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EFFECTS OF EXPERIMENTAL ALTERATION OF NASAL FUNCTION ON CRANIOFACIAL GROWTH IN THE GUINEA PIG

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EFFECTS OF EXPERIMENTAL ALTERATION OF NASAL FUNCTION

ON CRANIOFACIAL GROWTH IN THE GUINEA PIG

Abstract

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While the effects of breathing mode on craniofacial growth have received great interest, no studies exist on the effects of imbalanced nasal function. Deviated nasal septum, hypertrophied turbinates, unilateral choanal atresia, and foreign objects are some of the common causes underlying imbalanced nasal function.

It was the purpose of the present study to examine experimentally any possible effects of imbalanced nasal function on craniofacial growth. Nineteen, three week old guinea pigs were used for this study. Six of them were used as control and the remaining 13 as experimental group. In the experimental group the right nasal airway was blocked by suturing the anterior nasal opening. In this way, inspiration and airconditioning, two functions of the nasal breathing cycle, were stopped. The animals were kept for four months after which they were sacrificed. Histological, ground sectioning and cephalometric data were prepared to evaluate any effect.

The right and left sides were compared in regards to size and shape of nasal cavities, type and distribution of epithelia, type

and distribution of glands, size and shape of the turbinates, bone deposition pattern, size of the nasolacrimal duct and shape of nasal septum.

It was found that there was no significant difference between the blocked and the unblocked sides. This finding lead to the following conclusions.

1. The relationship between nasal functions and nasomaxillary growth and morphology has been related in the literature, up to now, has been oversimplified.
2. Inspiration and airconditioning, at least for guinea pigs, do not have apparent effects on nasomaxillary growth patterns.
3. Not all of the functions of an organ or tissue necessarily affect its growth pattern.

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INTRODUCTION

The craniofacial complex enlarges by means of regional growth sites which provide a final, composite of structural and functional "Balance". It is believed that these sites have some degree of an inherited growth potential which combine with local and general functional factors to determine the vectors of growth that occur.

A craniofacial component that has received much interest recently is the nasomaxillary area. How abnormal breathing affects the growth plan of this region is not presently well understood. The interest to date has concentrated on the effects of the mode of breathing. Problems with the adenoids, a frequent cause of nasal obstruction, have attracted a great number of clinical investigations. Mouth breathing is known to cause functional and structural alterations.

Based on current knowledge, it seems that normal nasal inspiration is essential for normal nasomaxillary growth. Factors affecting nasal airflow, nasal resistance, the nasal cycle and conditioning of the air, have been studied in detail.

To our knowledge, however, no studies have been carried out examining the effect of nasal functional imbalance on the growth plan of the nasomaxillary region. A nasal functional imbalance does not necessarily co-exist with mouth breathing. Mouth breathing is induced when adequate oxygen requirements cannot be provided through the nasal airway.

The purpose of this study is to determine experimental effects of nasal functional imbalance on the nasomaxillary growth plan.

LITERATURE REVIEW

I) ANATOMY OF THE NOSE

The nasal complex comprises the external nasal protuberance and the internal nasal chambers, both of which are divided by a septum into a right and left part. A phylogenic explanation of this division is lacking, but Meyerhoff (1980), suggested that possible reasons are to allow one side to function if the other becomes closed.

The total length of the nasal complex is about 12-14 cm (Proctor, 1977; Anderson et al, 1971). The volume of each side is about 7-8 ml (Hilding, 1976) or even 10 ml (Bouhuys, 1974). Montgomery et al., (1979), using computed tomography, estimated an average volume of 13.4 ml. However, since his sample was not only small but also utilized only cadavers, this value should be regarded with reservation because vascularity of the living nasal membrane was not considered.

The External Nose

The external nose is often called the nasal pyramid because of its configuration. The portion contiguous with the frontal bone is termed the root, while the tip of the nose is often referred to as the apex. The rooflike portion between the apex and the root is called simply the dorsum. The part of the dorsum formed by the nasal bones is referred to as the bridge. The base of the nose extends from the apex to the lip and is divided by the columella into the two anterior nares. Each anterior naris is bounded medially by the columella and septum, laterally and superiorly by the nasal alae and inferiorly by the floor.

The supporting framework of the external nose consists of a bony part and a number of paired and unpaired cartilages.

The Bony Components: The bony nasal compartment is comprised of the anterior part of the body of the maxilla, with its frontal processes, and the paired nasal bones. The nasal bones articulate laterally with the medial surface of the frontal process of the maxilla, superiorly with the medial part of the nasal notch of the frontal bone, medially between each other and inferiorly along the midline, with the lower part of the perpendicular plate of the ethmoid bone. Their free low borders are attached to the upper lateral nasal cartilages. The bony aperture of the external nose is bounded by the projecting free lower borders of the nasal bones above, the anterior edges of the maxilla on each side, and the fused bodies of the maxillae (premaxillae) below.

The bony part of the nasal septum does not project into the external nose, and thus the anterior nasal aperture of the skull itself is a single pear-shaped opening. On the midline of the lower border of the aperture is a small projecting process, the anterior nasal spine.

The Cartilaginous Components: The cartilaginous parts consist of two upper and two lower nasal cartilages, the two smaller alar cartilages and the central septal cartilage (Hamilton and Harrison, 1979).

The upper nasal cartilage, also termed the lateral cartilage or the triangular cartilage (Dion et al, 1978) is triangular in shape

and is attached medially to the septal cartilage, laterally and superiorly to the anterior edge of the nasal bones and the frontal process of maxilla, and inferiorly to the lower nasal and alar cartilages.

The lower nasal cartilages, also termed greater alar, lobular or tip cartilage (Dion et al, 1978), partially encircle the nares and assist in keeping them open and providing shape to the base of the nose. These cartilages consist of a medial and lateral crura, which are continuous at the apex, giving an arched contour to the nasal tip. The medial crura approximate each other bounding a deep median groove, extend posteroinferiorly in the nasal columella and end in a free out-turned border. Each lateral crus extends posterolaterally in the ala towards the maxilla but does not reach it. The internal is filled with a strong sheet of fibrous tissue in which are embedded the smaller alar (also termed minor, less, accessory) cartilages. In some cases, the lateral crus is so elongated that it replaces the smaller alar cartilages (Fairbanks, 1983).

The septal cartilage is somewhat quadrilateral in shape. It is attached posterosuperiorly to the perpendicular plate of the ethmoid bone, superiorly to the internasal suture and crest, posteriorly to the anterior border of the vomer and inferiorly to the nasal crest of the maxilla and the anterior nasal spine. Anterosuperiorly the septal cartilage is attached to the two upper nasal cartilages. The posterior border of the septal cartilage often has an elongated, narrow extension which fits between the vertical plate of the ethmoid bone and the anterior border of the vomer.

Nasal Chambers

The internal compartments of the nasal complex, extending anteriorly from the nares (nostrils) to the choanae posteriorly to communicate with the nasopharynx, is divided into two parts, the paired vestibules and the nasal fossae which include the respiratory and olfactory regions (Weiss and Greep, 1977; Ham and Cormack, 19?).

The vestibule is the anterior skin-lined part of the nasal cavity that contains the vibrissae (nasal hairs) and numerous sebaceous and sweat glands. It is limited above and behind by the nasal valve (or internal ostium, limen nasi), (Walike, 1983; Mygind, 1978). A curved elevation lateral to the valve corresponds to the lower margin of the upper lateral cartilage, which can be palpated as a sharp prominence with the finger, some 15 mm from the nostril. The nasal valve plays an important role in nasal function and it is the narrowest place in the cavity, the cross-section being approximately 0.3 cm^2 on each side (Grundfast, 1983; Mygind, 1978; Cottle, 1955).

Each nasal fossa has a roof, a floor, a lateral and a medial wall and it communicates with the maxillary, sphenoidal, frontal and ethmoidal sinuses of the same side.

The roof displays a sloping anterior part, a more horizontal central part and a sloping posterior part. The front part comprises the nasal bones and the inferior surface of the nasal spine of the frontal bone. The middle part is the cribriform plate of the ethmoid bone. The posterior part is the inferior surface of the body

of the sphenoid bone which is partially covered by the alae of the vomer, the sphenoidal process of the palatine and the vaginal process of the medial pterygoid plate.

The nasal floor is concave from side to side and is slightly concave from front to back. The anterior three-quarters is formed by the union of the horizontal plates of the two maxillae, and the posterior quarter by the union of the horizontal plates of the two palatine bones.

The lateral wall is dominated by the presence of the three turbinates (conchae) which project from the wall as scroll or baffle-like projections. These are the inferior, middle and superior turbinates, the latter two of which are parts of the ethmoid bone, while the first is a separate bone. Sometime a superior turbinate is also present (Hilding, 1976). The bones that contribute to the lateral wall of the nasal cavity are: the frontal process of the maxilla anteriorly, the nasal bone and the nasal process of the frontal bone anterosuperiorly, and the medial pterygoid plate and the perpendicular plate of the palatine bone posteriorly. The spaces between the turbinates are termed the meati, and include the superior, middle and inferior meatus. Into them drain the various paranasal sinuses.

The inferior turbinate extends nearly the entire length of the nasal cavity, protects the inferior meatus and is covered with a thick vascular membrane. The bony part of the inferior turbinate may be close to the lateral nasal wall, or it may jut prominently

into the airway causing nasal obstruction. The inferior meatus, the largest of the three, is narrow anteriorly and posteriorly but wider in its middle part.

The middle turbinate, a part of the ethmoid bone, is smaller than the inferior turbinate and overhangs the middle meatus. It often contains an air cell, and this turbinate may be enlarged to a point that causes nasal obstruction. The middle meatus contains the openings of the frontal, maxillary and anterior ethmoid sinuses.

The superior turbinate is shorter and less prominent than the middle turbinate. Its mucous membrane is thinner and less vascular than that of the other two turbinates, and it also contains the olfactory epithelium with sensory cells. The superior meatus is a small groove between the superior turbinate and the posterior one-half of the middle turbinate form which most of the posterior ethmoid cells empty.

The medial wall is represented by the nasal septum, the major parts of which are the septal cartilage, the perpendicular plate of the ethmoid bone and the vomer. A small contribution is made by the membranous septum and columella, the part of the upper nasal cartilages with their attachments to the septal cartilage, the nasal bones which are fused in the midline, the nasal process and spine of the frontal bone, the sphenoid bone, the nasal crest of the palatine bone, the nasal crest of the maxilla and the anterior nasal spine.

The perpendicular plate of the ethmoid articulates with the nasal spine of the frontal bone above. The cribriform plate behind

and the body of the sphenoid posteriorly. The upper end of the vomer is thickened to form two alae, which spread out on the undersurface of the sphenoid and are held in place by the vaginal processes of the medial pterygoid plates. The rostrum of the sphenoid bone, a small anteriorly directed wedge of bone projecting from the body of the sphenoid bone, fits between the posteroinferior edge of the ethmoid bone and the anterosuperior angle of the vomer, inferiorly, the vomer articulates with the nasal crest, the latter being a slight ridge at the junction of the horizontal plates of the two maxillae and palatine bones.

II) HISTOLOGY OF THE NOSE

Bone, cartilage and to a small degree, dense connective tissue provide rigidity to the walls, floor and roof of the nasal cavities preventing collapse during inspiration.

The epidermis and dermis covering the external surfaces of the fleshy external extend into and line the anterior part of each vestibule. Inside the vestibule, at a variable distance from the exterior, this stratified squamous epithelium becomes non-keratinized and more posteriorly it becomes pseudostratified ciliated columnar with goblet cells. The epithelial surface is normally covered with mucus provided by its goblet cells and the glands of the underlying lamina propria. At a few points, where the exterior air strikes an exposed surface, as at the lower extremities of the inferior turbinates, the epithelium undergoes metaplasia to become stratified squamous in type (Sorokin, 1980).

The basement membrane, separating the epithelium from the lamina propria, is distinct and much thicker than that of most epithelia. The lamina propria is rich in collagenous and elastic fibers and contains both mucous and serous glands. It is continuous with the periosteum of the bone or the perichondrium of the cartilage. Lymphocytes, plasma cells, macrophages, and scattered granular leukocytes occur in the lamina propria.

The vascular supply to the nose is rich and has several unusual features. The main vessels be adjacent to the periosteum or perichondrium in a latticework pattern that becomes a close-meshed net

as they run either obliquely or horizontally forward across the nasal septum and lateral walls (Sorokin, 1980). The main arteries send superficial, arcading branches from which other vessels extend perpendicularly toward the surface where they become arterioles that supply a network of capillaries just beneath the epithelium. However, Harper (1947), showed that the main arteries can occupy an interosseous position, and that branches pass from them to form a fine subepithelial plexus.

Within the lamina propria of the turbinates, in addition to the rich vascular supply which is common to all nasal mucosal, there are a large numbers of venous structures, called swell bodies, that under normal conditions are collapsed (Weiss and Greep, 1977). However, under certain circumstances they can become distended with blood thus increasing the thickness of the mucosa. This mucous membrane can encroach on the airway to an extent that nasal breathing is made difficult or blocked entirely.

III) NASAL BREATHING

Human evolution has been marked by an increasing cranial capacity and a reduction of certain facial components. Reduction of the nose has proceeded much faster than the other portions of the face. Actually further reduction in the size of the nose cannot take place because the normal function would be impaired. This differential in facial reduction has created a unique human attribute, the nasal pyramid, which is an individuals chief characteristic facial feature and according to Groner (1963) of basic importance in the formation and preservation of the body image.

Galen, about 2000 years ago, wrote:

> "...the apertures of the nose, how marvelously they come next after the spongelike (ethmoid)bone and how the connection was cut through into the mouth at the palate in order that inspiration may not begin in a straight line with the rough artery (trachea) and that the air entering it may be berit and convoluted, so to speak. For I think this should be doubly advantageous:...the parts of the lung will never be chilled...and the particles of dust... will not penetrate as far as the artery (trachea)."

It is admirable how meaningfully this man perceived the nasal functions. Today, we have a much more broad knowledge about normal upper airway functions, and to list them, according to Meyerhoff, 1980; Douek, 1979; Reidy, 1968, the following are prominent:

1) Respiration
2) Filtration
3) Olfaction
4) Vocal resonance, and
5) Nasal reflex functions.

Of these, the present review deals only with the first. Specifically, airflow, pressure differences along the nasal tract, the resistance provided by the nasal cavities, the nasal cycle and the air-conditioning of the air stream are considered.

Airflow

During quiet breathing, a healthy person utilizes only nasal breathing. Nasal volume is about 16-20 ml (Bouhuys, 1974). Bearing in mind that on average a normal person at rest breathes about 16 times per minute and that the volume of the inspired air is about 500 ml, it follows that the nasal air changes approximately 30 times in 4 seconds (English, 1976; Hilding, 1976).

Because of the inaccessibility of most of the airway in a living subject, most studies of airflow have been conducted using models (Daves, 1952; Proetz, 1951). From these studies it seems that there is only a small difference regarding the pathway of the inspired and expired air. Factors determining this pathway are:

1) the direction of the anterior nares
2) the shape of the nasal vault, and
3) the smallness of the nasal valves when compared to the size of the posterior choanae (Douek, 1979; Ballenger, 1969).

The inspired air entering the anterior naris is directed upwards for 1.5 to 2 cm and then converges to pass through the smaller cross section of the nasal valve. Then the air turns at an angle of 60° to 130° from the initial direction to pass primarily along the

middle meatus with a lesser stream along the nasal floor (Proctor, et al., 1973; Martensson, 1956). Finally at the choanae there is another turn of about 90° (Proctor, 1977).

In expiration the air enters the choanae from the nasopharynx and follows mostly the same route as the inspiratory current (Douek, 1979). However, anteriorly when meeting the constriction of the upper limit of the vestibule the airstream is divided into a portion which passes out through the nostril and another which forms a large central eddy, whirling back through the inferior meatus and rising to join the main stream from the nasopharynx (Douek, 1979; Ballenger, 1969). Part of the central eddy passes under the middle turbinate.

The type of airflow is controversial, since in quite breathing some investigators describe it as laminar[1] (Tonndorf, 1958) some as atypical (Bouhuys, 1974) and finally others as mixed that is partially laminar and partially turbulent[2] (Drettner, 1978; Mygind, 1978; Hilding, 1976; Lanier and Tremblay, 1979).

From a theoretical point of view the type of airflow depends on the Reynold's number, which in turn depends on the velocity of the flow, the viscosity of the air, the density of the air and the diameter of the tube (Ballenger, 1969; Spoor, 1967). There is a critical value after which the airflow becomes turbulent. For simple flow in pipes, this critical value is 2000 (Solomon and Stohrer,

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[1]The laminar flow is characterized by parallel streams.
[2]The turbulent flow has an irregular character with eddies.

1965; Comroe, 1965). However, Num (1977), claimed that when Reynold's number is less than 1000, the flow is laminar and above a value of 1500 the flow is entirely turbulent. In between these values both types of flow can exist. The flow within the nose is turbulent as the air passes through the narrow spacers, over irregularities such as the conchae and septal ridges and as it makes directional changes (Num, 1977; Hilding, 1976; Graig et al., 1956). Lanier and Tremblay (1979) pointed out that turbulence and air-flow patterns are considerably different during inspiration and expiration, and that the turbulence produced by expiration is much greater.

The flow rate is measured by special devices, called flowmeters. The most widely used instrument is the pneumotachograph and another less commonly used device is the warm-wire anemometer.

The pneumotachograph operates on the principle that as air flows across a resistance the pressure resultant difference is linearly related to airflow rate. It contains a flowmeter, and the resistance is provided by a wire mesh screen. A differential pressure transducer is included with pressure taps on each side of the screen. The pressure difference is converted to an electrical voltage which is amplified and recorded.

The warm-wire anemometer uses an electrically heated wire as the sensory unit. The cooling effect of the airflow alters its resistance, and the resultant change in voltage is amplified and recorded.

Air movement through the nose is produced by a pressure difference between the nasopharynx and the atmosphere during each inspiration and expiration. This ranges in quiet breathing between -6 mm to +6 mm of water (Meyerhoff, 1980; Douek, 1979). Other investigators (Taylor, 1983) reported a negative pressure of about 10 mm H_2O lasting for 2 seconds, and a positive pressure of about 8 mm H_2O lasting for 3 seconds. As the pressure difference increases so does the airflow, until a critical pressure is reached at which point the nasal airflow becomes constant even if the pressure difference increases (Bridger and Proctor, 1970). When the pressure difference is positive air is moving out, and when negative air is moving in.

Different methods exist to measure the pressure difference (also called pressure change, pressure drop, driving pressure or pressure differential) between the nasopharynx and the exterior. Actually, pressure difference and airflow rate are recorded simultaneously. The procedure used to measure air pressure and the rate of airflow within the nasal airway during breathing is called rhinomanometry. The methods used can be categorized as 1) those that record variations of pressure difference and airflow rate simultaneously, while the individual actively initiates his own respiration, and 2) those which measure the one variable by keeping the other constant. Maran et al., (1971) stated:

> "Any method of rhinomanometry must fulfill the following criteria:
> 1. There must be no interference with the anatomy of the nose; this precludes introducing measuring devices into the nares or nasopharynx since they alter airflow characteristics.

2. There must be a physiological airflow through the nose with normal intranasal pressures.

3. The method must be comfortable for the subjects and should involve the use of warm humidified air.

4. It must be simple to perform on the part of the subject. This is difficult to achieve since nearly all methods depend on palatal relaxation which many people find difficult.

