.5	+1.8	
N	Mean	86.8	- 1	86.3	-0.5	26.8	+0.3	29.6	+1.5	45.3	+0.5	+ 0.2	
3	Bltn	93	+0.8	84	-0.4	23.5	+0.9	32	+2.1	41	+1.5		+ 1.0
		90	±3.5	85.7	± 3	27.3	±4.5	24	± 4	47	± 4		
AGE	# 8	92.5	+0.7	86	+0.1	17	+2.3	37	+3.3	40	+1.8	+1.6	
5,	Mean											+1.6	
N	Btln	91.5	0.4	84	0.6	24	0.7	29	1.3	44	0.8		+ 0.5
		90	±3.5	86	± 3	27	±4.5	24.5	± 4	47	± 4		
AGE	# 9	89	-0.3	81	-1.7	33	-1.3	28	+0.9	50	-0.8	- 0.6	
	# 10	95	+1.4	91	+1.7	19	+1.8	25	+0.1	38	+2.3	+ 1.5	
6,	# 11	87	-0.9	84.5	-0.5	28	-0.2	27.5	+0.8	46	+0.3	- 0.1	
	# 12	88.5	-0.4	87	+0.3	27	0	35	+2.9	42	+1.3	+ 0.8	
N	Mean	89.9	-0.1	85.9	-0.1	24.3	+0.1	28.9	+1.2	44	+0.8	+ 0.4	
4	Btln	91	+0.3	84	-0.6	25	+0.4	30	+1.4	42	+1.3		+ 0.6
		90	±3.5	86.3	± 3	26.7	±4.5	25	± 4	47	± 4		
AGE	# 13	86	-1.1	84.5	-0.6	29	-0.5	31.5	+1.6	46	+0.3	- 0.1	
	# 14	85	-0.3	85	-0.4	21	+1.3	34	+2.3	42	+1.3	+ 0.8	
	# 15	86.5	- 1	87	+0.4	24.5	+0.5	29	+ 1	45	+0.5	+ 0.3	
7,	# 16	90	0	91	+1.6	23	+0.8	30	+1.3	41	+1.5	+ 1.1	
	# 17	80	-2.9	82.5	-1.3	32	-1.2	25	0	48	-0.3	+ 1.6	
N	# 18	90	0	88	+0.6	26	+0.2	23.5	-0.4	46.5	+0.2	+ 0.2	
	# 19	85	-1.4	88	+0.6	25	+0.4	35	+2.5	44	+0.8	+ 0.5	
7	Mean	86	-1.1	86.6	+0.1	25.8	+0.2	29.7	+1.2	44.6	+0.6	+ 0.2	
	Btln	91	+0.3	85	+0.4	25	+0.4	31	+1.5	43	+ 1		+ 0.6
All	Mean	87.5	-0.6	85.4	-0.1	26.5	+0.2	29.7	+1.4	44.1	+0.8	+ 0.2	
All	Btln	91.5	+0.4	83.9	-0.6	25	+0.5	30.2	+1.6	43.2	+0.7		+ 0.5