5. It should be repeatable.

6. If it is to be used clinically it should be easily constructed, inexpensive and easily maintained."

It is obvious that value taken by applying the second methodology are invalid not only because the methods are non-physiologic but also because these two parameters vary from hour to hour among normal subjects and even more in subject with a deranged nasal complex (Moran et al., 1971, Principato and Orenberger, 1970). Factors such as air temperature (Drettner, 1961), head posture (Olow and Greve, 1979) and body position (Rundcrantz, 1964) have been shown to influence these parameters.

The most effective method used to date for simultaneously recording of pressure difference and nasal airflow rat is "posterior mask rhinomanometry" (Kern, 1983).

Nasal airway resistance

As air passes through the nose, certain factors impede the air flow. Collectively, the impeding factors are referred to as "resistance". Of total respiratory resistance, the nose accounts for approximately 30% (Douek, 1979; English, 1976), 50% (Grundfast, 1983) or 50-70% (Hilding, 1976; Ferris et al., 1964). The major

resistance effect is provided by the nasal valve (Proctor, 1977). Factors affecting the nasal airway resistance are: 1) nasal vascular congestion, 2) body position, 3) pharmacologic agents, 4) ambient temperature, 5) nasal diseases, 6) shape of the nasal cavity, 7) emotions and 8) head posture (Grundfast, 1983; Meyerhoff, 1980; Solow and Greve, 1979; Proctor, 1977; Gottle, 1960).

It should be emphasized that nasal resistance is not measured directly. Resistance is a derived or calculated quantity. Some investigators, by measuring pressure difference between the nasopharynx and the environment and also the airflow, calculated the nasal resistance using the basic principle expressed by Ohm's law (Kern, 1983; Sarver, 1979; Warren, 1979; Hershey et al., 1976; Warren et al., 1974; Solomon et al., 1965). However, Ohm's law is valid only when flow is laminar, in which case the quantity of air passing through a conducting system is directly proportional to the pressure difference between the two ends (Foxen et al., 1971). Since the nasal airflow is partly laminar and partly turbulent, values for nasal resistance taken by applying the Ohm's law should be accepted with reservation.

Other investigators, in an attempt to overcome this problem, proposed more complex formula for nasal resistance calculation (Nolte and Luder-Luhr, 1973; Richerson and Seebohm, 1968; Spoor, 1967; Solomon and Stohrer, 1965).

Although many investigators have ignored the fact, inspiratory and expiratory nasal resistance levels are not equal (Craig et al., 1965; Ferris et al., 1964).

Nasal Airway Resistance and Palatal Expansion

Although the otologist Eysell, almost 100 years ago, conceived the idea for influencing the internal volume of the nose by expanding the maxilla, the subject has only recently received appropriate attention. While there seems to be agreement regarding the structural effect of palatal expansion on the nose (Wertz, 1970; Wertz, 1968; Linder-Aronson, 1968; Haas, 1961), that is lowering of the palatal vault and straightening the deviated septum, argument exists regarding the effect on the nasal resistance. Watson et al., (1968), Linder-Aronson (1968) and Hershey et al. (1976) pointed out that there is a reduction in nasal resistance but Wertz (1968), failed to demonstrate it.

Nasal Resistance and Mouth Breathing

The nasal resistance to airflow is about twice that of the mouth (Bouhuys, 1974). As stated, during quiet breathing the nasal passages contribute nearly 50% of the total flow resistance. People normally prefer to accept the extra effort that breathing through the nose requires and only turn to mouth breathing during sound production and exercising. What controls the mechanisms that determine breathing mode is not clear. However, it seems possible that nasal airway resistance plays an important role. Based on the fact that the organism must be supplied with a certain amount of oxygen in a given period, it seems that when the nose cannot supply this amount, due to a high resistance, mouth breathing is induced. This critical value for nasal resistance has not yet been fully estab-

lished. However, Patrick and Sharp (1970) found this critical value to be 4 cm H_2O. Watson et al. (1968) on the other hand reported that when the nasal airway resistance was higher than 4.5 cm/L/sec, 77% of their sample presented mouth breathing.

Nasal cycle

The nasal cycle, a term proposed by Kayser in 1895 is a phenomenon in which alternating changes in patency of the two nasal cavities occur. One or the other nasal cavity may be partially or completely obstructed at a given time (Taylor, 1983). Kayser was the first to notice these changes, and he suggested that there is a continual shifting in autonomic balance between the two body halves, which in turn causes a continually changing blood balance in the erectile-like tissues in the mucosae of the turbinates and septum.

In 1923, Lillie took up this problem and noticed that as one nasal cavity opens and its mucosal glands express their secretions, the erectile tissues of the opposite nasal chamber becomes engorged and the mucosal glands diminish their secretions. Heetderks (1927) in a clinically and more comprehensive and careful study confirmed Lillie's observations. He reported that an individually characteristic nasal cycle, ranging from about 30 minutes to 5 hours, from one corresponding phase to another, is present in about 80% of normal individuals while in an upright posture. He also found that a damp and cold atmosphere created the greatest swelling of the mucosa, while dry and warm air created less swelling. Optimal atmospheric conditions (temperature 15-18°C, humidity 50-60%) caused cycles of the least degree.

Hasegawa and Kern (1977), Stoksted (1976), Arbour and Kern (1975), Stoksted (1952), applying rhinomanometry confirmed these original observations. The initiating factor in the nasal cycle has not been identified. Based on the studies of Keuning (1968) and Connell (1972), it is now generally accepted that the cycle is controlled by the autonomic nervous system. Sympathetic impulses mediate vasoconstriction and thus mucosal shrinkage of the turbinates, while the parasympathetic system mediates mucosal swelling.

What actually takes place in the turbinates during the cycle has not yet received much attention. A swelling of the mucous membrane may be due to arterial hyperaemia, venous engorgement, edema, or any combination of these. The temperature of the mucosa on the swollen side is usually higher than that of the shrunken side, but this does not necessarily indicate that the swelling is caused by arterial hyperaemia. The nasal mucosa is cooled by inspiratory air and this effect is less absent on the swollen side (Hilding, 1976).

If one side of the nasal airway is abnormally narrow and the other abnormally wide, much less if any airflow occurs through the narrow side (Hilding, 1976). Unless bilateral obstruction is such as to force mouth-breathing, this air will be adequate for respiratory requirements. However, if conchal congestion on the wide side, due to nasal cycle, is severe, a temporary nasal obstruction with dyspnea and mouth breathing may occur.

Hadegawa and Kern (1977), Principato and Ozenberger (1970), Guilerm et al. (1967) and Spoor (1967, 1963), demonstrated that the

total nasal resistance remains constant in spite of continuous cross sectional changes caused by congestion and decongestion of turbinal and septal erectile tissues in the separate nasal airways.

IV) NASAL OBSTRUCTION

Physiological nasal function can be impaired by nasal obstruction, which is the most commonly occurring symptom of disorders of the nose (Capoot, 1976). In evaluating nasal obstruction one must first determine if it is unilateral or bilateral, complete or partial, intermittent or constant, and congenital or acquired. Examination of the nose may determine the anatomic site of obstruction. Depending on the location the possible causes of nasal obstruction can be classified as extranasal, intranasal or nasopharyngeal.

Extranasal

The nasal pyramid is formed by the nasal bones, the maxillary frontal processes and the upper and lower lateral nasal cartilages. The external nares are bounded by the flaring wings of the lower lateral cartilages and the columella which is formed by the media crura of both the lower lateral cartilages and the septum. Deformities of these parts, whether congenital or acquired, can impair nasal airway function.

Lateral dislocation of the upper lateral cartilages is a very common traumatic nasal deformity, occurring both pre- and postnatally (May and West, 1973) and may impair nasal function. Congenitally thin, weak, upper lateral cartilages may be unable to support the alar openings and can "suck in" during nasal inspiration. Excessive narrowing of the lower lateral cartilage or collapse of the alar wings likewise cause inspiratory obstruction. Impaired nasal airway function may also be caused by an excessive long and depressed nasal tip and by a thick and broad columella.

Intranasal

Intranasal obstruction may be caused by hypertrophied turbinates, a deformed nasal septum, nasal polyps, congenital choanal atresia, foreign bodies and rhinitis.

Hypertrophied Turbinates

Hypertrophy of the turbinates, particularly the inferior one, may occur when a combination of allergic rhinitis and low-grade inflammation exists (DeWeese and Saunders, 1982) and when the airway is large.

Extra space available in one nasal cavity can be created by a lateral displacement of the septum into the other (Fairbanks, 1983). Whether turbinate enlargement pushes the septum to become deformed or the turbinate simply enlarges to fill the space created by the septal deformity, is a developmental puzzle. Those favoring the second possibility, reason that hypertrophy is the result of efforts to keep the airconditioning function of the nose intact. The enlargement involves both osseous and mucosal elements and may be observed to approximate or nearly approximate the nasal septum to a degree that produces a mechanical blockage to nasal airflow (Subtenly, 1980).

Deformed Nasal Septum

Anatomically, a perfectly straight midline septum is rare (Teppensen and Windfield, 1972; Kirchner, 1955). Fairbanks (1983), stated that approximately 80% of the population have some deformity of the nasal septum. The asymmetry may affect any or all parts of

the septum, except for the posterior free border at the choanae where it is always midline.

Septal deformities, either developmental or traumatic, can be classified (Walter, 1980) as: 1) Spurs, 2) deviations or 3) dislocations (subluxations). Spurs are sharp angulations which may occur at the junction of the vomer below, with the septal cartilage and/or ethmoid bone above. Deviations are characterized by a more generalized bulge. They are usually shaped into a "C" or "S" and involve both the cartilage and the bone. Gray (1968) subdivides deviations into a cartilaginous type when only the septal cartilage is buckled and a combined type when both bone and cartilage are bent. Subluxations are characterized by a displacement of the septal cartilage from its medial position and a projection into one of the nostrils.

Nasal Polyps

Nasal polyps are the most common kind of growth found within the nose (Tangen, 1959). These are pedunculated pieces of oedematous upper respiratory mucosa and can arise from any part of the nasal or antral mucosa (Ballantyne, 1979). They may be unilateral or bilateral and are sometimes large enough to occlude the nose completely (Tangen, 1959). The most common site of origin are the ethmoidal labyrinths and, less common, the maxillary sinuses. At first they are small but then enlarge and, when fully developed, appear as smooth, pale tumors.

Choanal Atresia

Choanal artresia occurs in approximately 1 in 800 births (Jaffe, 1981) and produces incomplete or complete obstruction of one or both posterior nares. Pracy (1979) stated that the incidence is 1:60,000 of live births, and that unilateral atresia occurs in up to 60% of it. The condition is more frequent in females and is often associated with other malformations (Freng, 1978). The atresia may be membranous or bony (Freng, 1980), but Tanger (1959) describes a cartilaginous type in addition to these two types.

Membranous atresia, occurring in 5-10% of the cases, probably results when the endoderm of the foregut meets the ectoderm of the nasal cavity. If this nasopharyngeal membrane fails to rupture, a membrane persists.

The bony atresia plate, occurring in 90-95% (Skolik et al., 1973) of the cases, is probably formed by a medial outgrowth of the vertical and horizontal processes of the lateral bones. It may be up to 1 cm in thickness and is located about 3 cm from the alar rim (Jaffe, 1981).

Foreign Bodies

The foreign body may enter the nose by itself or it may be placed there by the child or some other person. Children at play are prone to place small objects in the nasal passages. Usually they do this on the right side (Tangen, 1959). The more common foreign bodies found in children are beads, buttons, erasers, peas, beans, nuts, plasticine, pieces of wood, bread, small toys, chewing

gum and seeds (Shapiro, 1983; Tangen, 1959). Essentially any object which can fit into the nose has probably been found there, and the literature contains numerous reports involving unusual foreign bodies.

Rhinitis

Chronic rhinitis can cause nasal obstruction. The most common types are allergic and atrophic rhinitis.

Nasopharyngeal

The nasopharynx can cause nasal obstruction when nasopharyngeal tumors or hypertrophied adenoids exist.

Nasopharyngeal Tumors

These occur almost exclusively in males and arise from fibrous tissue in the sphenoethmoidal recess, the base of the sphenoid and in the nasopharynx. Histologically they represent fibroma, fibroangioma or fibromyxoma.

Hypertrophied Adenoids

Anatomically, the pharyngeal tonsils (adenoids), which appear at about the sixth month of the fetal life (Subtenly, 1980) as a subepithelial infiltration of lymphocytes, lie on the roof and upper posterior wall of the nasopharynx and comprise the upper part of Waldeyer's ring. The bony boundaries are the body of the sphenoid, the basilar portion of the occipital bone and the arch of the atlas. They consist of lymphoid tissue containing five or more anteroposterior tissues, with many tubular crypts extending inward to mucous glands. Their surface is covered with ciliated pseudo stratified columnar epithelium.

According to Scammon et al. (1930) in the lymphoid tissue growth curve there is rapid growth in infancy and early childhood, a slower increase during late childhood and prepuberty, peaking before adolescence and then gradually declining to the adult condition. However, Scammon's lymphoid growth curve was based on postmortem material and, furthermore, the lymphoid tissue used did not include adenoid tissue. Based on this, Scammon's lymphoid growth curve is questionable regarding application to adenoidal growth (Subtenly, 1980; Pruzansky, 1975).

Subtenly (1980, 1954), using cephalometry and laminagraphy, found that adenoid tissue follows a definite growth pattern. It enlarges predominantly downward and forward, rapidly during the early years of life and then at a somewhat retarded rate until its greatest bulk is attained. After the peak of growth is reached, the growth process seems to reverse itself and the adenoid tissue progressively decreases in mass. By adulthood the adenoids have usually atrophied completely. However, Proctor (1977) stated:

> "contrary to common belief, adenoids do not disappear in the adult, although they do decrease in size in late childhood."

In normal persons there is a balance between the growth rate of adenoid tissue and surrounding bony structures. If this balance becomes disrupted then an obstruction can occur. This may be due either to more rapid growth of the adenoid tissue or to lesser growth of the nasopharyngeal cavity.

V) CRANIOFACIAL ASYMMETRIES

Ludwig (1932) reported that in civilized man, right-handedness occurs in about 95% of the population. Related to right and left handedness there is also a preference for using the corresponding leg as dominant. Moreover, there is a preference for using one eye as dominant to the other. These relationships suggest that the preference of one side over the other may relate to the central nervous system (Lundstrom, 1961). It is well-known that the speech-center is situated in the left cerebral hemisphere in right-handed persons and in right cerebral hemisphere in left-handed persons.

The human facial skeleton is made up of certain constituent parts each of which is characterized by individual variations between the right and left sides. Facial asymmetries seen in ancient Greek art imply that the artists of that time were quite familiar with such conditions.

Numerous studies have been carried out on craniofacial asymmetries. The assessing methods used can be grouped as follows:

1) Direct observation
2) Direct measurements
3) Photogrammetry
4) Posteroanterior cephalometry, and
5) Inferosuperior (submento-vertex or basilar) cephalometry.

Direct Observations

Sved (1931), Moore and Huges (1942), Campbell (1950), Herzberg (1952) and Vassal (1953) are among others who studied facial

asymmetries by direct observation. Particularly, Sved (1931), claimed that:

> "an occular examination may oftern be more accurate than an apparently accurate scientific method", while Campbel (1950) stated that:
> "it is not popularly appreciated howaccurate is the eye when it is used as a measuring instrutment."

Direct Measurements

Direct measurements have been applied on both dry skulls and living subjects. Woo (1931) carried out direct chordal and arcual measurements on about 800 skulls from the 26th to the 30th Egyptian dynasties. He found that the human skull is markedly asymmetrical, with the right side often being larger, reflecting the development of the right hemisphere of the brain. The left zygoma and left maxilla, however, were larger. The low third of the face was not investigated.

White (1982) using 73 skulls with no data for age, sex and race applied bilateral measurements and found a tendency toward right-side dominance in size and a tendency for right frontopetalia and right temporopetalia.

Sutton (1968) applied direct measurements on a sample of 1029 people and he concluded that:

> "the mean lateral position of the nose increased with age, but the curves showing the mean position of the chin are of a bimodal shape with marked fluctuations at certain ages".

Farkas and Cheung (1981), using six paired lateral measurements done on the faces of 308 healthy Canadian Caucasians ageds 6, 12 and 18

years, concluded that asymmetries, though not immediately obvious, were very common with the right side dominant over the left.

Photogrammetry (Standardized Photography)

Tanner and Wiener (1949), Burke and Beard (1967) and Burke (1971), used standardized photography to assess facial asymmetry. This method seems to overcome many of the problems of cephalometric measurements such as posing and distortion. Burke (1971), working on 24 pairs of like-sexed twins, found the left maxillary area to have a slight tendency to be larger.

Posteroanterior Cephalometry

Posteroanterior cephalometric radiographs have been used for assessment of craniofacial asymmetries by applying linear, angular analyses and analysis of indices.

Linear Analysis

In this method, a median plane was established and the distances of bilateral points were measured and compared. Ray (1960), pointed out difficulties which arise in investigations involving the use of this plane, and Jackson (1937) concluded that to determine the medial plane of the skull, "is not easy, if at all possible".

Harvold (1951, 1954) and Harvold *et al.* (1959), developed the X-line, a horizontal line drawn between the lateral parts of the zygomaticofrontal sutures. He defined the median plane as one drawn at right angles to the horizontal line and through the root of the crista galli.

Sassouni (1960) recommended a supraorbital line, a translateral orbital line, a bizygomatic line, a bimastoid line and a bigonial line. These lines are ideally parallel. The median plane is established by tracing a bisecting plane. When they are not all parallel, then the three which are parallel are used and the bisecting line is drawn.

Cheney (1961) defined the median plane as one which passes through nasion and the anterior nasal spine.

Fisher (1954) modified Harvold's X-line and used a plane bisecting the X-line as a median plane. Mulick (1965) recommended tracing the transzygomaticofrontal suture plane, the transporionic plane - utilizing either machine registration or anthrometric poria - and the transforamen rotunda plane. The median plane can be established as a perpendicular to any of these three planes, drawn through midline anatomic structures such as crista galli, midpalatal suture and vomer, or as an absolute vertical drawn through major midline anatomic structures.

Angular Analysis

Letzer and Kronman (1967) applied a proportional angular analysis to avoid linear measurement problems and concluded that facial asymmetry is a normal finding.

Hewitt (1975), Vig and Hewitt (1975) and Shah and Toshi (1978), applied a triangulation method in which each side of the face was divided into several triangles representing the component areas of the face.

Analysis of Indices

Chebib and Chamma (1981) introduced indices for the assessment of craniofacial asymmetry using coordinates obtained from P-A cephalometric radiographs. The indices were developed utilizing two axes, one based on median plane points and the other on midpoints between bilateral structures.

Inferosuperior Cephalometry

Berger (1961, 1964) proposed the use of the basilar cephalometric view. He traced a midline through the odontoid process of the axis, the anterior tubercle of the atlas and the outline made by the vomer and the crista galli.

Nahoum et al. (1964) suggested the verticosubmental projection which, when combined with the conventional lateral and posteroanterior view, was said to provide a more informative three-dimensional image.

Marmary et al. (1979) accepting Moss and Salentign's (1971) view that certain landmarks of the skull remain constant through growth and development, connected the centers of the foramina spinosa and drew a midperpendicular which used as the medial plane.

Cook (1980) discussing the importance of the Basilar cephalometry stated:

> "The main point that emerged from the study of the SMV (submento-vertex) films was that some cases of facial asymmetry are associated with asymmetry of the skull base and it seems possible that they have a different etiology from those with symmetrical skull base"

VI NASOMAXILLARY GROWTH

What is growth?