Fig.47 Statistical calculation of experimenatl group

Factor		FACIAL AXIS		FACIAL DEPTH		MAND. ANGLE		MAND. ARC		LOWER FACE HT.		FACIAL PATTERN	BOLTON FACE P.
AGE		NORM	S.D.	NORM	S.D.	NORM	S.D.	NORM	S.D.	NORM	S.D.		
		90°	±3.5	85°	± 3	28°	±4.5	23°	± 4	47°	± 4		
3, N	# 1	87	-0.9	83	-0.7	30	-0.4	30	+1.8	47	0	-0.1	
	# 2	87.5	-0.7	83	-0.7	31	-0.7	20	-0.8	47	0	-0.6	
	# 3	91	+0.3	80	-1.7	30	-0.4	25	+0.5	43	+1	-0.1	
	# 4	89	-0.3	80	-1.7	22.5	+1.2	27	+ 1	43	+1	+0.2	
4	Mean	88.6	+0.4	81.5	-1.2	28.4	-0.1	25.5	+0.6	45	+0.5	- 0.1	
	Bltn	91	+0.3	82.5	-0.8	26	+0.4	29	+1.5	45	+0.5		+ 0.4
AGE		90	±3.5	85.3	± 3	27.7	±4.5	23.5	± 4	47	± 4		
4, N	# 5	96	+1.7	82.5	-0.9	225	+1.3	31	+1.9	41.5	+1.4	+1.1	
	# 6	92	+0.6	86	+0.2	23	+1.0	30	+1.6	44	-0.8	+0.8	
	# 7	94	+1.1	83	-0.2	21.5	+1.4	32	+2.1	45	+0.5	+0.9	
3	Mean	94	+1.1	83.8	-0.5	22.2	+1.2	31	+1.9	43.3	+0.9	+ 0.9	
	Bltn	93	+0.8	84	-0.4	23.5	+0.9	32	+2.1	41	+1.5		+ 1.0
		90	±3.5	85.7	± 3	27.3	±4.5	24	± 4	47	± 4		
AGE 5, N	# 8	86	-1.1	87	+0.4	22	+1.1	31	+1.6	44	+0.8	+0.6	
	Mean											+0.6	
1	Btln	91.5	0.4	84	0.6	24	0.7	29	1.3	44	0.8		+ 0.5
		90	±3.5	86	± 3	27	±4.5	24.5	± 4	47	± 4		
AGE 6, N	# 9	88	-0.6	84	-0.7	29.5	-0.6	30	+1.2	42	+1.3	+ 0.1	
	# 10	88	-0.6	80	- 2	25.5	+0.3	32	+1.9	46	+0.3	0	
	# 11	91	+0.3	835	- 1	22	+1.1	345	+2.4	39	+ 2	+ 0.1	
	# 12	90	0	85	-0.3	21.5	+1.2	31	+1.6	36	+2.8	+ 1.1	
4	Mean	89.3	-0.2	83	- 1	24.6	+0.5	31.8	+1.8	40.7	+1.6	+ 0.5	
	Btln	91	+0.3	84	-0.6	25	+0.4	30	+1.4	42	+1.3		+ 0.6
		90	±3.5	86.3	± 3	26.7	±4.5	25	± 4	47	± 4		
AGE 7, N	# 13	89	-0.3	84	-0.8	25	+0.4	31	+1.5	48	-0.3	+ 0.1	
	# 14	87	-0.9	81	-1.8	25	+0.4	31.5	+1.6	42	+1.3	+ 0.1	
	# 15	90	0	82	-1.4	21	+1.3	35	+2.5	45	+0.5	+ 0.6	
	# 16	92	+0.6	87	+0.2	18	+1.9	30	+1.3	40	+1.8	+ 1.2	
	# 17	87	-0.9	81.5	-1.6	25	+0.4	34	+2.3	45	+0.5	+ 0.1	
	# 18	88.5	-0.4	86	+0.1	21	+1.3	33	+ 2	45	-0.5	+ 0.7	
	# 19	87	-0.9	83	-1.1	26	+0.2	25	0	42	+1.3	+ 0.1	
7	Mean	88.6	+0.5	83.5	-0.9	23	+0.8	31.4	+1.6	43.8	+0.8	+ 0.4	
	Btln	91	+0.3	85	+0.4	25	+0.4	31	+1.5	43	+ 1		+ 0.6
All Mean		89.5		87.7		24.3		30.1		43.6		+ 0.4	
All Btln		91.5	+0.4	83.9	-0.6	25	+0.5	30.2	+1.6	43.2	+0.7		+ 0.5

Fig.48 Statistical calculation of control group

Since the number of the sample was nineteen, which is small, the Mann-Whitney U.test(Fig.49) which is a nonparametric test was a suitable statistical analysis for this study. The null hypothesis stated that there is no difference between the two samples; i.e. that they are drawn from the same population.

The process of statistical analysis for facial pattern and each of the five measurements in this study are shown as figures 49 to 55 as following:

Fig.49 Table of Mann-Whitney U. test

Table G Critical Values for the Mann-Whitney *U* Test*

One Tailed Test — $\alpha = .01$ (roman), $\alpha = .005$ (**boldface**) — *Two Tailed Test* — $\alpha = .02$ (roman), $\alpha = .01$ (**boldface**)