"a measurable increase of a tissue, organ or system of organs or organism." Baume (1961).

"the net balance of mass produced and retained over mass destroyed and otherwise lost." Goss (1964).

"any change, in time, which is measurable: length, volumes concentrations, pressures etc." Moss (1964).

The nasomaxillary growth in general is a complex one and this is due not only to its embryological developmental and structural variety but also to its multifunctional nature. The nasomaxillary complex grows by two fundamental processes: 1) by bone deposition and resorption (growth remodeling) and 2) by bone displacement (Enlow, 1982).

Enlow (1963, 1964, 1968, 1982), Enlow and Bang (1965), and Enlow and Hunter (1966) offer a unique account as to where remodeling takes place.

Displacement, defined as a physical movement of a whole bone is classified as either primary or secondary. The primary type takes place in conjunction with a bone's own enlargement, while secondary displacement is not directly related to its own enlargement.

While the remodeling growth and secondary displacement are almost universally accepted, controversies exist regarding the source or sources of the force responsible for producing primary displacement.

Theories presented in the past have located the source of this force in the sutures, nasal septum, the soft tissues and the oro-

nasopharyngeal spaces (functional matrix theory), and the periosteum (intrinsic maxillary-displacing growth theory).

Sutures

The fibrous connective tissue of growing sutures has been described as composed of 3 layers (Weinmann and Sicher 1955; Sicher and Dubrul 1975) or 5 layers (Prichard et al., 1956; Seiton, 1964).

The 3-layered concept claims that the growing sutures, being a remnant of the membranous part of the cranial model, consist of a middle, highly cellular zone with irregularly oriented collagen and precollagen fibers, while the two peripheral layers adjacent to the bones consist of dense connective tissue, whose fibers continue into the bones as Sharpey's fibers. The 5-layered concept accepts the sutures as a union of the periosteal layers of the contiguous bones with bilateral cambial and fibrous layers separated by a middle, highly vascular layer. While Pritchard et al., (1956) stress that five tissue layers can be clearly discerned in adult sutures, Enlow (1968), and Latham (1971) claim that sutures are reduced to a single fibrous membrane after completion of growth.

Are sutures participating in craniofacial growth? Brash (1934) using vital dyes on pigs concluded that the sutures play no significant role in the growth of the skull. However, probably of lacking data, the dyes were given after sutural growth was almost completed. The concept that sutures are areas of growth has been demonstrated by:

I) Biometry (Gans and Sarnat, 1951; Selman and Sarnat, 1955; Bjork, 1966)

II) Histology (Moss 1954; Pritchard et al., 1956)

III) Vital dyes (Moore, 1949; Massler and Schour, 1951; Baer, 1954) and

IV) Autoradiography (Dixon, 1961; Young, 1962).

Sutural growth, according to Moore (1949), Weinmann and Sicher (1955), Sicher and Dubrul (1975) and Dubrul (1980), is active and autonomous.

Prahl (1968) while supporting the above view, concluded that sutural fibers also have a separating force in cranial growth.

In an effort to make the nature of sutural growth clear researchers have been able to show that:

a) Extirpation of the sutural area in facial and cranial sutures in experimental animals show no inhibitory effect on growth in the region (Moss, 1954; Selman and Sarnat, 1957; Stenstrom and Thilander, 1967).

b) Excision and autotransplantation of sutures in a neutral environment (intracranially or intraperitoneally) do not show any bone separation (Moss, 1957; Watanabe et al., 1957).

c) Culture of sutural tissue fails to grow (Petzovic et al., 1968; Lewin and Irwing, 1970).

Based on the above findings and on the fact that bone resorbs under pressure, the theory that sutural tissue growth generates the required force to push the bones apart has been rejected. Instead, it is universally accepted now that the sutural growth is passive and bone deposition along the borders is of a filling in nature,

secondary to the bone separation caused by forces of extra-sutural origin.

However, Azuma et al., (1975) and Enlow (1982) put forward another view regarding the source of the force causing sutural bone separation. According to them, myofibroblasts - a special type of fibroblast - exist at the linkage zone of sutures. These cells, having contractile properties, provide at least in part a bio-mechanical system contributing to displacement of the nasomaxillary complex downward and forward. Other types of fibroblasts function to remodel the collagen fibers and the ground substance involved in relinkage changes.

Nasal Septum

Scott (1953) reported:

> "In the growth of the upper part of the face, however, no satisfactory mechanism has been postulated to account for the separation of the bones at the sutures...
> ...It would appear more probable that separation of the adjacent bones must take place at the sutures before bone growth can take place and that this separation must be brought about by the growth of some organ like the brain or eyeball or of a tissue like cartilage...
> ...The cartilage of the nasal septum is an extension of the cartilage of the cranial base. In its growth it separates the facial bones from one another and from the cranial portion of the skull and allows growth to take place at the sutures by the ordinary mechanism of surface deposition."

This view, although not new, was the beginning of the famous "nasal septum" theory. Subsequently, this theory received a large number of laboratory studies to test its validity. Organ culture, extirpation, enzymatic interference, and immunology are some of the methods which have been used. The studies done can be divided into

those supporting nasal septum theory (Ronning, 1968; Sarnat and Wexler, 1967; Sarnat, 1970; Kremenak and Searls, 1971; Krinnsland, 1973, 1974) and those rejecting it claiming that the nasal septum growth is secondary and totally compensatory in nature (Moss et al., 1978; Stenstrom and Thilander, 1970, 1972; Moss, 1976).

Functional Matrix Theory

Moss and Moss et al. in a series of reports (1960, 1962, 1964, 1967, 1968a, 1968b, 1969a, 1969b, 1971,1975) formulated the functional matrix theory. In brief, this concept states that the head is a composite structure, operationally consisting of a number of relatively independent functions: neural integration, balance, audition, vision, olfaction, respiration, speech, swallowing, mastication and digestion. Each function is carried out by a separate functional cranial component. Each component consists of all of the tissues, organs, functioning spaces and skeletal parts necessary to carry out a given function completely. The tissues, organs and functioning spaces taken as a whole comprise the functional matrix, while the osseous and cartilaginous tissues related to this specific functional matrix represent the skeletal unit.

Functional matrices are divided into the periosteal functional matrices and are defined as those acting directly and actively upon their related skeletal units, e.g. muscles, teeth, blood vessels, nerves, fat pads and salivary glands and the capsular functional matrices acting indirectly and passively on their related skeletal units, producing a secondary, compensatory translation (displace-

ment) in space. The later are subdivided into the neurocranial matrix (brain, cerebrospinal fluid, leptomeninges) and the orofacial matrix (oral, nasal and pharyngeal cavities). The neurocranial matrix is enclosed by the neurocranial capsule (from outside to inside the epidermis, corium, subcutaneous tissue, galea aponeurotica, loose connective tissue, pericranium (periosteum), bone, endocranium and dura matter). The orofacial matrix is enclosed by the orofacial capsule (everything between the skin and the mucosa lining the above mentioned cavities).

In other words, according to the functional matrix theory, the expansion of the oronasopharyngeal cavity is the source of the force which causes primary displacement.

The following quotations point out certain relationships:

Moss (1962)

"The hard palate simultaneously serves two distinct functions, respiration and digestion."

"It seems clear that the respiratory function is primarily responsible for the position of maxillary corpus in space."

"Deficiency in growth of the phylogenetically - and ontogenetically old nasal capsule will, on the other hand, seriously disturb maxillary position."

Moss et al., (1968)

"The growth of the upper face is, in part, a response to the functional demands for increased respiratory volume"

"The growth of the mid-facial skeleton does not depend in any way upon the presence of the nasal cavity or nasal septum."

Moss and Salentijn (1969a)

"The oronasopharyngeal functioning space is particularly related to the relatively dominant cranial respiratory functional space volume."

Moss and Salentijn (1969b)

"Although this functioning space comes into being under genetic (intrinsic) control, its subsequent growth and maintenance in being are functionally (environmentally, extrinsically) determined."

Maxillary Periosteum

Latham (1968, 1970, 1971(a), 1975) and Latham and Scott (1970) offered the following explanation regarding the maxillary displacement growth process.

Maxillary displacement involves two different force-generating mechanisms. The first, being active during early prenatal life, is located in the nasal septum, which seems to apply a tractive force via the septomaxillary ligament. The second comes into play during late prenatal life, replacing gradually the role of the nasal septum. The displacing force for the second mechanism originates from the cellular proliferation in the osteogenic layer of the relatively large posterior and superior maxillary periosteum, which acts against the surrounding mass of fatty tissue to cause maxillary displacement.

VII GROWTH OF NASAL CAVITIES

Surprisingly, only a few studies have been carried out related specifically to the growth of the nasal cavities.

Scott (1959) stated that during the early part of foetal life the growth of the nasal cavity is regulated entirely by the growing cartilage of the nasal capsule. In later foetal life, with the atrophy of parts of the lateral cartilaginous wall or its replacement by bone, the growth sites are situated at the sutures between the facial bones. The sutures which contribute to the nasal cavity growth are those between maxilla and nasal, lacrimal, facial ethmoid, palative and vomer. Bone deposition and resorption also contributes to the nasal cavity enlargement.

A line drawn between the lower margins of the orbital cavities divides the nasal cavities into an upper (interorbital) and a lower (maxillary) region. At one year of age the upper part is about twice the height of the lower part and is about 60% of its adult size, while the lower part is only about 25% of its adult size. By the 7th year upper nasal height has attained more than 80% of adult size and the lower part about 50%.

The width of the upper part of the nasal cavities has reached 75% of its adult size by the end of the first year and over 90% by the 7th year. The depth of the nasal cavities has reached its adult dimensions by the 7th year.

The whole nasal region of the infant is vertically shallow (Enlow 1968, 1975, 1982). The level of the nasal floor lies just

below the inferior orbital rim. However, in the adult, the midface has become greatly expanded and the nasal floor has descended well below the orbital region. The growth of the nasal cavities follows the general mechanism by which the whole face grows, that is, remodeling and displacement. A characteristic point is that the orbital and nasal floors are displaced in the same direction (they are parts of the same bone), but they undergo remodeling growth in opposite directions (Enlow, 1982). The remodeling growth plan for the nasal cavities has been described by Enlow (1982):

> "With the exception of the uppermost part of the roof in each nasal chamber, the lining surface of the bony walls and floor are predominantly resorptive. The mucosal side of each nasal bone is also resorptive. These regional patterns cause a lateral and anterior expansion of the nasal chambers and a downward movement of the palate; the oral side of the bony palate is depository. The nasal side of the cribriform plate (roof of the nasal chamber) is depository, and the cranial side is resorptive. This enlarges the small, paired olfactory fossae and lowers them in conjunction with the downward cortical drift of the entire anterior cranial floor."

Factors Affecting the Shape of the Nasal Cavities

The nasal index is the ratio of the maximum nasal width to maximum height, multiplied by 100.

$$\text{Nasal index} = \frac{\text{nasal breadth (width)}}{\text{nasal height}} = 100$$

A nasal index between 55-70 denotes a leptorhine or "narrow-nosed" nasal type; between 85-100 denotes a platyrhine or "broad-nosed" type; and between 70 and 85 a mesorhine or "intermediate-nosed type (Negus, 1965). Williams (1956) made it clear that beside the anthropological nasal index, cephalometrical and clinical nasal indices have also been introduced.

The nasal index, regardless as to how measured, has extensively been used to describe nasal form. Glanville (1969), however, questions its validity, basically because nasal height growth and nasal breadth growth differ not only in rate but also in time. Furthermore, Hoyme (1965) and Wolpoff (1968) criticized the use of the index on the grounds that skeletal nasal height is functionally unrelated to nose form. This is because nasion, a point that has little to do with the skeletal nasal aperture, is used as one of the landmarks for determining nasal height.

Wolanski and Charzweska (1967) claimed that nasal dimensions are under rather strict genetic control of a multifactorial mature, and that variation in mean nasal index implies differences in the genetic composition of the populations concerned. However, it is almost universally accepted today that certain factors can affect final nasal form. Climate, is one such factor, and has been studied extensively. Although not a cause-effect relationship, it is believed that the platyrhine nose has been adapted to hot and moist climates and the leptorhine nose to cold and dry (Thomson and Buxton, 1923; Davies, 1932; Coon et al., 1950; Weiner, 1954; Steegman and Platner, 1968; Carey and Steegman, 1981 and Crognier 1981; among others).

There is strong evidence also that climate affects head form. Crognier (1981) and Beals (1972) showed that hot climate favors the dolichocephalic type and cold, the brachycephalic type. These authors are of course in accordance with those relating climate and

nasal form, since a dolichocephalic head is accompanied by a leptorhine nose and a brachycephalic with a platyrhine one (Enlow, 1982). The latter could be used to suggest a close relationship between head and nasal form, although it is not clear which affects what.

Glanville (1969) studied the relationship between the position of the upper canine teeth and nasal breadth. This association is most obvious in some of the apes but even in man the long canine roots may reach to a level equal or beyond the lower border of the pyriform aperture. Stress on the canines from biting and tearing food is neutralized in part by the bony butresses on either side of the nasal opening and a relationship between intercanine distance and nasal breadth thus seem possible. Using a sample of about 486 skulls including Caucasian and Black populations, Glanville found a high relationship between intercanine distance and nasal breadth. The relationship has been also reported by Schwalbe (1887, cited by Wolpoff, 1968). Hylander (1975) without rejecting cold-hot nasal adaptability, pointed out the significance of biting forces on the nasal form.

Another factor, which seems to affect nasal form is the breathing mode. It has been shown that mouth breathing is accompanied by a typical facial appearance, the so called adenoid face. A typical adenoid face is characterized by narrow nasal airways, contracted maxilla, long face, lip incompetence, a v-shaped upper dental arch and a high palate. However, the relationship between mouth breathing and the adenoid face is a controversial point and

the existing views range from those denying any relationship (Huber and Reynold, 1946; Ballard and Gwynne-Evans, 1958; Humphrey and Leighton, 1950; Tsui, 1980) to those accepting almost a direct cause-effect relationship (Rickets, 1978; Quin and Pickrell, 1978 ; Quin, 1978 ; Washbon, 1983). Between them are those holding that there is evidence for close relationship but not a a cause-effect level (Linder-Aronson, 1960, 1970, 1974; Harvold et al., 1981).

STATEMENT OF THESIS

Form the review just presented it is apparent that the mechanisms of nasal growth and the factors controlling it are not fully understood. While there is some evidence that a relationship exist between nasal form and head form and intercanine distance, it is not known how this relationship is determined. In other words, whether the nose has some effect on the head and maxillary dentition form, or if the latter have some effect on nasal form is uncertain.

It is almost universally accepted that the functional requirements of an area determine at least in part its morphology. While this concept applies well to some functions, e.g. muscles, kidneys, some doubt exists when other functions are considered. The problem becomes more complex when an area, such as the nasal cavity, is multifunctional, because if a function-form relationship is accepted then it should be determined which function is the more important and how each function contributes to the final form.

Bearing in mind the current views regarding the possible effects of nasal functions on the growth pattern of the nasal cavities, it is apparent that they are oversimplified and not fully documented. It was therefore decided to test the effect of two nasal functions, that is, inspiration and airconditioning, on nasomaxillary form. These functions were disturbed by suturing one side of the nasal airway. Although the reason for existence of two nasal airways has not yet been adequately accounted for, it was assumed that the presence of both is important for normal nasal function.

If these two functions, (inspiration and airconditioning), have an effect on the growth of the nasomaxillary complex, it was theorized that the blocked (nonfunctional) side will show growth retardation, and the functional side will experience growth acceleration.

If the functional matrix theory is correct in the statement that

> "These functioning spaces (oronasopharyngeal) are genetically determined and functionally maintained. Specifically, I believe that the functioning spaces first are formed in a process of enclosure by the genetically determined growth of the primordial capsular tissues. Once formed these spaces are maintained by their own functioning" (Moss, 1968)

then it follows that the blocked nasal airway should be replaced by some other reactive tissues.

METHODS AND MATERIALS

Sample

The present investigation employed 19 CWR: (HA) albino guinea pigs, provided by the Animal Facilities Department, Case Western Reserve University. The age of the animals when delivered was about three weeks. They were kept for the entire experimental period in metalic cages with meshed floors and were fed guinea pig chow. Once per week fresh lettuce - as source of vitamin C - was provided to them. All cages were kept in the same room. The guinea pig was chosen as the experimental model for the following reasons:

1. It is a relatively large animal, thus facilitating the experimental work.
2. Its craniofacial growth period is relatively short, extending up to 5-6 months of age (Manson, 1968; Stenstrom and Thilander, 1972).
3. Its nasal septum is complete.
4. The occlusal surfaces of the molars are flat, thus avoiding intercuspation interference.
5. It is a relatively inexpensive animal to purchase.
6. It is docile and easy to handle.

Experimental Procedure

The experimental period (Table I) lasted 120 days, after which the animals were sacrificed. From the 19 animals, 13 were used for the experimental group and 6 as the control group. The experimental procedure was the following:

TABLE I

Summary of Experimental Procedure

	Work Done								
Group	Day 1	Day 4	Day 7	Day 12	Day 50	Day 68	Day 83	Day 98	Day 120
Experimental	Anesthesia Cephalometric X-rays	Anesthesia Repeat Block of nose	Anesthesia Repeat block of the nose	Anesthesia Suture of right nasal opening	Anesthesia Cephalometric X-rays	Anesthesia 1st vital dye	Anesthesia 2nd vital dye	Anesthsia 3rd vital dye	Sacrifice
Control	Anesthesia Cephalometric X-rays	Nothing	Nothing	Nothing	Anesthesia Cephalometric X-rays	Anesthesia 1st vital dye	Anesthesia 2nd vital dye	Anesthesia 3rd vital dye	Sacrifice

Day One (1)

On day one, after anesthesia, a dorsoventral cephalometric X-ray was taken for each animal in both groups. In the experimental group the right nasal airway was then blocked.

Anesthesia was provided by intramuscular injection of a combination of ketaset (ketamine HCL, Bristol Laboratories, NY 13201) 50 mgr/kgr body weight and ACE-Promaxine 0.5 mgr/kgr body weight. Although this combination does not produce general anesthesia, however the induced immobilization was adequate for our procedures.

The cephalometric X-rays were obtained by using cephalometer (Plate I), modified after Palmer (1978). This cephalometer was mounted on a Bolton's cephalometric apparatus as for a cephalometric cassette. Thus the distance between the X-ray source and the object was constantly maintained. All the standards for a human postero-anterior cephalometric X-ray were followed in this procedure. The exposure was under 90 KVP, 15 mA and for 3 seconds. Standard occlusal films (Kodak, DF46, Rochester, N.Y.) were used and were processed by an automatic processor to insure consistancy of the technique.

The easiest way to block the nasal airway is by suturing the nasal opening. However, considering the possibility that scar tissue developed on the nares may interfere with the nasomaxillary growth, the nasal airway was blocked with a system of plastic tubes.

Day Four (4)

It was noticed that the plastic tubes were soon removed by the

animals. So, by anesthetizing the animals as on day one the whole technique regarding block of the nasal airway was repeated.

Day Seven (7)

Because the plastic tubes were again rejected the same procedure was repeated but the external tube was stabilized on the nare's rim with four sutures. However after a few days the tubes were again rejected, because the animals used to scratch their nose on the cages's meshed floor. Considering these problems it was finally decided to suture the nasal opening.

Day Twelve (12)

The animals were anesthetized as on day one. Local anesthesia (lidocaine HCl 2% with 1:100,000 epinephrine) was administered for the tissues surrounding both nasal openings. A narrow strip of skin from the nares's rim was surgically removed from both nasal openings, and the right one was sutured while the left one was kept open with a plastic tube (Plate II) until healing was completed. Bilateral skin removal was performed in order to induce a bilateral effect, if any, of resultant scar tissue.

Day Fifty (50)

All the animals were anesthetized and a new set of dorsoventral cephalometric X-rays was taken.

Day Sixty-Eight (68)

After anesthesia, the animals were weighted and the first vital dye (oxytetracycline, 50 mgr/kgr body weight, Liquamycin 100, for I.M., Pfizer) was administered intramuscularly in the thigh.

PLATE I

The modified cephalometer

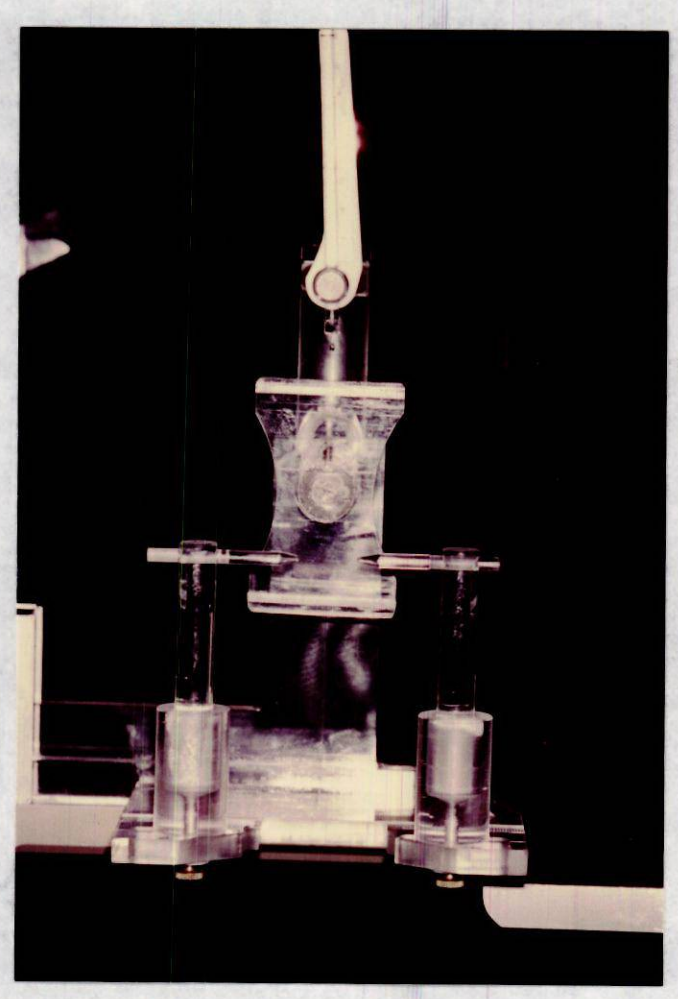


PLATE II

a)Sutured right nasal airway and plastic tube on the left one.

b)The nose after healing



a



b

Day Eighty-Three (83)

After anesthesia, the animals were weighed and the second vital dye [DCAF (2,4 Bis) N,N'Di (carboxy-methyl, amino methyl fluorescien), 15 mgr/kgr body weight, ICN Pharmaceuticals, Inc.] combined with $NaHCO_3$ to a ratio 133 mgr $NaHCO_3$/100 mgr DCAF was administered intraperitoneally. To avoid irritation the injection was performed at a very slow rate.

Day Ninety-Eight (98)

The animals were anesthetized, weighed and the third vital dye (xylenol orange tetrasodium salt, 90 mgr/kgr body wgt, Am. Sci. Products) was administered intraperitoneally at a slow rate.

Day One-Hundred-Twenty (120)

After the animals were anesthetized in the usual way, an intraperitoneal injection of pentobarbital Na at 50 mgr/kgr was administered for deep anesthesia. When there was no reflex response, the heart was exposed and the animals were perfused with 10% buffered formalin phosphate (Fisher Scientific). After perfusion the heads were removed and cleaned of all skin and the majority of the soft tissues. Then they were placed in solution of 10% buffered formalin, the same with that used for perfusion, and kept there for the next 4 days, for more complete fixation.

Laboratory Procedure

The mandibles were disarticulated and each skull was further cleaned of all soft tissues. At this point another set of cephalo-

metric x-rays was taken, using the same cephalostat and standards utilized perviously.

Four skulls (3 experimental and 1 control) were used for decalcified sectioning, while the remaining 15 (10 experimental and 5 control) were prepared for undecalcified sectioning.

Decalcified Technique

Routine steps were followed, including washing in running water, decalcifying, dehydrating, embedding in parafin, sectioning and staining with hematoxylin and eosin.

The decalcification was carried out with rapid bone decalcifier (RDO, Dupage Kinetic Laboratories, Inc., Naperville, IL) for 2½ hours.

After decalcification was complete, the snouts were removed from the rest of the skulls at the level of the first molars. Each snout then was divided into three convenient-sized blocks (Fig. 1) Successive sections were prepared from each block in a direction perpendicular to long axis of the snout. Care was taken to have all sections oriented in precisely the same way.

Undecalcified Technique

The steps followed for this procedure were: washing in running water for 12 hours, dehydration, embedding in uncatalyzed bioplastic (Ward's, Rochester, N.Y.), embedding in catalyzed bioplastic, sectioning and mounting.

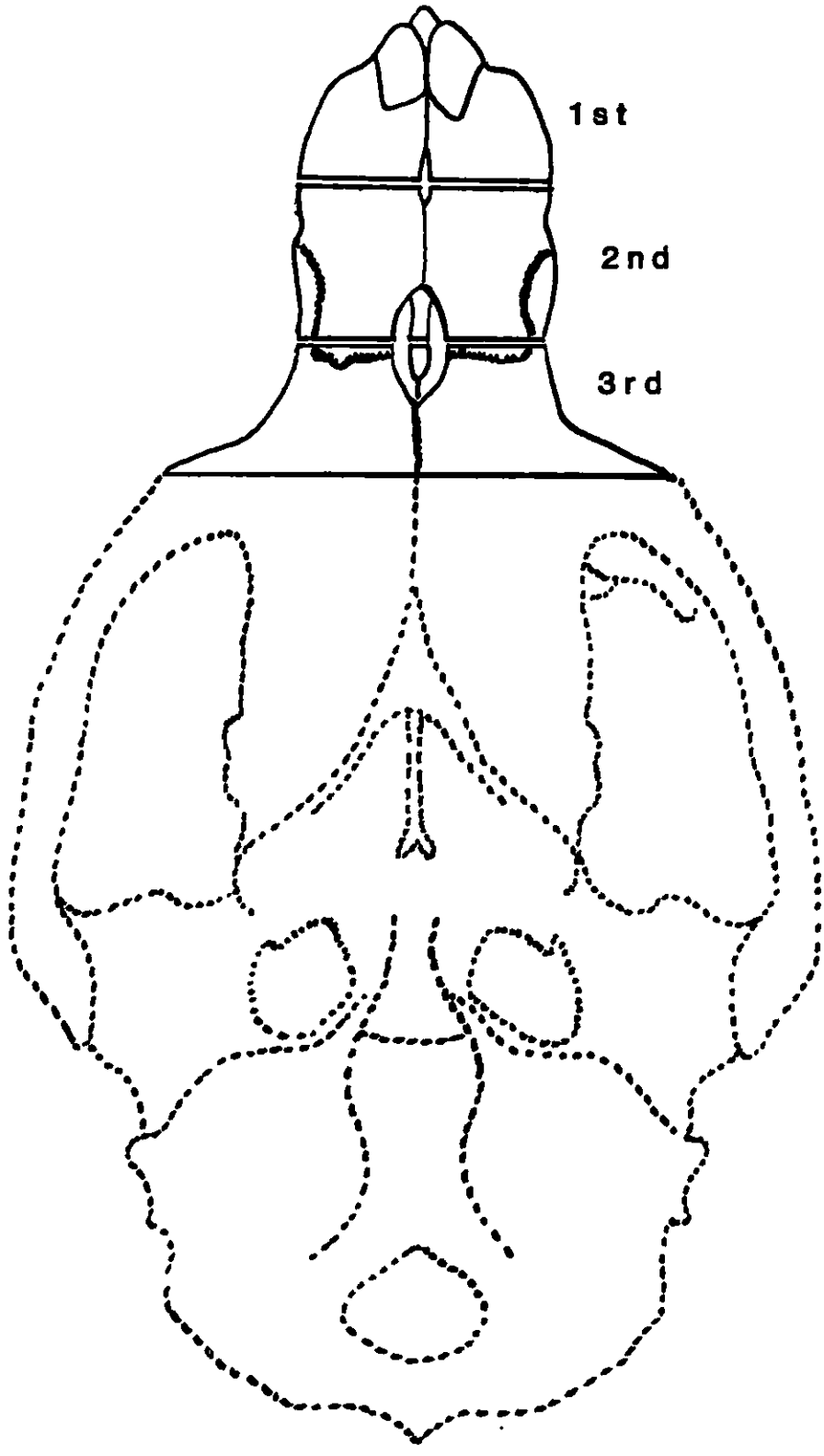
The dehydration was performed as follows:

a) alcohol 50%, 1 day

b) alcohol 70%, 1 day

FIGURE 1

The parts for decalcified sectioning


1st
2nd
3rd

c) alcohol 80%, 1 day

d) alcohol 90%, 1 day

e) alcohol 95%, 1 day

f) alcohol 100%, 1 day

g) alcohol 100%, a day

h) equal parts of alcohol 100% and xylene, 1 day

i) xylene, 1 day

j) xylene, 1 day

For adequate impregnation the specimens were embedded in uncatalyzed bioplastic for 3 days.

In order to obtain standardized orientation of the sections, a specifically designed plastic box (Plate III) was used to embed the specimens into catalyzed bioplastic. In this box the skulls were positioned so that the transporionic axis was parallel with the width of the box, while the long axis - defined as the line passing through the nuchal ridge and the roof of nasal aperture - was parallel with the base (floor) of the box (Plate IV). All the boxes had the same dimensions and the rods were placed in the same position. The boxes containing the skulls and catalyzed bioplastic were cured for 3 days and then were placed for 24 hours in an electrical oven heated at 65°F for more complete polymerization.

A special cutting machine was used for sectioning the specimens. The following points were marked with a pencil on the plastic boxes: One mm anterior to the anterior edge of the incisive foramen, the anteroposterior midpoint between the posterior end of

PLATE III

The plastic box

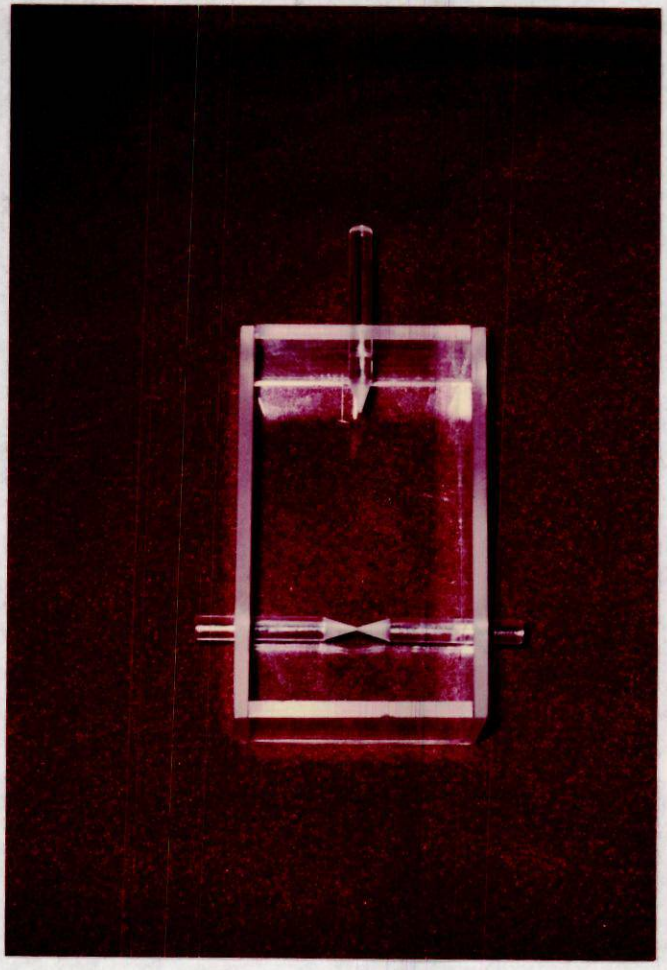
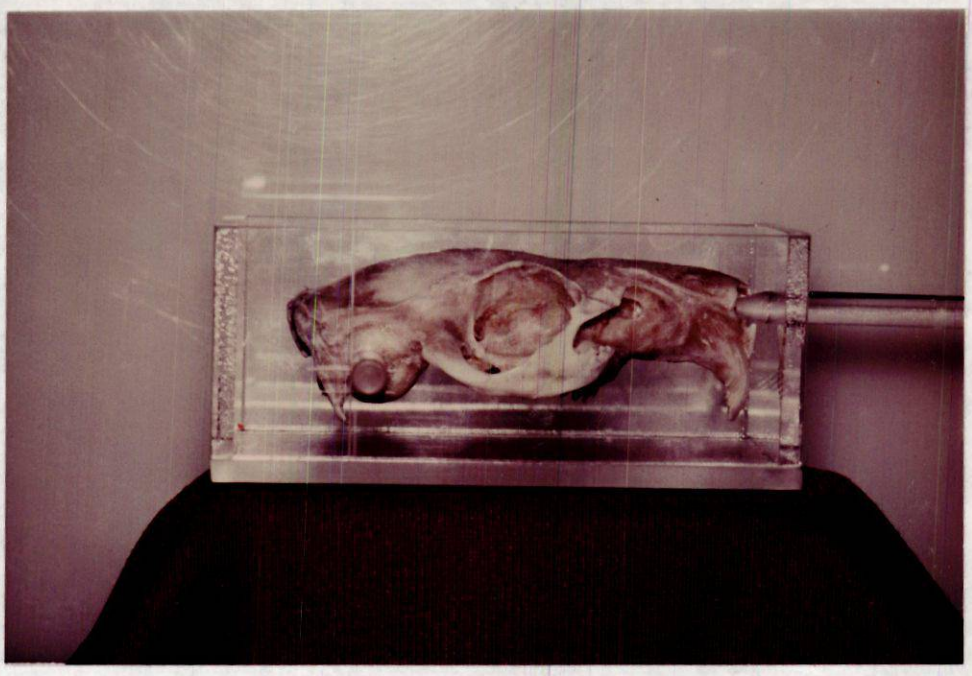


PLATE IV

The skull orientation into the plastic box



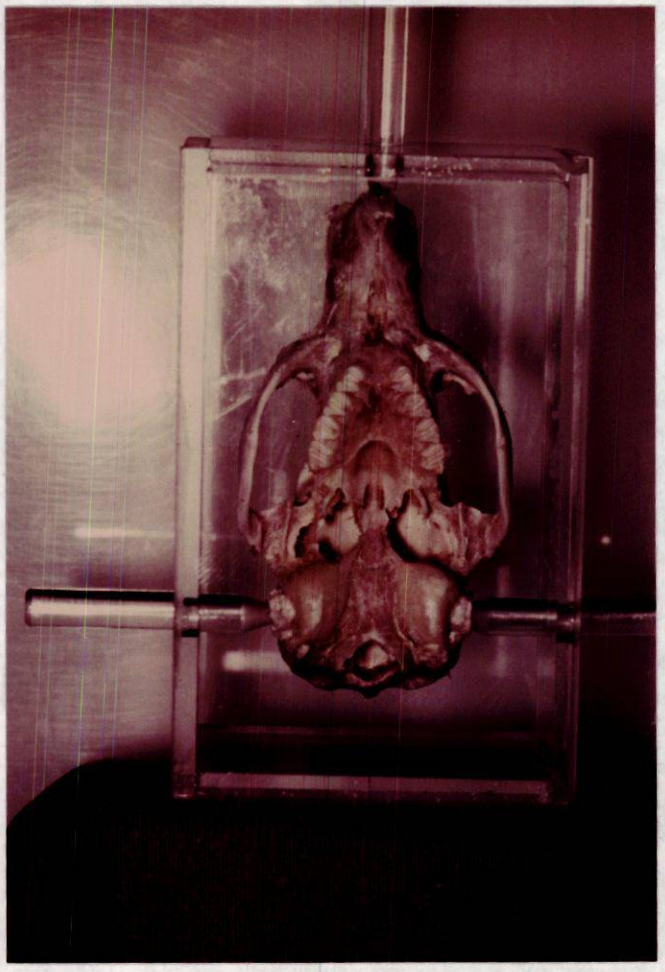
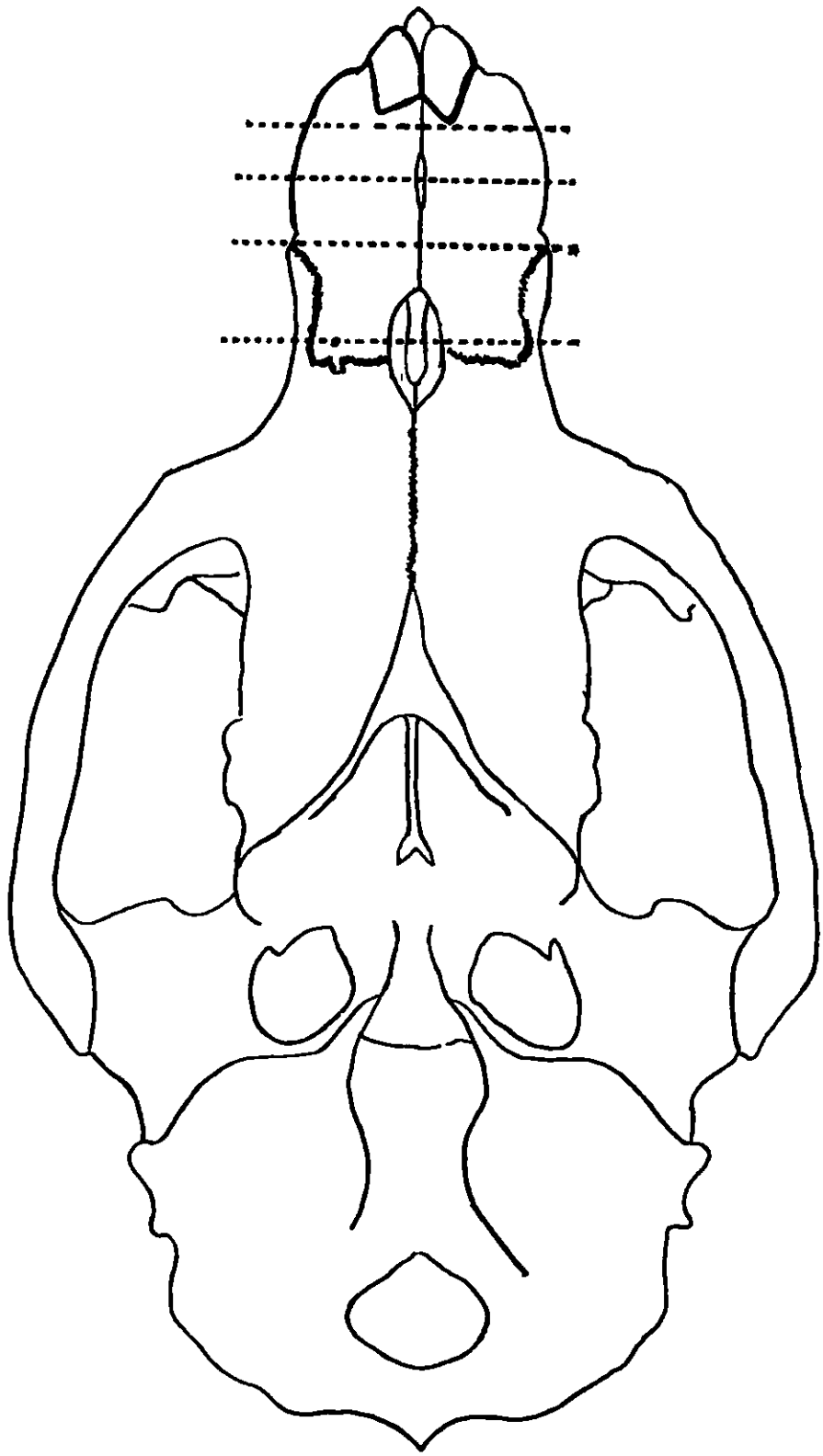


FIGURE 2

The points at which undecalcified sections were taken.



the incisive foramen and the anterior end of the palatal foramen, the anteroposterior midpoint of the palatal foramen and the most anterior point of the right first molar (Fig. 2). Although care was taken to have sections taken at these points, in some animals it did not always work in this way because the desired sections were destroyed during cutting. The sections were parallel with the transporionic plane and perpendicular to the long axis. The width of the sections ranged between 65-75 μ. From each specimen 5 sections were taken.