N_2 \ N_1	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
2	—	—	—	—	—	—	—	—	—	—	—	—	0	0	0	0	0	0	1	1
													—	—	—	—	—	—	**0**	**0**
3	—	—	—	—	—	—	0	0	1	1	1	2	2	2	3	3	4	4	4	5
							—	—	**0**	**0**	**0**	**1**	**1**	**1**	**2**	**2**	**2**	**2**	**3**	**3**
4	—	—	—	—	0	1	1	2	3	3	4	5	5	6	7	7	8	9	9	10
					—	**0**	**0**	**1**	**1**	**2**	**2**	**3**	**3**	**4**	**5**	**5**	**6**	**6**	**7**	**8**
5	—	—	—	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
				—	**0**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**7**	**8**	**9**	**10**	**11**	**12**	**13**
6	—	—	—	1	2	3	4	6	7	8	9	11	12	13	15	16	18	19	20	22
				0	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**9**	**10**	**11**	**12**	**13**	**15**	**16**	**17**	**18**
7	—	—	0	1	3	4	6	7	9	11	12	14	16	17	19	21	23	24	26	28
			—	**0**	**1**	**3**	**4**	**6**	**7**	**9**	**10**	**12**	**13**	**15**	**16**	**18**	**19**	**21**	**22**	**24**
8	—	—	0	2	4	6	7	9	11	13	15	17	20	22	24	26	28	30	32	34
			—	**1**	**2**	**4**	**6**	**7**	**9**	**11**	**12**	**15**	**17**	**18**	**20**	**22**	**24**	**26**	**28**	**30**
9	—	—	1	3	5	7	9	11	14	16	18	21	23	26	28	31	33	36	38	40
			0	**1**	**3**	**5**	**7**	**9**	**11**	**13**	**16**	**18**	**20**	**22**	**24**	**27**	**29**	**31**	**33**	**36**
10	—	—	1	3	6	8	11	13	16	19	22	24	27	30	33	36	38	41	44	47
			0	**2**	**4**	**6**	**9**	**11**	**13**	**16**	**18**	**21**	**24**	**26**	**29**	**31**	**34**	**37**	**39**	**42**
11	—	—	1	4	7	9	12	15	18	22	25	28	31	34	37	41	44	47	50	53
			0	**2**	**5**	**7**	**10**	**13**	**16**	**18**	**21**	**24**	**27**	**30**	**33**	**36**	**39**	**42**	**45**	**48**
12	—	—	2	5	8	11	14	17	21	24	28	31	35	38	42	46	49	53	56	60
			1	**3**	**6**	**9**	**12**	**15**	**18**	**21**	**24**	**27**	**31**	**34**	**37**	**41**	**44**	**47**	**51**	**54**
13	—	0	2	5	9	12	16	20	23	27	31	35	39	43	47	51	55	59	63	67
		—	**1**	**3**	**7**	**10**	**13**	**17**	**20**	**24**	**27**	**31**	**34**	**38**	**42**	**45**	**49**	**53**	**56**	**60**
14	—	0	2	6	10	13	17	22	26	30	34	38	43	47	51	56	60	65	69	73
		—	**1**	**4**	**7**	**11**	**15**	**18**	**22**	**26**	**30**	**34**	**38**	**42**	**46**	**50**	**54**	**58**	**63**	**67**
15	—	0	3	7	11	15	19	24	28	33	37	42	47	51	56	61	66	70	75	80
		—	**2**	**5**	**8**	**12**	**16**	**20**	**24**	**29**	**33**	**37**	**42**	**46**	**51**	**55**	**60**	**64**	**69**	**73**
16	—	0	3	7	12	16	21	26	31	36	41	46	51	56	61	66	71	76	82	87
		—	**2**	**5**	**9**	**13**	**18**	**22**	**27**	**31**	**36**	**41**	**45**	**50**	**55**	**60**	**65**	**70**	**74**	**79**
17	—	0	4	8	13	18	23	28	33	38	44	49	55	60	66	71	77	82	88	93
		—	**2**	**6**	**10**	**15**	**19**	**24**	**29**	**34**	**39**	**44**	**49**	**54**	**60**	**65**	**70**	**75**	**81**	**86**
18	—	0	4	9	14	19	24	30	36	41	47	53	59	65	70	76	82	88	94	100
		—	**2**	**6**	**11**	**16**	**21**	**26**	**31**	**37**	**42**	**47**	**53**	**58**	**64**	**70**	**75**	**81**	**87**	**92**
19	—	1	4	9	15	20	26	32	38	44	50	56	63	69	75	82	88	94	101	107
		0	**3**	**7**	**12**	**17**	**22**	**28**	**33**	**39**	**45**	**51**	**56**	**63**	**69**	**74**	**81**	**87**	**93**	**99**
20	—	1	5	10	16	22	28	34	40	47	53	60	67	73	80	87	93	100	107	114
		0	**3**	**8**	**13**	**18**	**24**	**30**	**36**	**42**	**48**	**54**	**60**	**67**	**73**	**79**	**86**	**92**	**99**	**105**

*To be significant the *U* obtained from the data must be *equal to or less than* the value shown in the table. Dashes in the body of the table indicate that no decision is possible at the stated level of significance.