The sections obtained were cleaned with water and then processed through ascending alcohol (70°, 80°, 95°, 100°, 100°) and xyline (twice) for 2 minutes in each solution. Finally they were mounted on microscope slides using a standard histological mounting medium (Permount, Fisher Scientific) and covered with microscope cover glass.

Data Collection

Gross Observations

The effects of altered nasal function on craniofacial growth were initially determined by noting any asymmetries existing between the right and left sides of each skull that could be identified by visual inspection.

Cephalometric analysis

Cephalometric analysis was based primarily on the last set of x-rays. Because the external meatuses were uncovered by soft tissues, head position errors were minimized. Also, without the

covering soft tissues and with the mandible removed it was possible to locate anatomical points with better accuracy.

The cephalograms were enlarged three times using a photographic copy stand with projection onto acetate paper. To test enlargement error all the films were punched at their corner with a vernier calipers (Helios Calipers, DCA Corporation, Washington D.C.) in a certain distance of 0.5 cm. The following anatomical points were then identified and marked with fine pin-holes (Fig. 3).

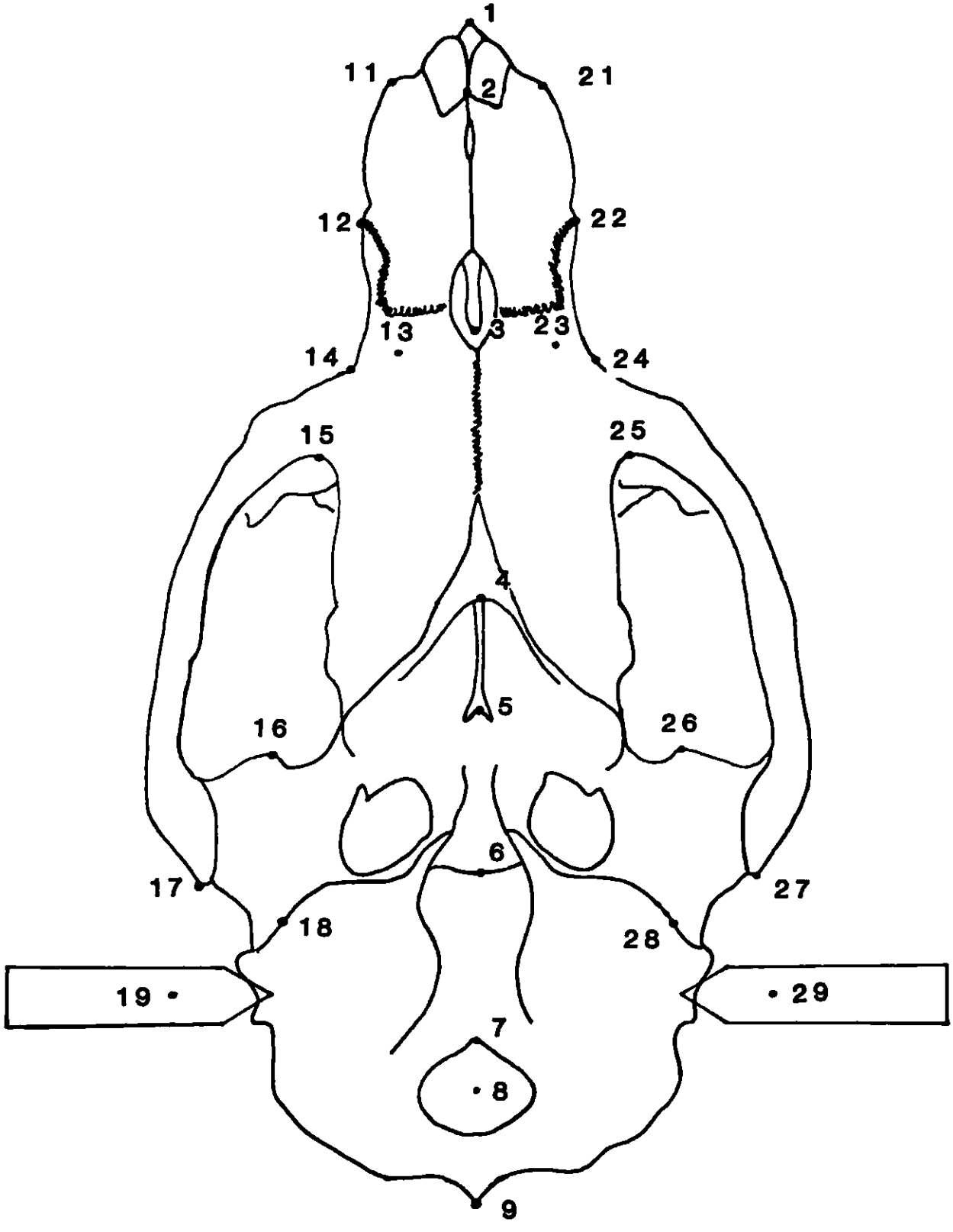
1. Tip of the anterior nasal spine (1)
2. Contact point between the medial surfaces of the incisors (2)
3. Most posterior point of the part of th nasal septum being on the palatine foramen (3)
4. Most anterior point of the choana (4)
5. Most posterior point of the median longitudinal ridge of the body of the presphenoid (5)
6. Midpoint of the spheno-occipital synchondrosis (6)
7. Most anterior point of foramen magnum
8. Center of foramen magnum (8)
9. Most posterior point of the external occipital crest (9)
10. * Most anterior and lateral point of the nasal bone (11,21)
11. * Most lateral point of the incisivo-maxillary suture (12,22)
12. * Most anterior point of the lst molar (13,23)

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* Bilateral points

FIGURE 3

The points on the acetate paper


1
11
2
21
12
22
13
3
23
14
24
15
25
4
5
16
26
6
17
27
18
28
19
29
7
8
9

13.* Most anterior point of the anterior surface of the zygomatic process of the maxilla (14,24)

14.* Most anterior point of the posterior surface of the zygomatic process of the maxilla (15,25)

15.* Most anterior point of the mandibular fossa (16,26)

16.* Most posterior point of the mandibular fossa (17,27)

17.* Most lateral point of the tymapanic bulla (18,28)

18.* Midpoint of the ear-rod (19,29)

A combination of direct measurements and X-Y coordinates were used. The midline which served as the ordinate was established in five different ways, (as defined by points 5-9, 5-8, 4-7, 1-4, perpendicular from 1 to 19-29) and the line passing through the midpoints of the ear-rods formed the abcissa.

A total of 112 linear measurements were employed to characterize the bilateral size, shape and position of the craniofacial components. The measurements employed are listed in Table II and depicted in figures 4 to 10. This analysis was adapted from earlier works by Behrents (1975) and Palmer (1978).

All linear measurements were executed geometrically by a computer program from X-Y coordinates of anatomic landmarks previously located and identified by pin holes placed in acetate paper.

Digitization of cephalometric landmarks was done in the following manner. The acetate paper containing the punched landmarks was secured to digitizing pad (summagraphics). A movable stylus was then placed over each individual landmark and its X-Y coordinates taken.

TABLE II

CEPHALOMETRIC ANALYSIS

Variable	Measure	Landmarks employed
V_1	Right lateral nasal bone development	11 - ordinate (5-9)
V_2	Left lateral nasal bone development	21 - ordinate (5-9)
V_3	Right lateral development of incisivo-maxillary suture	12 - ordinate (5-9)
V_4	Left lateral development of incisivo-maxillary suture	22 - ordinate (5-9)
V_5	Right lateral advancement of 1st molar	13 - ordinate (5-9)
V_6	Left lateral advancement of 1st molar	23 - ordinate (5-9)
V_7	Right lateral development of zygomatic process of maxilla [anterior surface]	14 - ordinate (5-9)
V_8	Left lateral development of zygomatic process of maxilla [anterior surface]	24 - ordinate (5-9)
V_9	Right lateral development of zygomatic process of maxilla [posterior surface]	15 - ordinate (5-9)
V_{10}	Left lateral development of zygomatic process of maxilla [posterior surface]	25 - ordinate (5-9)
V_{11}	Right lateral development of the anterior part of the mandibular fossa	16 - ordinate (5-9)
V_{12}	Left Lateral development of the anterior part of the mandibular fossa	26 - ordinate (5-9)
V_{13}	Right lateral development of the posterior part of the mandibular fossa	17 - ordinate (5-9)
V_{14}	Left lateral development of the posterior part of the mandibular fossa	27 - ordinate (5-9)
V_{15}	Right lateral development of tympanic bulla	18 - ordinate (5-9)
V_{16}	Left lateral development of tympanic bulla	28 - ordinate (5-9)

TABLE II (Cont)

CEPHALOMETRIC ANALYSIS

Variable	Measure	Landmarks employed
V_{17}	Right lateral nasal bone development	11 - ordinate (5-8)
V_{18}	Left lateral nasal bone development	21 - ordinate (5-8)
V_{19}	Right lateral development of incisivo-maxillary suture	12 - ordinate (5-8)
V_{20}	Left lateral development of incisivo-maxillary suture	22 - ordinate (5-8)
V_{21}	Right lateral advancement of 1st molar	13 - ordinate (5-8)
V_{22}	Left lateral advancement of 1st molar	23 - ordinate (5-8)
V_{23}	Right lateral development of zygomatic process of maxilla [anterior surface]	14 - ordinate (5-8)
V_{24}	Left lateral development of zygomatic process of maxilla [anterior surface]	24 - ordinate (5-8)
V_{25}	Right lateral development of zygomatic process of maxilla [posterior surface]	15 - ordinate (5-8)
V_{26}	Left lateral development of zygomatic process of maxilla [posterior surface]	25 - ordinate (5-8)
V_{27}	Right lateral development of the anterior part of the mandibular fossa	16 - ordinate (5-8)
V_{28}	Left Lateral development of the anterior part of the mandibular fossa	26 - ordinate (5-8)
V_{29}	Right lateral development of the posterior part of the mandibular fossa	17 - ordinate (5-8)
V_{30}	Left lateral development of the posterior part of the mandibular fossa	27 - ordinate (5-8)
V_{31}	Right lateral development of tympanic bulla	18 - ordinate (5-8)
V_{32}	Left lateral development of tympanic bulla	28 - ordinate (5-8)

TABLE II (Cont)

CEPHALOMETRIC ANALYSIS

Variable	Measure	Landmarks employed
V_{33}	Right lateral nasal bone development	11 - ordinate (4-7)
V_{34}	Left lateral nasal bone development	21 - ordinate (4-7)
V_{35}	Right lateral development of incisivo-maxillary suture	12 - ordinate (4-7)
V_{36}	Left lateral development of incisivo-maxillary suture	22 - ordinate (4-7)
V_{37}	Right lateral advancement of 1st molar	13 - ordinate (4-7)
V_{38}	Left lateral advancement of 1st molar	23 - ordinate (4-7)
V_{39}	Right lateral development of zygomatic process of maxilla [anterior surface]	14 - ordinate (4-7)
V_{40}	Left lateral development of zygomatic process of maxilla [anterior surface]	24 - ordinate (4-7)
V_{41}	Right lateral development of zygomatic process of maxilla [posterior surface]	15 - ordinate (4-7)
V_{42}	Left lateral development of zygomatic process of maxilla [posterior surface]	25 - ordinate (4-7)
V_{43}	Right lateral development of the anterior part of the mandibular fossa	16 - ordinate (4-7)
V_{44}	Left Lateral development of the anterior part of the mandibular fossa	26 - ordinate (4-7)
V_{45}	Right lateral development of the posterior part of the mandibular fossa	17 - ordinate (4-7)
V_{46}	Left lateral development of the posterior part of the mandibular fossa	27 - ordinate (4-7)
V_{47}	Right lateral development of tympanic bulla	18 - ordinate (4-7)
V_{48}	Left lateral development of tympanic bulla	28 - ordinate (4-7)

TABLE II (Cont)

CEPHALOMETRIC ANALYSIS

Variable	Measure	Landmarks employed
V_{49}	Right lateral nasal bone development	11 - ordinate (1-4)
V_{50}	Left lateral nasal bone development	21 - ordinate (1-4)
V_{51}	Right lateral development of incisivo-maxillary suture	12 - ordinate (1-4)
V_{52}	Left lateral development of incisivo-maxillary suture	22 - ordinate (1-4)
V_{53}	Right lateral advancement of 1st molar	13 - ordinate (1-4)
V_{54}	Left lateral advancement of 1st molar	23 - ordinate (1-4)
V_{55}	Right lateral development of zygomatic process of maxilla [anterior	14 - ordinate (1-4)
V_{56}	Left lateral development of zygomatic process of maxilla surface]	24 - ordinate (1-4)
V_{57}	Right lateral development of zygomatic process of maxilla [posterior	15 - ordinate (1-4)
V_{58}	Left lateral development of zygomatic process of maxilla surface]	25 - ordinate (1-4)
V_{59}	Right lateral development of the anterior part of the mandibular fossa	16 - ordinate (1-4)
V_{60}	Left Lateral development of the anterior part of the mandibular fossa	26 - ordinate (1-4)
V_{61}	Right lateral development of the posterior part of the mandibular fossa	17 - ordinate (1-4)
V_{62}	Left lateral development of the posterior part of the mandibular fossa	27 - ordinate (1-4)
V_{63}	Right lateral development of tympanic bulla	18 - ordinate (1-4)
V_{64}	Left lateral development of tympanic bulla	28 - ordinate (1-4)

TABLE II (Cont)

CEPHALOMETRIC ANALYSIS

Variable	Measure	Landmarks employed
V_{65}	Right anterior nasal bone development	11 - abscissa (19-29)
V_{66}	Left anterior nasal bone development	21 - abscissa (19-29)
V_{67}	Right anterior development of incisivo-maxillary suture	12 - abscissa (19-29)
V_{68}	Left anterior development of incisivo-maxillary suture	22 - abscissa (19-29)
V_{69}	Right anterior advancement of 1st molar	13 - abscissa (19-29)
V_{70}	Left anterior advancement of 1st molar	23 - abscissa (19-29)
V_{71}	Right anterior development of zygomatic process of maxilla [anterior	14 - abscissa (19-29)
V_{72}	Left anterior development of zygomatic process of maxilla surface]	24 - abscissa (19-29)
V_{73}	Right anterior development of zygomatic process of maxilla [posterior	15 - abscissa (19-29)
V_{74}	Left anterior development of zygomatic process of maxilla surface]	25 - abscissa (19-29)
V_{75}	Right anterior development of the anterior part of the mandibular fossa	16 - abscissa (19-29)
V_{76}	Left anterior development of the anterior part of the mandibular fossa	26 - abscissa (19-29)
V_{77}	Right anterior development of the posterior part of the mandibular fossa	17 - abscissa (19-29)
V_{78}	Left anterior development of the posterior part of the mandibular fossa	27 - abscissa (19-29)
V_{79}	Right anterior development of tympanic bulla	18 - abscissa (19-29)
V_{80}	Left anterior development of tympanic bulla	28 - abscissa (19-29)

TABLE II (Cont)

CEPHALOMETRIC ANALYSIS

Variable	Measure	Landmarks employed
V_{81}	Right lateral nasal bone development	11 - ordinate*
V_{82}	Left lateral nasal bone development	21 - ordinate*
V_{83}	Right lateral development of incisivo-maxillary suture	12 - ordinate*
V_{84}	Left lateral development of incisivo-maxillary suture	22 - ordinate*
V_{85}	Right lateral advancement of 1st molar	13 - ordinate*
V_{86}	Left lateral advancement of 1st molar	23 - ordinate*
V_{87}	Right lateral development of zygomatic process of maxilla [anterior	14 - ordinate*
V_{88}	Left lateral development of zygomatic process of maxilla surface]	24 - ordinate*
V_{89}	Right lateral development of zygomatic process of maxilla [posterior	15 - ordinate*
V_{90}	Left lateral development of zygomatic process of maxilla surface]	25 - ordinate*
V_{91}	Right lateral development of the anterior part of the mandibular fossa	16 - ordinate*
V_{92}	Left Lateral development of the anterior part of the mandibular fossa	26 - ordinate*
V_{93}	Right lateral development of the posterior part of the mandibular fossa	17 - ordinate*
V_{94}	Left lateral development of the posterior part of the mandibular fossa	27 - ordinate*
V_{95}	Right lateral development of tympanic bulla	18 - ordinate*
V_{96}	Left lateral development of tympanic bulla	28 - ordinate*

* Perpendicular from point 1 to 19-29.