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Table G (continued)

One Tailed Test — $\alpha = .05$ (roman), $\alpha = .025$ (**boldface**) — *Two Tailed Test* — $\alpha = .10$ (roman), $\alpha = .05$ (**boldface**)

N_2 \ N_1	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0	0
2	—	—	—	—	0	0	0	1	1	1	1	2	2	2	3	3	3	4	4	4
					—	—	—	**0**	**0**	**0**	**0**	**1**	**1**	**1**	**1**	**1**	**2**	**2**	**2**	**2**
3	—	—	0	0	1	2	2	3	3	4	5	5	6	7	7	8	9	9	10	11
			—	—	**0**	**1**	**1**	**2**	**2**	**3**	**3**	**4**	**4**	**5**	**5**	**6**	**6**	**7**	**7**	**8**
4	—	—	0	1	2	3	4	5	6	7	8	9	10	11	12	14	15	16	17	18
			—	**0**	**1**	**2**	**3**	**4**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**11**	**12**	**13**	**13**
5	—	0	1	2	4	5	6	8	9	11	12	13	15	16	18	19	20	22	23	25
		—	—	**0**	**1**	**2**	**3**	**5**	**6**	**7**	**8**	**9**	**11**	**12**	**13**	**14**	**15**	**17**	**18**	**19**	**20**
6	—	0	2	3	5	7	8	10	12	14	16	17	19	21	23	25	26	28	30	32
		—	—	**1**	**2**	**3**	**5**	**6**	**8**	**10**	**11**	**13**	**14**	**16**	**17**	**19**	**21**	**22**	**24**	**25**	**27**
7	—	0	2	4	6	8	11	13	15	17	19	21	24	26	28	30	33	35	37	39
		—	—	**1**	**3**	**5**	**7**	**8**	**10**	**12**	**14**	**16**	**18**	**20**	**22**	**24**	**26**	**28**	**30**	**32**	**34**
8	—	1	3	5	8	10	13	15	18	20	23	26	28	31	33	36	39	41	44	47
		—	**0**	**2**	**4**	**6**	**8**	**10**	**13**	**15**	**17**	**19**	**22**	**24**	**26**	**29**	**31**	**34**	**36**	**38**	**41**
9	—	1	3	6	9	12	15	18	21	24	27	30	33	36	39	42	45	48	51	54
		—	**0**	**2**	**4**	**7**	**10**	**12**	**15**	**17**	**20**	**23**	**26**	**28**	**31**	**34**	**37**	**39**	**42**	**45**	**48**
10	—	1	4	7	11	14	17	20	24	27	31	34	37	41	44	48	51	55	58	62
		—	**0**	**3**	**5**	**8**	**11**	**14**	**17**	**20**	**23**	**26**	**29**	**33**	**36**	**39**	**42**	**45**	**48**	**52**	**55**
11	—	1	5	8	12	16	19	23	27	31	34	38	42	46	50	54	57	61	65	69
		—	**0**	**3**	**6**	**9**	**13**	**16**	**19**	**23**	**26**	**30**	**33**	**37**	**40**	**44**	**47**	**51**	**55**	**58**	**62**
12	—	2	5	9	13	17	21	26	30	34	38	42	47	51	55	60	64	68	72	77
		—	**1**	**4**	**7**	**11**	**14**	**18**	**22**	**26**	**29**	**33**	**37**	**41**	**45**	**49**	**53**	**57**	**61**	**65**	**69**
13	—	2	6	10	15	19	24	28	33	37	42	47	51	56	61	65	70	75	80	84
		—	**1**	**4**	**8**	**12**	**16**	**20**	**24**	**28**	**33**	**37**	**41**	**45**	**50**	**54**	**59**	**63**	**67**	**72**	**76**
14	—	2	7	11	16	21	26	31	36	41	46	51	56	61	66	71	77	82	87	92
		—	**1**	**5**	**9**	**13**	**17**	**22**	**26**	**31**	**36**	**40**	**45**	**50**	**55**	**59**	**64**	**67**	**74**	**78**	**83**
15	—	3	7	12	18	23	28	33	39	44	50	55	61	66	72	77	83	88	94	100
		—	**1**	**5**	**10**	**14**	**19**	**24**	**29**	**34**	**39**	**44**	**49**	**54**	**59**	**64**	**70**	**75**	**80**	**85**	**90**
16	—	3	8	14	19	25	30	36	42	48	54	60	65	71	77	83	89	95	101	107
		—	**1**	**6**	**11**	**15**	**21**	**26**	**31**	**37**	**42**	**47**	**53**	**59**	**64**	**70**	**75**	**81**	**86**	**92**	**98**
17	—	3	9	15	20	26	33	39	45	51	57	64	70	77	83	89	96	102	109	115
		—	**2**	**6**	**11**	**17**	**22**	**28**	**34**	**39**	**45**	**51**	**57**	**63**	**67**	**75**	**81**	**87**	**93**	**99**	**105**
18	—	4	9	16	22	28	35	41	48	55	61	68	75	82	88	95	102	109	116	123
		—	**2**	**7**	**12**	**18**	**24**	**30**	**36**	**42**	**48**	**55**	**61**	**67**	**74**	**80**	**86**	**93**	**99**	**106**	**112**
19	0	4	10	17	23	30	37	44	51	58	65	72	80	87	94	101	109	116	123	130
	—	**2**	**7**	**13**	**19**	**25**	**32**	**38**	**45**	**52**	**58**	**65**	**72**	**78**	**85**	**92**	**99**	**106**	**113**	**119**
20	0	4	11	18	25	32	39	47	54	62	69	77	84	92	100	107	115	123	130	138
	—	**2**	**8**	**13**	**20**	**27**	**34**	**41**	**48**	**55**	**62**	**69**	**76**	**83**	**90**	**98**	**105**	**112**	**119**	**127**