TABLE II (Cont)

CEPHALOMETRIC ANALYSIS

Variable	Measure	Landmarks employed
V_{97}	Right squamosal advancement	9-17
V_{98}	Left squamosal advancement	9-27
V_{99}	Right anteroposterior squamosal growth	17-16
V_{100}	Left anteroposterior squamosal growth	27-26
V_{101}	Right zygomatic arch length	16-15
V_{102}	Left zygomatic arch length	26-25
V_{103}	Right 1st molar advancement	15-13
V_{104}	Left 1st molar advancement	25-23
V_{105}	Right nasal bone advancement	13-11
V_{106}	Left nasal bone advancement	23-21
V_{107}	Right	11-1
V_{108}	Left	22-1
V_{109}	Right snout deviation	15-1
V_{110}	Left	25-1
V_{111}	Right	17-1
V_{112}	Left	27-1
V_{113}	enlargement error	10-20

FIGURE 4

Measurements from the ordinate defined by points 5 and 9

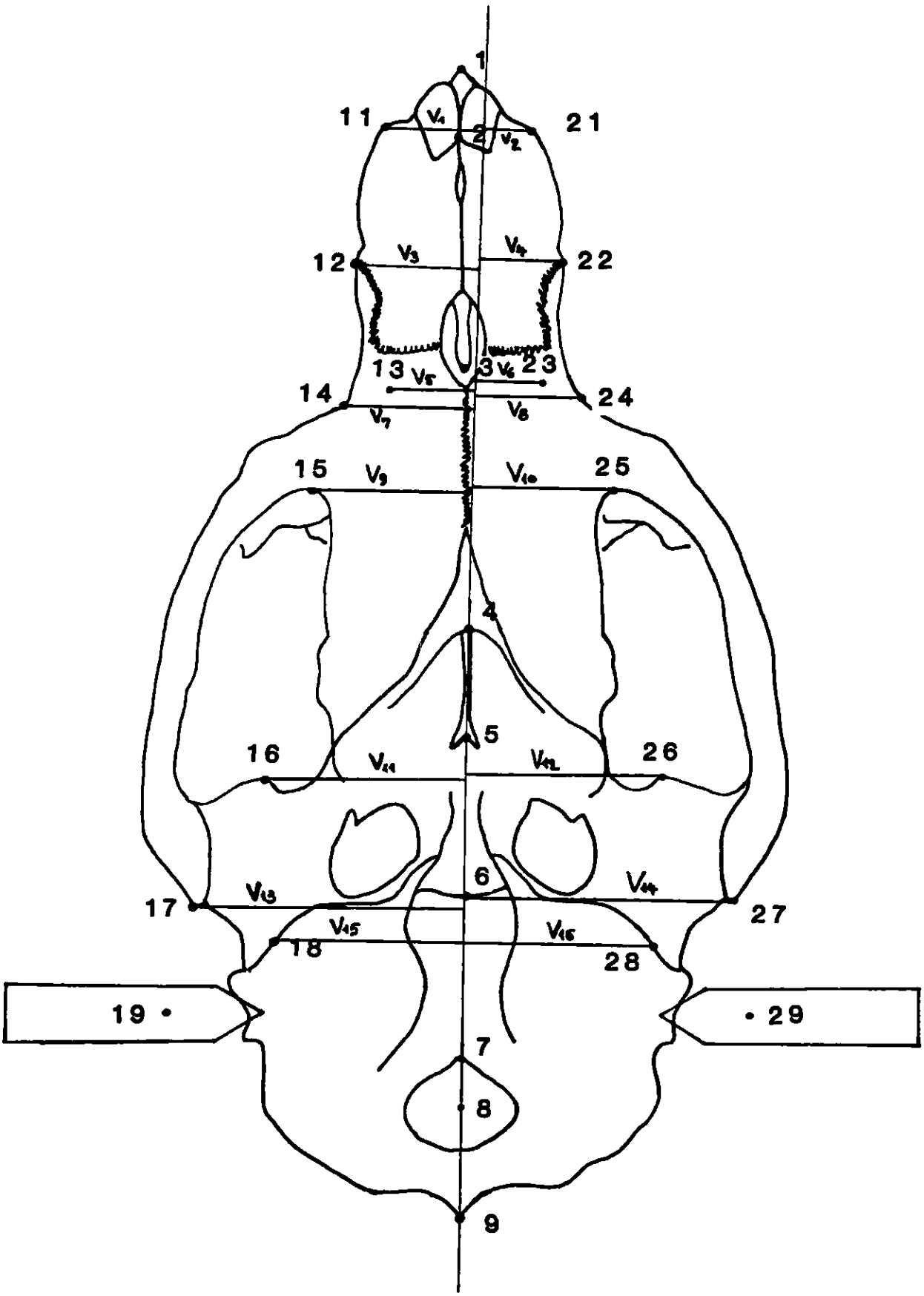

1
11
V1
2
V2
21
12
V3
V4
22
13
V5
3
V6
23
14
V7
V8
24
15
V9
V10
25
4
5
16
V11
V12
26
6
17
V13
V14
27
V15
V16
18
28
19
29
7
8
9

FIGURE 5

Measurements from the ordinate defined by points 5 and 8

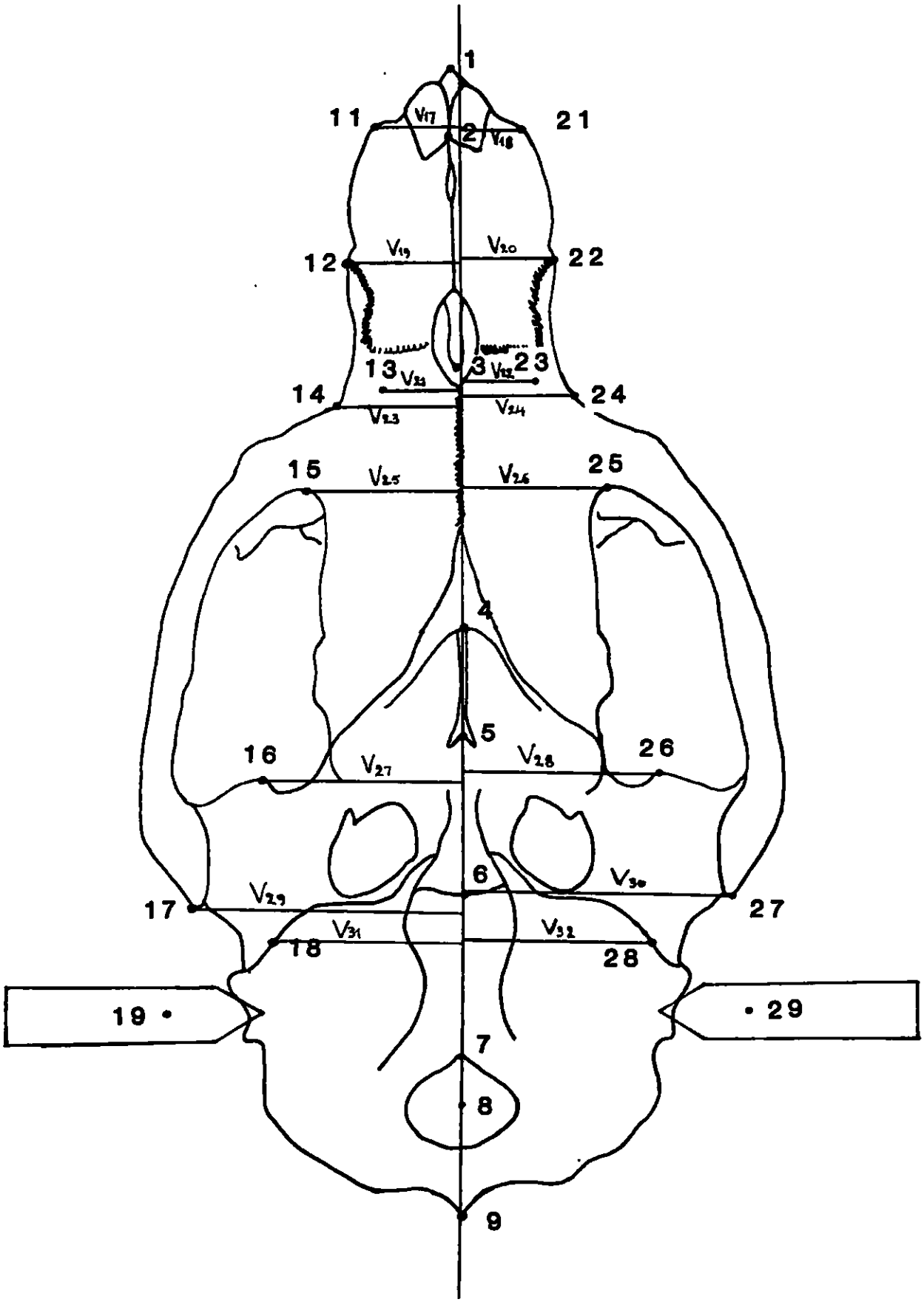

1
11
V17
2
V18
21
12
V19
V20
22
13
V21
3
V22
23
14
V23
V24
24
15
V25
V26
25
4
5
16
V27
V28
26
6
V30
17
V29
27
V31
V32
18
28
19
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9

FIGURE 6

Measurements from the ordinate defined by points 4 and 7

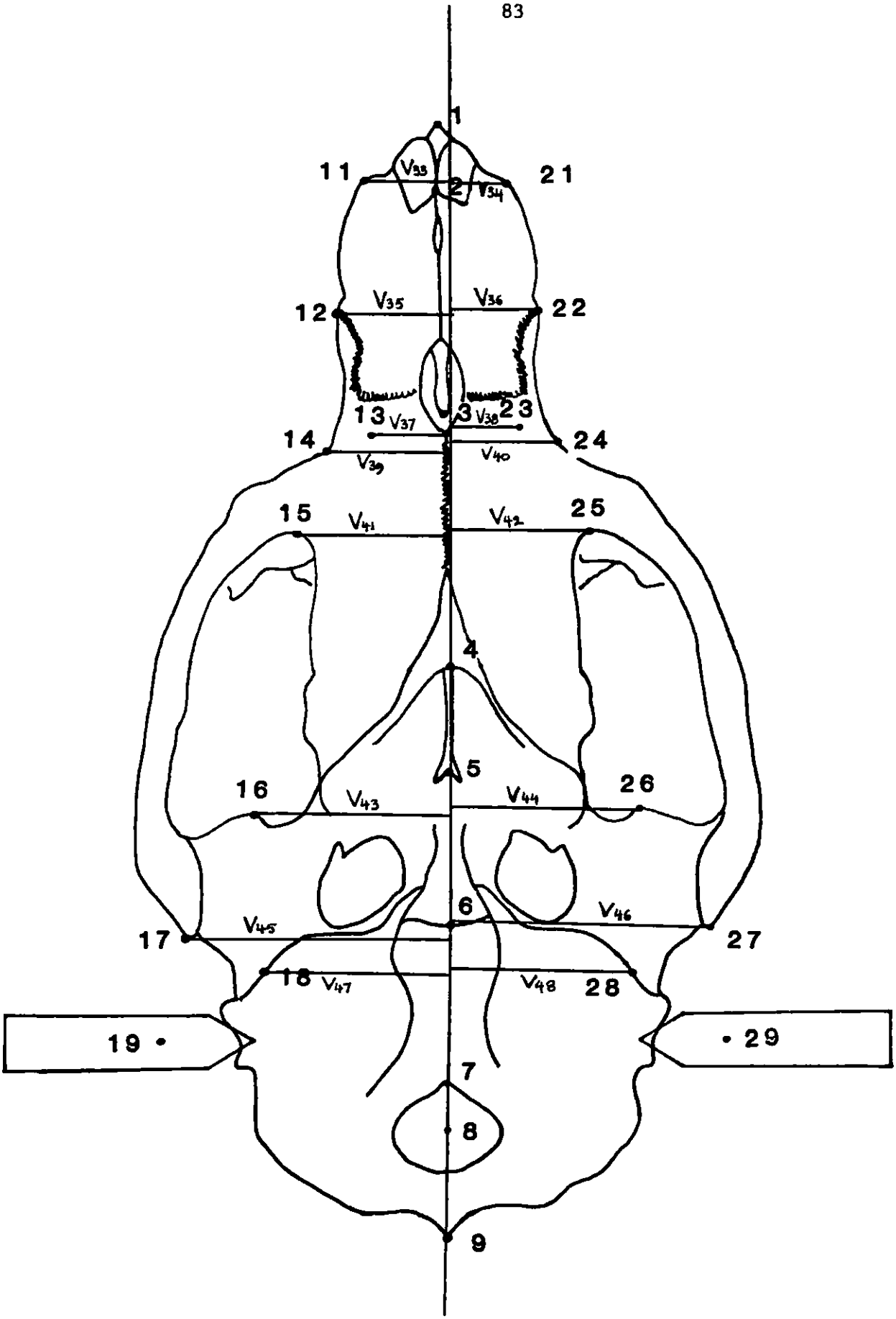

1
11
V33
2
V34
21
12
V35
V36
22
13
V37
3
V38
23
14
V39
V40
24
15
V41
V42
25
4
5
16
V43
V44
26
6
V45
V46
17
27
18
V47
V48
28
19
29
7
8
9

FIGURE 7

Measurements from the ordinate defined by points 1 and 4

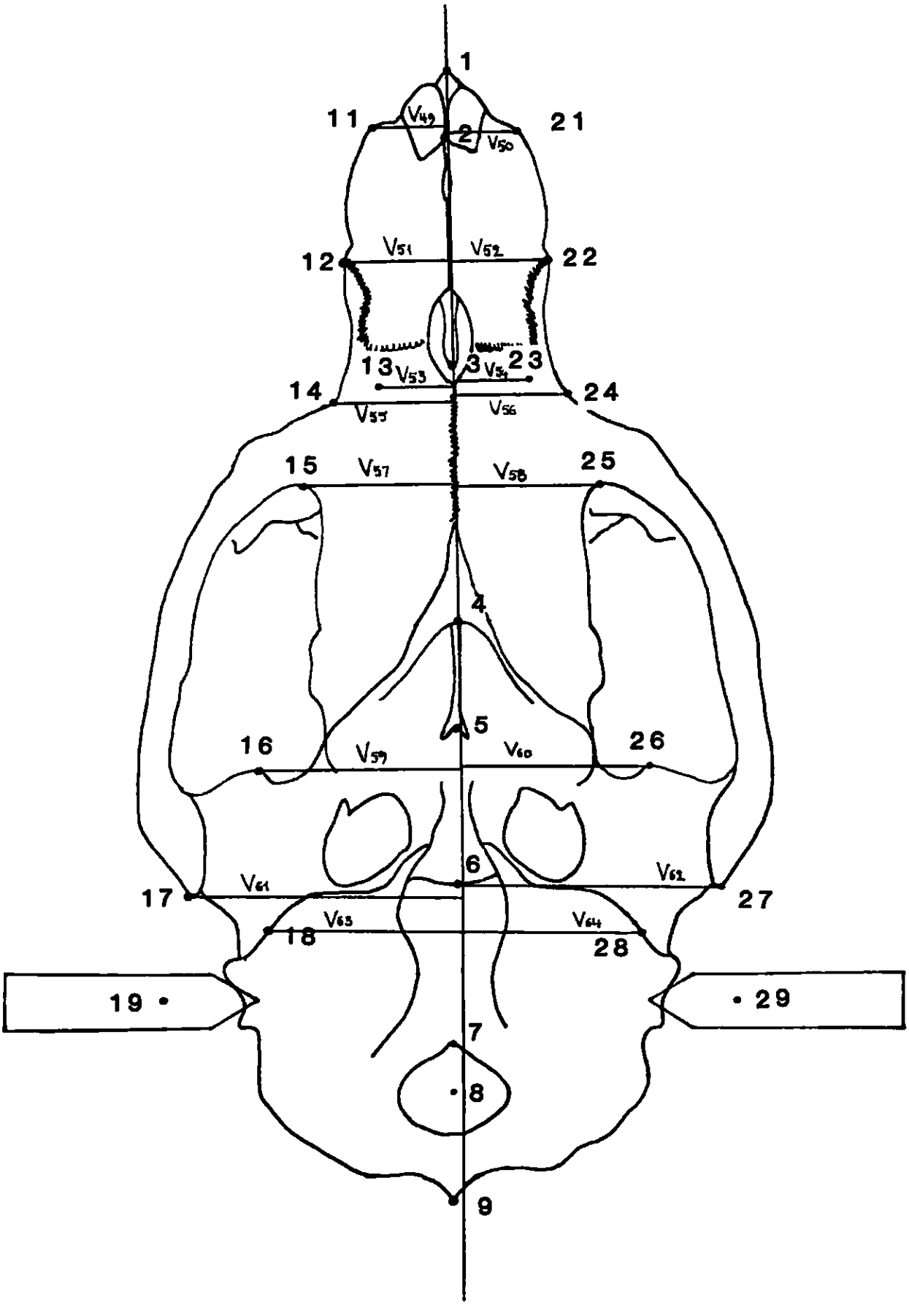

1
11
V49
2
V50
21
12
V51
V52
22
13
V53
3
V54
23
14
V55
V56
24
15
V57
V58
25
4
16
V59
V60
26
5
6
17
V61
V62
27
18
V63
V64
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19
29
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8
9

FIGURE 8

Measurements from the abscissa defined by points 19-29

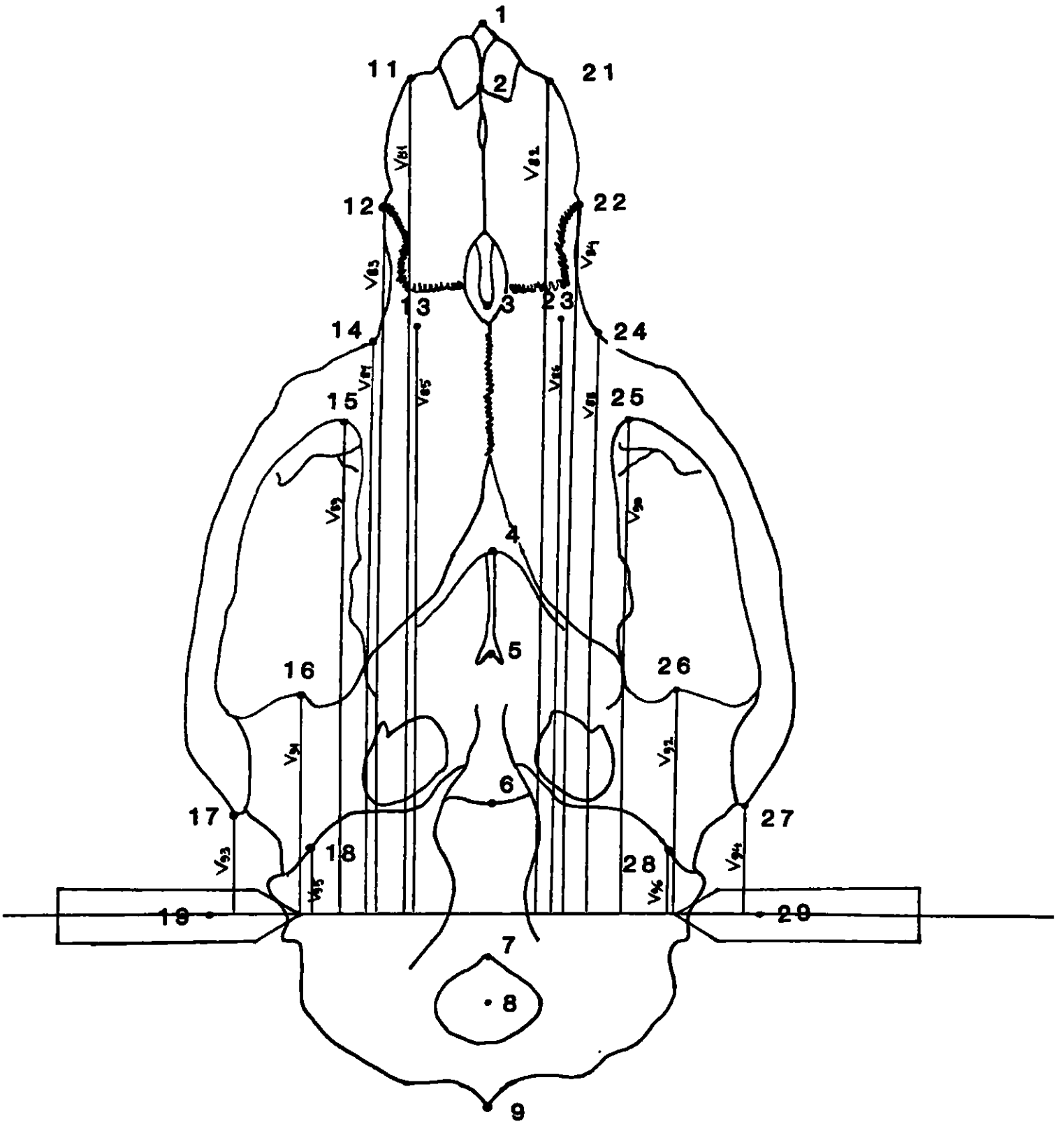

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FIGURE 9

Measurements from the ordinate defined as the perpendicular from the point 1 to the line 19-29.