The Mann-Whitney Test

Ho: Rank of MB = Rank of Non-MB
Ha: Rank of MB = Rank of Non-MB
(MB: Mouthbreathing)
= 0.05

Subject Facial patterns		Subject Ranks	
Mouth-breather	Non-Mouth breather	Mouth breather	Non-Mouth breather
N_1=19	N_2=19	N_1=19	N_2=19
-0.10	+0.05	26.5	29
-0.20	+0.60	21.5	36
-1.00	+0.06	8	30
-0.60	-0.24	15	20
+0.60	-1.08	36	6
+0.50	-0.84	34	11
-1.80	-0.98	1	9
-1.60	-0.56	2	17
+0.60	-0.12	36	24.5
-1.50	+0 02	3	28
+0.10	-0.96	32	10
-0.80	-1.06	12.5	7
+0.10	-0.10	32	26.5
-0.80	-0.12	12.5	24.5
-0.30	-0.58	19	16
+1.60	-1.16	38	4
-1.10	-0.14	5	23
-0.20	-0.70	21.5	14
-0.50	+0.10	18	32
		Total=373.5	367.5

$T = 373.5 - 19(19+1) / 2 = 183.5$ $\quad N_1 = 19, \quad N_2 = 19, \quad \alpha = 0.05$

From Table $W_{\alpha/2}(=0.025) = 113$

$W_{1-\alpha/2} = 19 \times 19 - 113 = 248$

$W_{1-\alpha/2} > T > W_{\alpha/2}$

So that the Ho can not be rejected.

It means that there is no difference between the two samples in terms of Facial Pattern

Fig.50 Statistical analysis for facial patterns

The Mann- Whitney U. Test

Ho: Rank of MB = Rank of Non- MB
Ha: Rank of MB = Rank of Non- MB
(MB: Mouthbreathing)
α = 0.005

Subject Facial Axis		Subject Ranks	
Mouth-breather	Non-Mouth breather	Mouth Breather	Non-Mouth Breather
N_1 = 19	N_2 = 19	N_1 = 19	N_2 = 19
86.5	87	8	12
86.5	87.5	8	15.5
90	91	27	31.5
90	89	27	22
83	96	2	38
87.5	92	15.5	33.5
90	94	27	36
92.5	86	35	5.5
89	88	22	17.5
95	88	37	17.5
87	91	12	31.5
88.5	90	19.5	27
86	89	5.5	22
85	87	3.5	12
86.5	90	8	27
90	92	27	33.5
80	87	1	12
90	88.5	27	19.5
85	87	3.5	12
		Total= 315.5	425.5

$T = 315 - 19(19+1) / 2 = 125$ $\quad N_1 = 19, \quad N_2 = 19, \quad \alpha = 0.05$

From Table $W_{\alpha/2}(=0.025) = 113$

$W_{1-\alpha/2} = 19 \times 19 - 113 = 248$

$W_{1-\alpha/2} > T > W_{\alpha/2}$

So that the Ho can not be rejected.