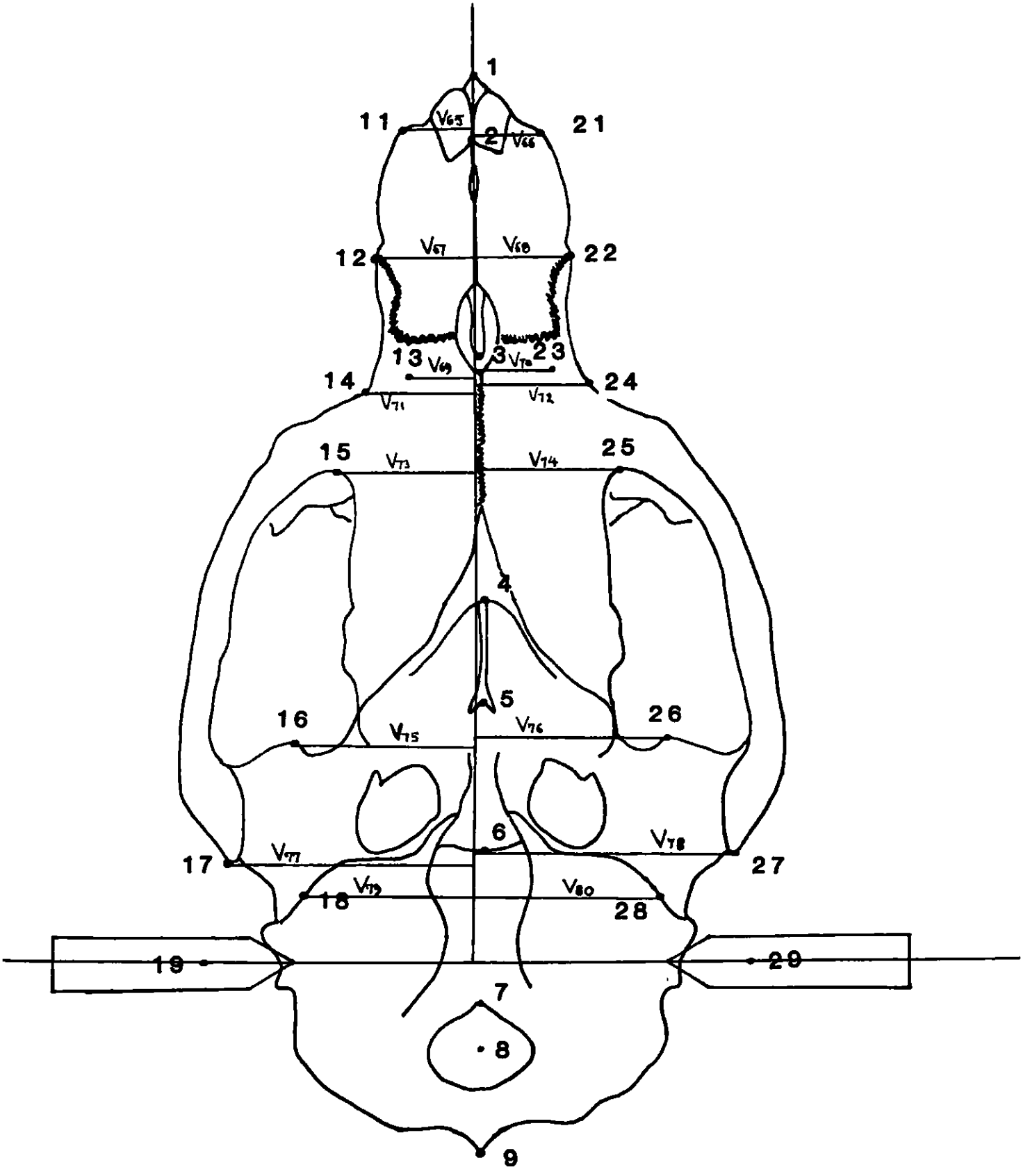
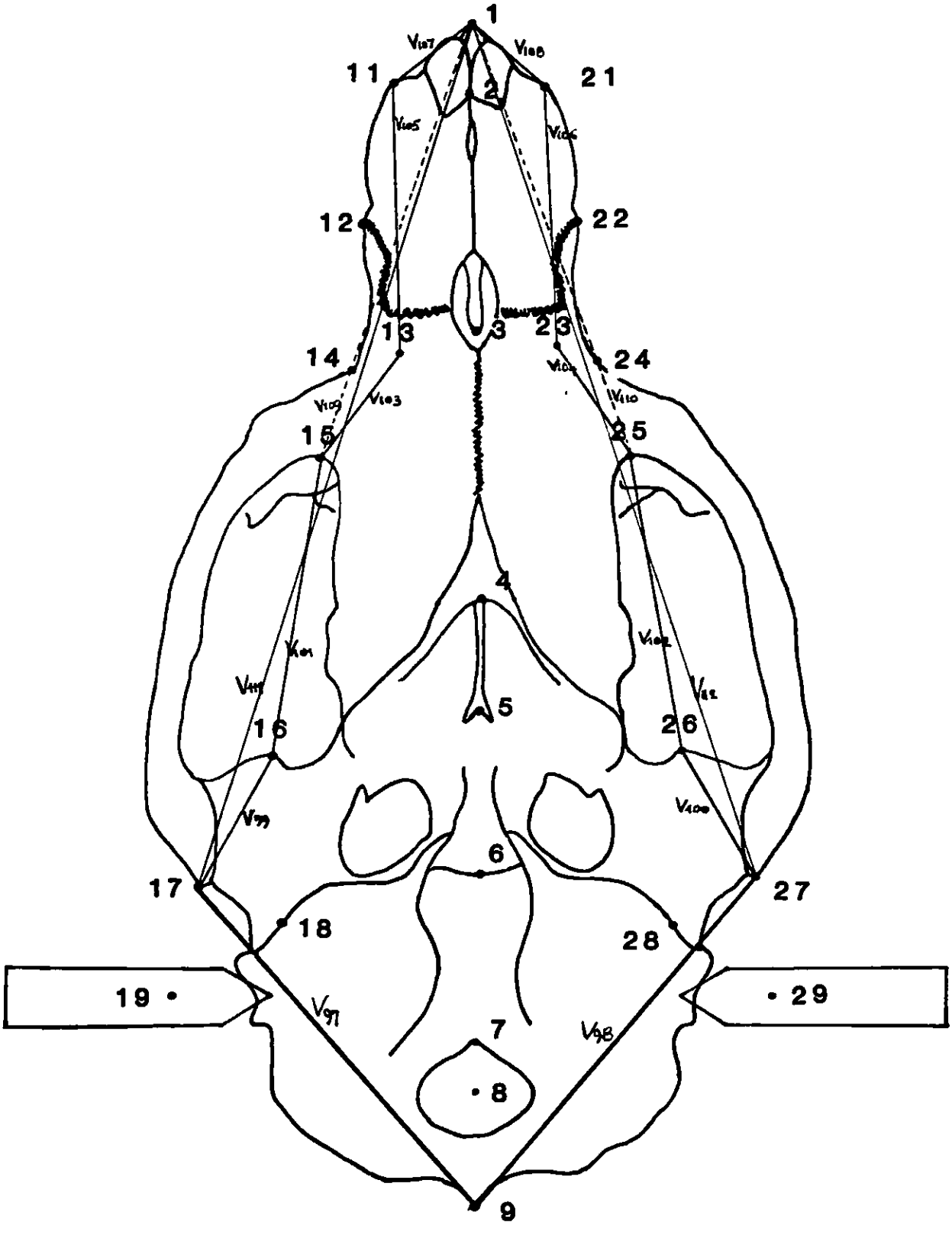

1
11
V65
2
V66
21
12
V67
V68
22
13
V69
3
V70
23
14
V71
V72
24
15
V73
V74
25
4
5
16
V75
V76
26
6
V78
17
V77
27
V79
V80
18
28
19
20
7
8
9

FIGURE 10

Anatomical dimensions


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Storage of information and execution of calculations was accomplished via normal methods, and utilized calculation programs developed previously (Behrents, 1984). Data reduction was accomplished via SPSS (statistical Program for the Statistical Sciences).

Histologic Analysis

Using various magnifications the histological preparations were examined for any differences between left and right sides regarding the types and the distribution of the epithelium, the type and the distribution of glands, the distribution of goblet cells, the size and the shape of the nasolacrimal duct and the size and shape of turbinates.

In order to insure unbiased evaluations, two other investigators[1], without any information regarding the identification of the specimens, examined and evaluated the slides with reference only to the number appearing on the tape.

Ground section examination

Using an ultraviolet light microscope the slides were examined for any differences between left and right sides regarding bone deposition pattern on the nasal walls, nasal septum, turbinates and the sutures. Two other investigators[2], without any information regarding the identification of the specimens, examined and evaluated the slides with reference only to the number of the slide.

---

[1] Dr. Anna Doukoudakis and Dr. Mintz
[2] Dr. Donald Enlow and Dr. Robert Cederquist

RESULTS

The results of the present investigation will be described under the following headings:

1. Gross Evaluation
2. Histological Evaluation
3. Bone Remodeling
4. Cephalometric Evaluation

Gross Evaluation

Although slight craniofacial asymmetries such as snout, zygomatic arche, and nasal operation deviations were noticed during visual examinations, these conditions did not appear to be consistently related to the experimental procedures.

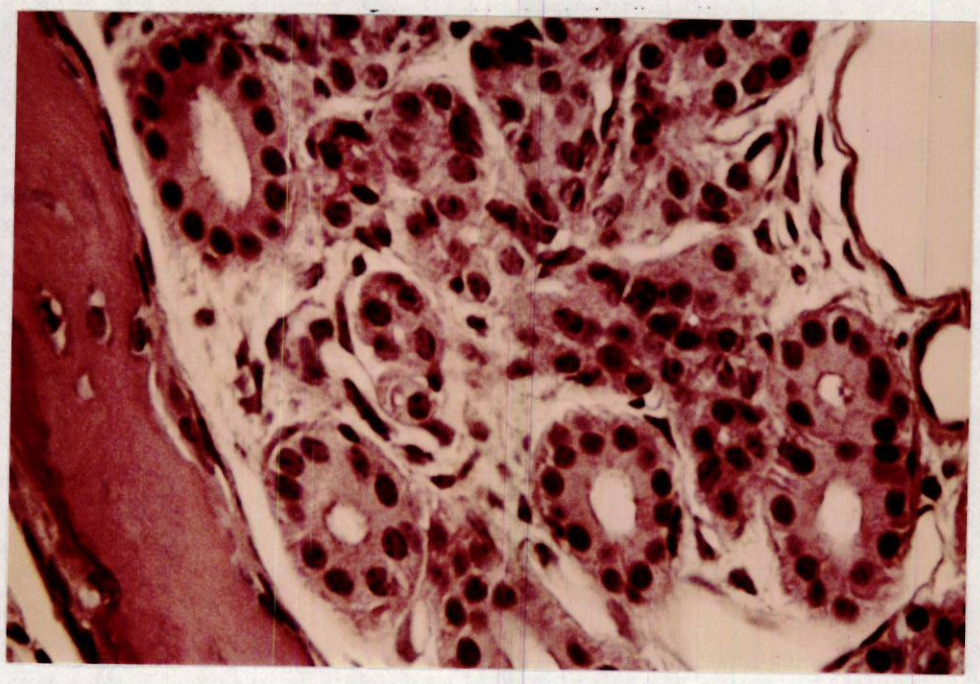
Histological Evaluation

Histological examination of the control animals revealed an almost perfectly symmetrical snout. At the most anterior part of the snout the epithelium was stratified squamous but more posteriorly became respiratory in type. The glands were mostly of the mucous type and were almost equally distributed on both sides (Plate V). The shape and size of both turbinates (nasoturbinal or concha-nasalis dorsalis and maxilboturbinal or concha-nasalis ventralis) were almost identical on both sides (Plate VI). The size and position of the nasolacrimal ducts were also similar (Plate VII). Goblet cells as described in the human nasal epithelium are not found in this species.

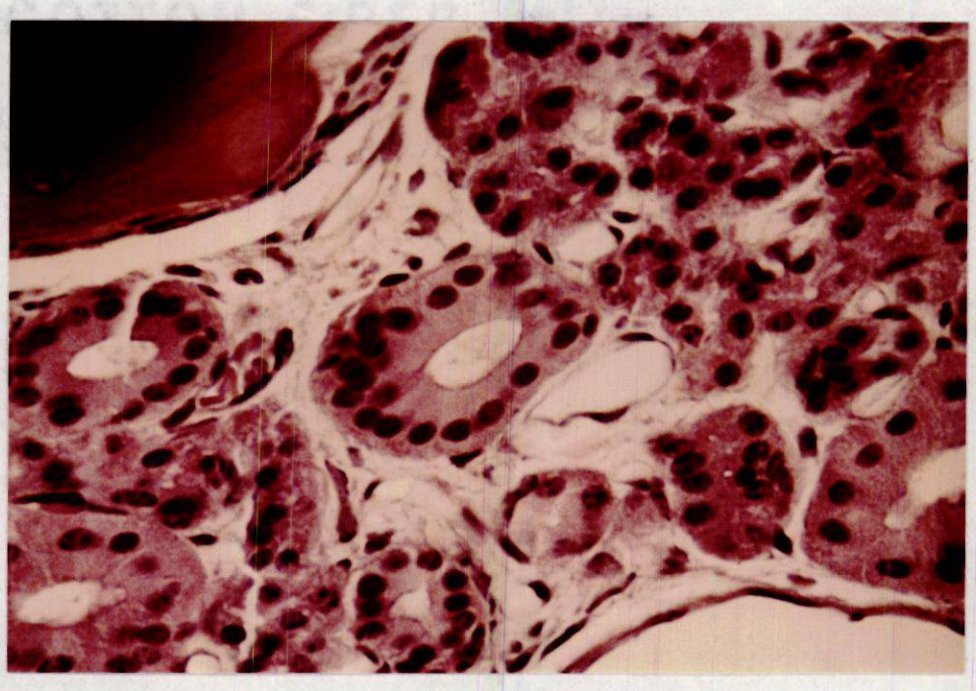
PLATE V

a) Distribution of glands on control animals.

b)Distribution of glands on experimental animals (unblocked side)



a



b

PLATE VI

The turbinates on control animals.

a) left side

b)right side



a



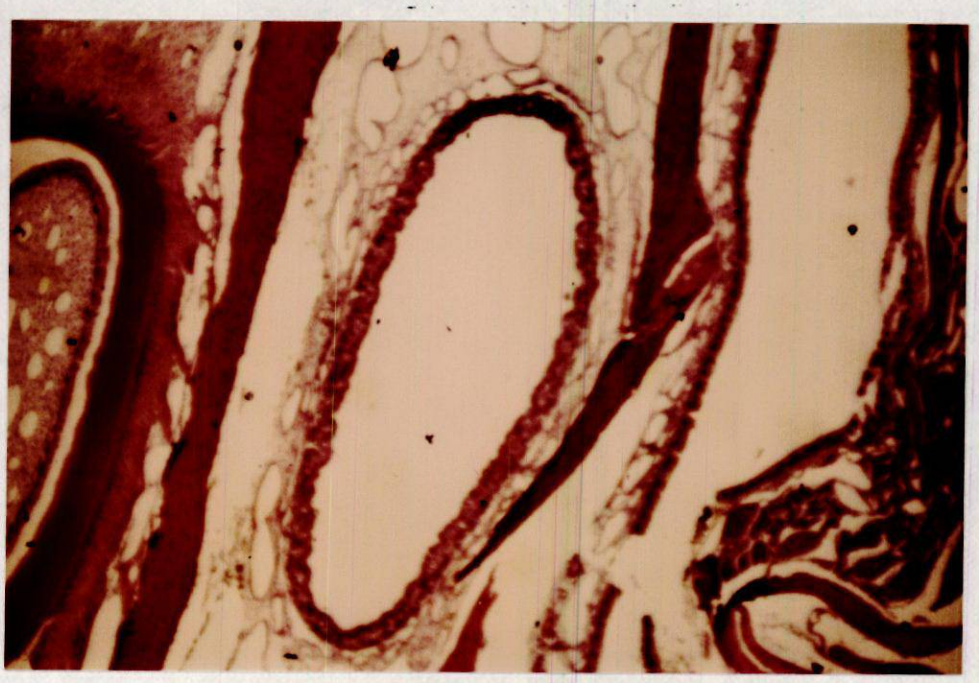
b

PLATE VII

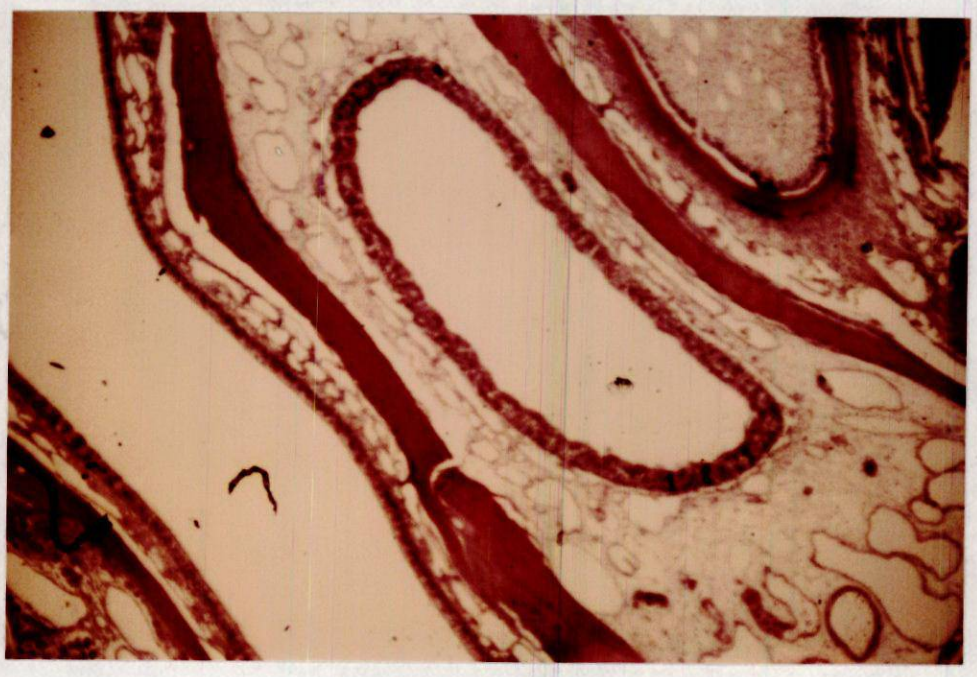
The nasolacrimal ducts on control animal.

a) left side

b) right side



a



b

In the experimental animals there were, however, some histologic differences in comparison to the controls but not in all of them. The epithelium in one of the experimental animals was almost similar with that of the control but in the two of them simple columnar epithelium covering hyperplastic papillae was observed in the unblocked side (Plate VIII). In the two of the three, on the unblocked side, the turbinates were larger in comparison with the unblocked side and different in shape (Plate IX). The size difference was due to hypertrophied epithelium and lamina propria. The nasolacrimal duct was larger on the unblocked side in two of the experimental animals, but there was no difference in the structure of its wall (Plate X).

Bone Remodeling

In sections taken in front of the palatal foramen of the control animals, the external surface of the nasal cavity is depository (Plate XI) and Fig. 11). The midnasal suture and the incisivo-nasal suture are almost equally active in bone deposition on both sides (Plate XII).