It means that there is no difference between the two samples in terms of Facial Axis.

Fig.51 Statistic analysis for facial axis

The Mann- Whitney U. Test

Ho: Rank of MB = Rank of Non- MB
Ha: Rank of MB = Rank of Non- MB
(MB: Mouthbreathing)
α = 0.005

Subject Facial Depth		Subject Ranks	
Mouth-breather	Non-Mouth breather	Mouth Breather	Non-Mouth Breather
N_1 =19	N_2 =19	N_1 =19	N_2 =19
84	83	20	15
82	83	9	15
88	80	34.5	2
83.5	80	18	2
82	82.5	9	11.5
81	86	5	27
88	83	34.5	15
86	87	27	30.5
81	84	5	20
91	80	37.5	2
84.5	83	22.5	15
87	85	30.5	24.5
84.5	84	22.5	20
85	81	24.5	5
87	82	30.5	9
82.5	87	11.5	30.5
91	81.5	37.5	7
88	86	34.5	27
88	83	34.5	15
		Total= 448	293

T= 448 - 19(19+1) / 2 = 258 N_1 = 19, N_2 = 19, α = 0.05

From Table $W_{\alpha/2}(=0.025) = 113$

$W_{1-\alpha/2} = 19 \times 19 - 113 = 248$

$T > W_{\alpha/2}$

$T > W_{1-\alpha/2}$

So that the Ho can be rejected.

It means that there is difference between the two samples in terms of Facial Depth.

Fig.52 Statistics analysis for facial depth

The Mann- Whitney U. Test

Ho: Rank of MB = Rank of Non- MB
Ha: Rank of MB = Rank of Non- MB
(MB: Mouthbreathing)
α = 0.005

Subject Mand. Plane Angle		Subject Ranks	
Mouth-breather	Non-Mouth breather	Mouth Breather	Non-Mouth Breather
N_1 = 19	N_2 = 19	N_1 = 19	N_2 = 19
31	30	34.5	32
27	31	26	34.5
25	30	19.5	32
27	22.5	26	13
31.5	22	36	11
30	23	32	14.5
19	21.5	3.5	8.5
17	22	1	11
33	29.5	38	30
19	25	3.5	19.5
28	22	28	11
27	21.5	26	8.5
29	25	29	19.5
21	25	6	19.5
24.5	21	16	6
32	18	37	2
23	25	14.5	19.5
26	21	23.5	6
25	26	19.5	23.5
		Total= 419.5	321.5

T= 419.5 - 19(19+1) / 2 = 229.5 N_1 = 19, N_2 = 19, α = 0.05

From Table $W_{\alpha/2}(=0.025) = 113$

$W_{1-\alpha/2} = 19 \times 19 - 113 = 248$

$W_{1-\alpha/2} > T > W_{\alpha/2}$

So that the Ho can not be rejected.

It means that there is no difference between the two samples in terms of Mandibular Plane Angle.

Fig.53 Statistical analysis for mandibular plane angle

The Mann- Whitney U. Test

Ho: Rank of MB = Rank of Non- MB
Ha: Rank of MB = Rank of Non- MB
(MB: Mouthbreathing)
α = 0.005

Subject Mandibular Arc		Subject Ranks	
Mouth-breather	Non-Mouth breather	Mouth Breather	Non-Mouth Breather
N_1 = 19	N_2 = 19	N_1 = 19	N_2 = 19
30.5	30	19.5	15.5
30	20	15.5	1
31	25	23	5
30.5	27	19.5	8.5
27	31	8.5	23
25	30	5	15.5
37	32	37.5	28.5
37	31	37.5	23
28	30	11	15.5
25	32	5	28.5
27.5	34	10	32
35	31	35	23
31.5	31	26.5	23
34	31.5	32	26.5
29	35	12	35
25	30	5	15.5
30	34	15.5	32
23.5	33	2	30
35	25	35	5
		Total= 355	386

$T = 355 - 19(19+1) / 2 = 165$ $\quad N_1 = 19, \quad N_2 = 19, \quad \alpha = 0.05$

From Table $W_{\alpha/2}(=0.025) = 113$

$W_{1-\alpha/2} = 19 \times 19 - 113 = 248$

$W_{1-\alpha/2} > T > W_{\alpha/2}$

So that the Ho can not be rejected.

It means that there is no difference between the two samples in terms of Mandibular Arc.

Fig.54 Statistical analysis for mandibular arc

The Mann- Whitney U. Test

Ho: Rank of MB = Rank of Non- MB
Ha: Rank of MB = Rank of Non- MB
(MB: Mouthbreathing)
α = 0.005

Subject Lower Facial Ht.		Subject Ranks	
Mouth-breather	Non-Mouth breather	Mouth Breather	Non-Mouth Breather
N_1 = 19	N_2 = 19	N_1 = 19	N_2 = 19
45	47	24.5	33
41	47	7	33
42	43	13.5	17.5
41.5	43	9.5	17.5
47	41.5	33	9.5
48	44	36	20
41	45	7	24.5
40	44	4.5	20
50	42	38	13.5
38	46	2	29
46	39	29	3
42	36	13.5	1
46	48	29	36
42	42	13.5	13.5
45	45	24.5	24.5
48	40	36	4.5
41	45	7	24.5
46.5	45	31	24.5
44	42	20	13.5
		Total= 378.5	362.5

T= 378.5 - 19(19+1) / 2 = 188.5 N_1 = 19, N_2 = 19, α = 0.05

From Table $W_{\alpha/2}(=0.025) = 113$

$W_{1-\alpha/2} = 19 \times 19 - 113 = 248$

$W_{1-\alpha/2} > T > W_{\alpha/2}$

So that the Ho can not be rejected.

It means that there is no difference between the two samples in terms of Lower Facial Height.

Fig.55 Statistical analysis for lower facial height

1. From the result of the Mann-Whitney U.test, the null hypothesis which stated that there was no difference between the two samples could not be rejected at the $p< 0.05$ level as far as facial pattern, facial axis, mandibular plane angle, mandibular arc and lower facial height are concernd. It could be concluded that both groups were from the same population distribution. That meant there were no statistical difference between the experimental and the control groups as far as facial pattern, facial axis, mandibular plane angle, mandibular arc and lower facial height were concerned.

2. The only measurement which had significant difference was the facial depth.

3. The result showed that there was no significant effect of the mouthbreathing on the craniofacial pattern for the children from three to seven years of age.

4. All the means of the facial patterns of the experimental group, the control group, and the Bolton standard templates were within one standard deviation, on the Brachyfacial side, i.e. though they were all within mesofacial pattern but on the brachyfacial side of the Rickett's norm.

5. The mean of facial pattern of the experimental is 0.2 on the brachyfacial side to the Rickett's norm. The mean of the facial pattern of the Bolton standard from age three to seven is 0.5 on the brachyfacial side. And the mean of the facial pattern of the control group which is taken from the

Bolton study is 0.4 on the Brachfacial side, which is pretty much close the the mean of facial pattern of its population i,e. the Bolton standard.

CHAPTER V

DISCUSSION

1. The experimental sample size was limited by the difficulty of getting a larger sample referred from an ENT office which is a little far away from the investigator's department. The small size of sample made not only the parametric analysis inadequate but also the original attempt to compare the facial patterns between each age group impossible. Future researches with a larger sample size might be proper to more completely evaluate the effect of mouthbreathing on the craniofacial development at each age group.

2. Because the depth of nasopharynx which was related to air flow was established in the first or second year of life,[7] [19] [20] and only minor changes in size of adenoid were observed between the ages of 2 to 15 years[26], it can be assumed that the air flow capacity was established as early as age two and kept constant until puberty when the adenoids started to recede. We suppose that mouthbreathing developed in those subjects who had adenoid obstruction at their age of examination may exist for many years until puberty if treatments were not given.

Based on the mentioned fact stated above, there is no evidence showing that mouthbreathing has effect on the

craniofacial deformity for those young children in this study. Early adenoidectomy to correct the mouthbreathing to nose-breathing was recommended by Linder-Aronson[57] as helpful to prevent the facial deformity.

3. Because the control group is from the Broadbent-Brush Growth Study Center, there is no way to be absolutely sure that they were nasal-breather. However, they were within normal value cephalometrically. For future study, samples with history of non-mouthbreathing from normal population will be proper for the control group.

4. Most Studies demonstrated a positive relationship between airway obstruction and a dolicofacial pattern.[10 11 12 13 14 15 16 17 18] The reasons that there was no significant relationship found in this research may be due to the following factors:

A. The period of time of mouthbreathing for the subjects in this research had been so short that changes on the development of facial deformity were not yet significant. Because the size of airway was established in the early childhood by the stable size of adenoid[24 25] and the stable depth and width of nasopharyngeal space[19 20 38], the subjects might continue the mouthbreathing until treatment or after puberty when adenoids gradually recede.[25] In Drs.' Vargervik and Harvold's animal study[21] it was concluded that the changes in mandibular morphology will only occur when lowering of the mandible was sufficiently persistent.

Most parents and children didn't remember the age of onset of mouthbreathing nor the exact period of time of the existence of the mouthbreathing problem. So we would not be able to know the period of time that mouthbreathing had its effect on the craniofacial development for each subject. A longitudinal research for mouthbreathers could be indicated to evaluate the effect of mouthbreathing on craniofacial development.

B. The subjects in this research were very young. Most of them didn't have first permanent molar eruption. So the effect of mouthbreathing could not have significant changes on the development of facial deformity. In the study by Drs' Vargervik and Harvold[21], they concluded that the lower face height was increased significantly when excessive molar extrusion occurred. Increased molar extrusion which may cause increased lower facial height would be expected to occur most rapidly during eruption of the first and second molars.

5. A small sample size of this study may also have an impact on the result. The underlying distribution of the experimental group in not known to be of a normal population. The data of this study can't be used in parametric statistical analysis to other studies such as Michigan, Burlington, or Ricketts' which had a large sample size as a normal population.

A further related research with large sample size, matched race, age, and sex would be indicated to either

confirm this research or may have different result.

6. Different sources of materials for the experimental and control groups may have another impact to this study. The lateral head plates of the Broadbent-Brush Growth Center are more than forty years old and might not be clear enough to identify exactly the needed anatomical points. The anatomical porions, which are very important in the Ricketts' facial pattern analysis, were converted from machine porions showing on those lateral head plates by a set of different templets. The different templates were used according to different types of ear roads and head fixer poles used in different years when those lateral head plates were taken during the whole period of time of Broadbent-Brush Growth Study. There might be errors between the real and the converted anatomical porions and which may influence the facial pattern analysis.

For further study, the control samples not only to be matched to the experimental sample in race, sex and age but also taken not from the other old study materials but by the exact the same method as the experimental samples would be suggested.

7. From the result that the mean of the facial pattern of the experimental group, which is 0.2 on the brachyfacial side, is a little bit more on the dolicofacial pattern side when it is compared to the means of facial patterns of the control group and the Bolton standard which are 0.4 and 0.5 on the brachyfacial pattern side seperately. So if from

inspection of comparison between the means of facial patterns of the expremental and the control group, it seems that there is possibility of tendency that the effect of mouthbreathing to the growth of facial pattern may have existed even to the young study group. Further study would be very necessary and interesting to confirm this assumption.

CHAPTER VI

SUMMARY

Many studies had shown the effect of nasopharyngeal obstruction to the development of craniofacial pattern on children aged above six. No study had ever been done to identify the effect on younger children. This study was trying to investigate such effect on children from three to seven years of age.

Nineteen children with history of chronic nasopharyngeal obstruction were referred from an Ear, Nose, and Throat specialist to us to take a lateral head X-Ray film. Cephalometric analysis according to Ricketts' facial pattern analysis had been calculated for each subjects. For the control group, nineteen tracings of lateral head X-Ray films of those who didn't have airway obstruction history were chosen to match the experimental group by race, sex and age from Broadbent-Brush Growth Center. They were also calculated as the same way as the experimental group for their facial patterns.

The result of the statistical analysis showed there is no difference between the two groups as far as the facial patterns were concerned. From this study the effect of mouthbreathing on the craniofacial development for children from three to seven years of age could not be found.

Further studies with large sample size would be indicated.

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