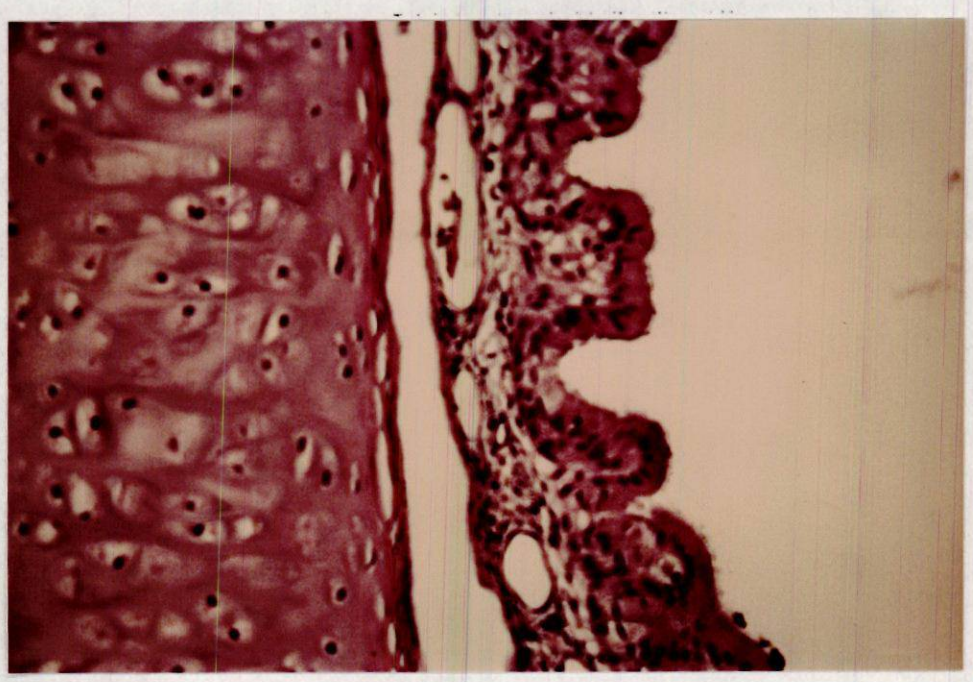
Bone deposition was also observed on the nasal floor just above the incisal roots (Plate XIII) and on both sides of nasal septum (Plate XIV).

In sections taken at the level of the palatal foramen, the expected "normal" remodeling pattern was observed (Enlow, 1982), with the exception of the nasal wall just above the incisors which was remodeling in an opposite way, that is, in an inward direction (Plate XV).

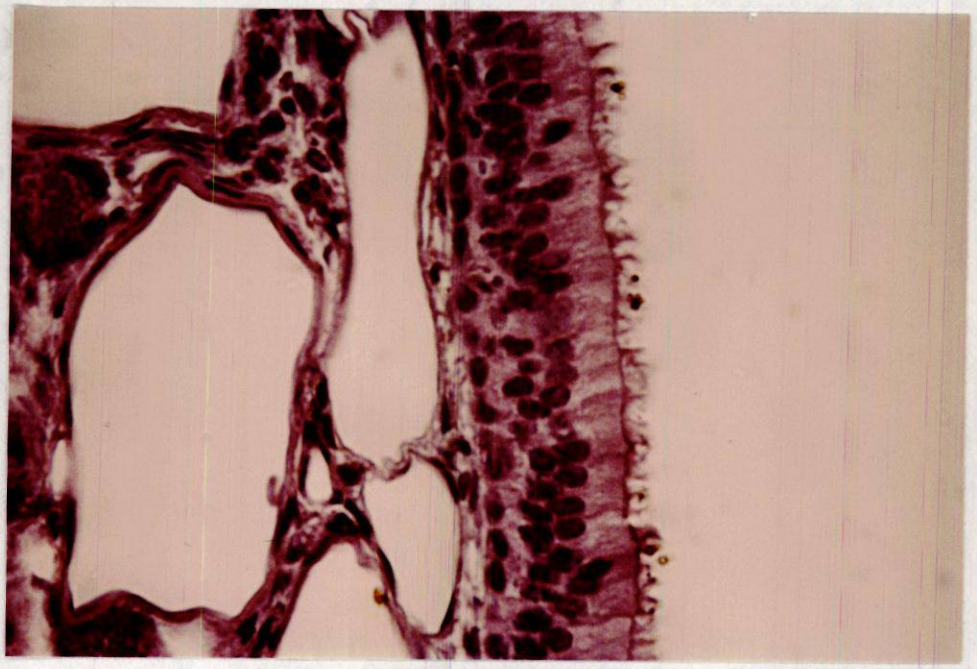
PLATE VIII

a) Simple columnar epithelium covering hyperplastic papillae on the unblocked side of experimental group.

b)Normal respiratory epithelium



a



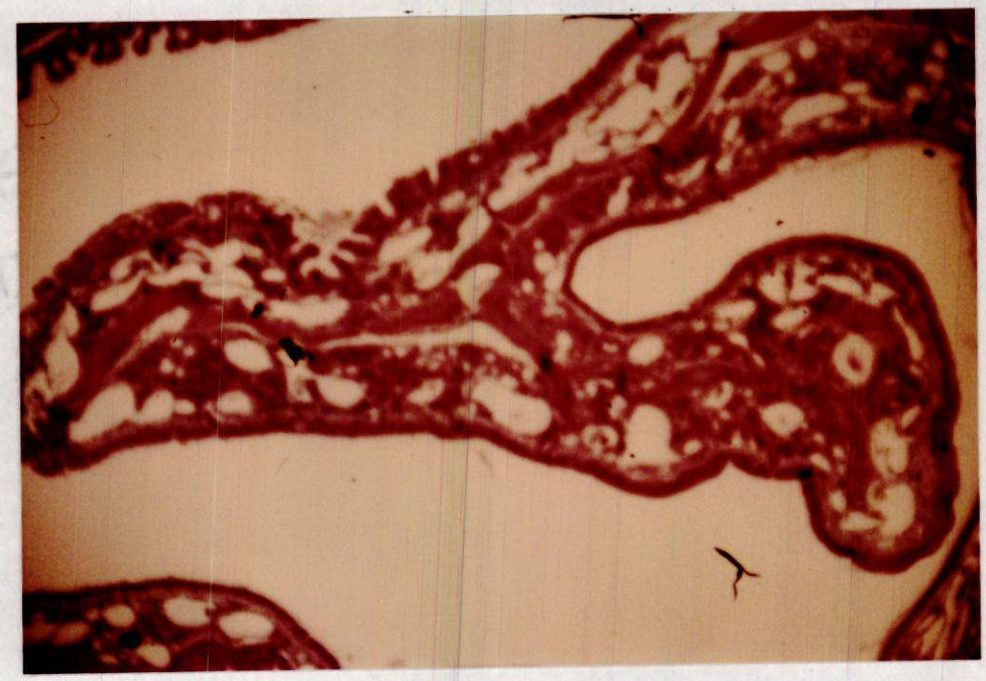
b

PLATE IX

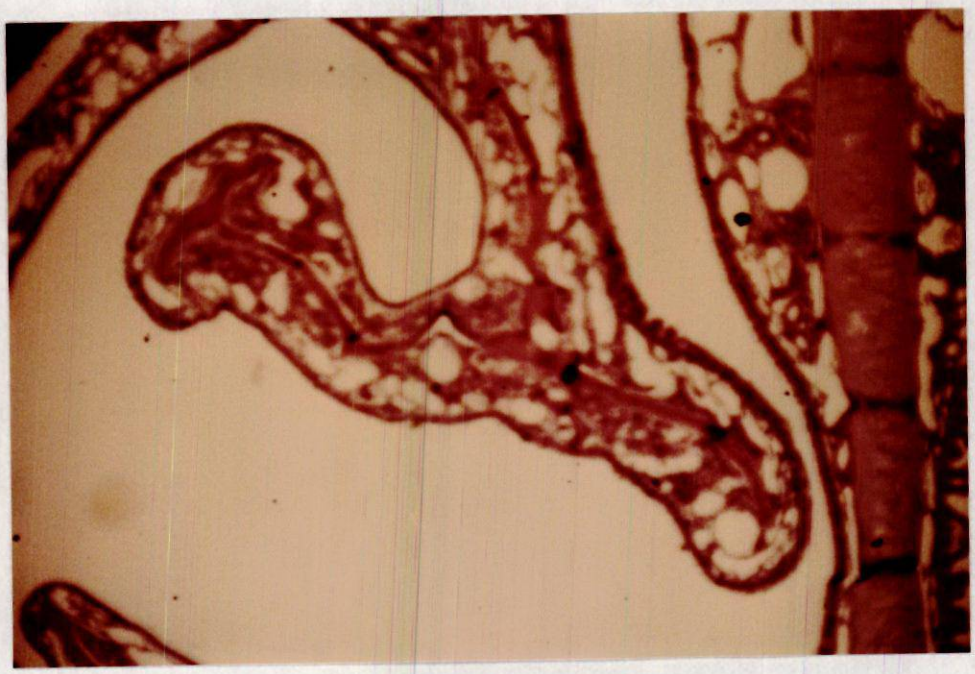
Turbinates on experimental animal.

a) blocked side

b) unblocked side



a



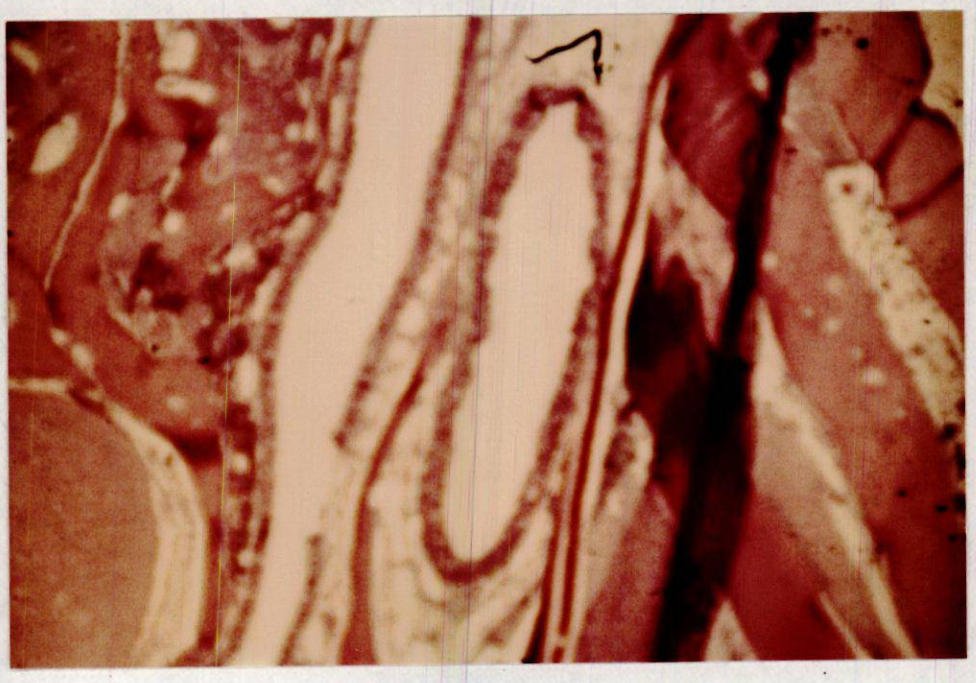
b

PLATE X

Nasolacrimal ducts on experimental animals

a) unblocked side

b) blocked side



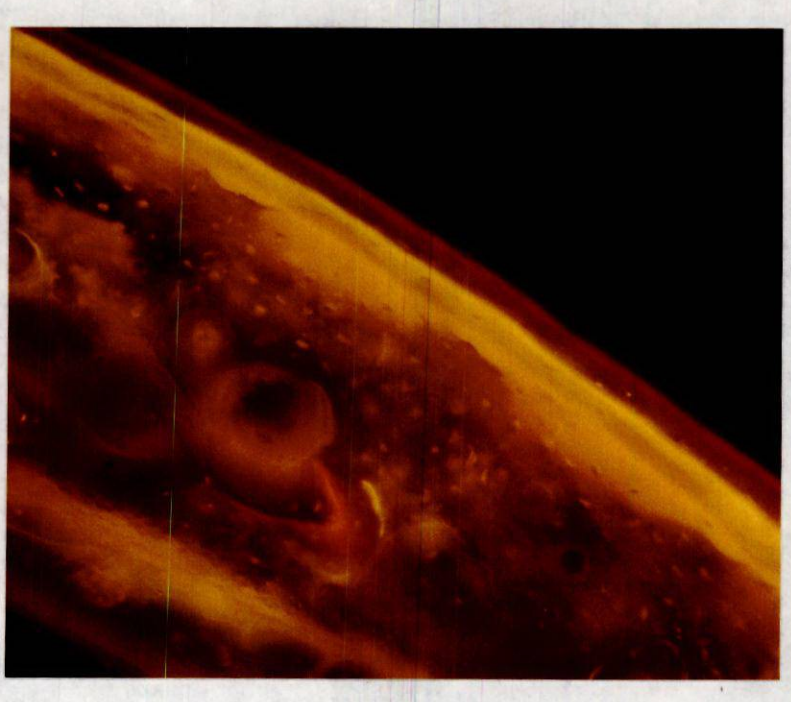
a

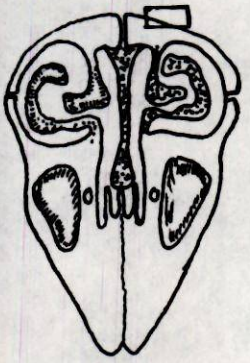


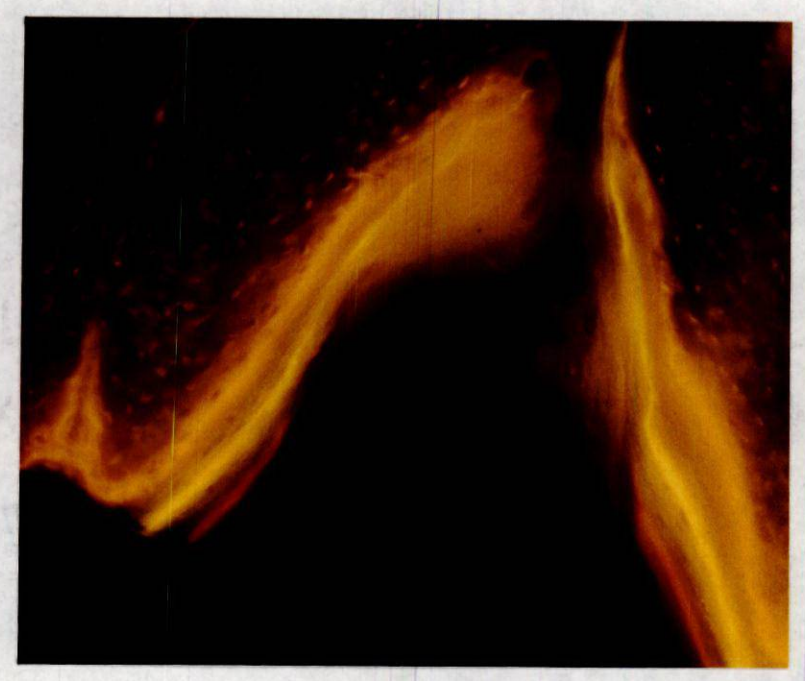
b

PLATE XI

The remodeling pattern of the anterior (in front of the palatal foramen) of the snout.







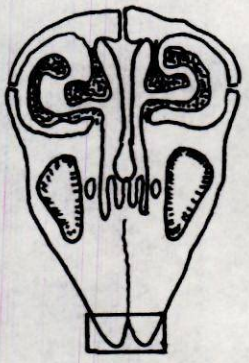


FIGURE 11

The remodeling pattern of the anterior part (in front of the palatal foramen) of the snout.

+ bone deposition

- bone resorption

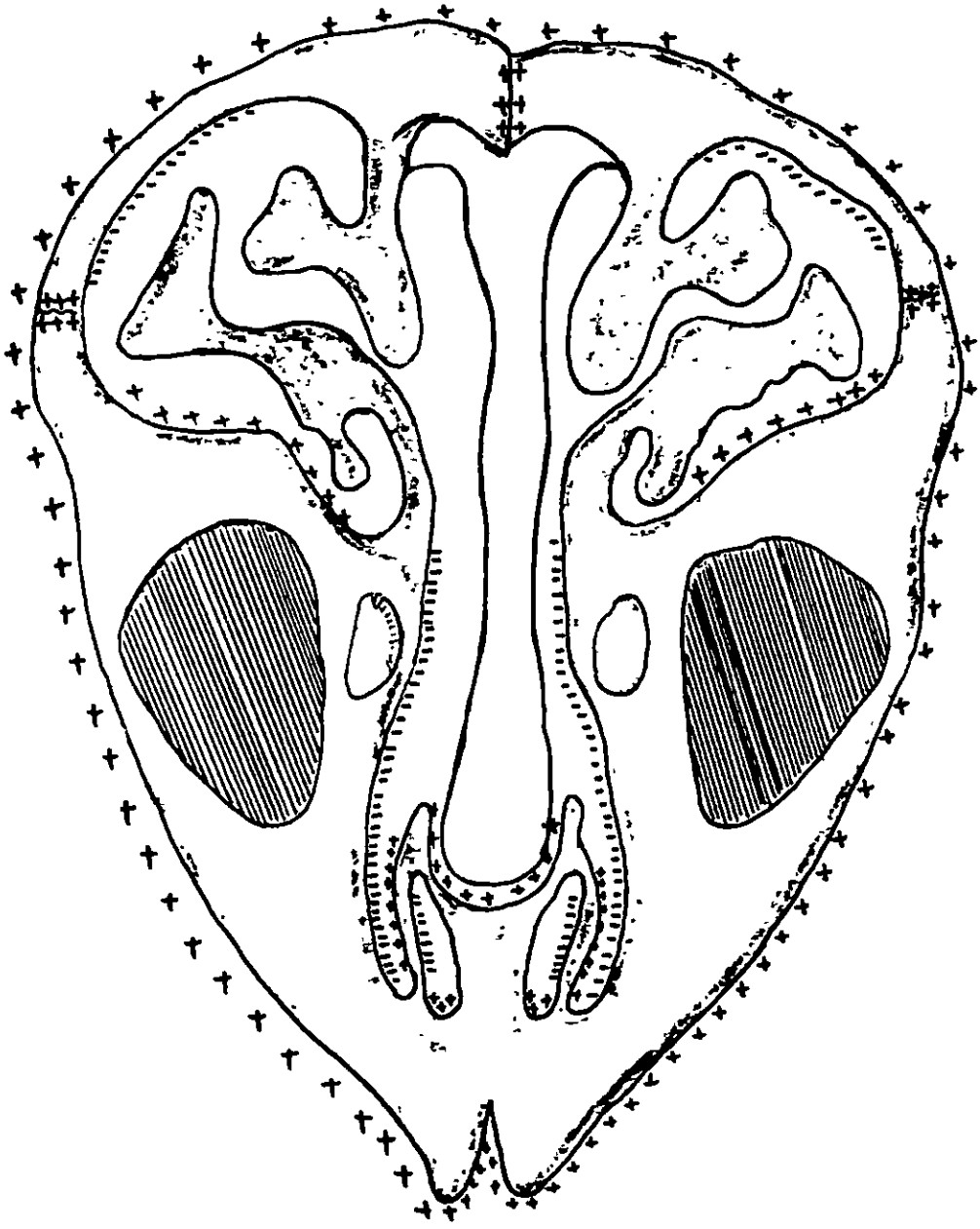
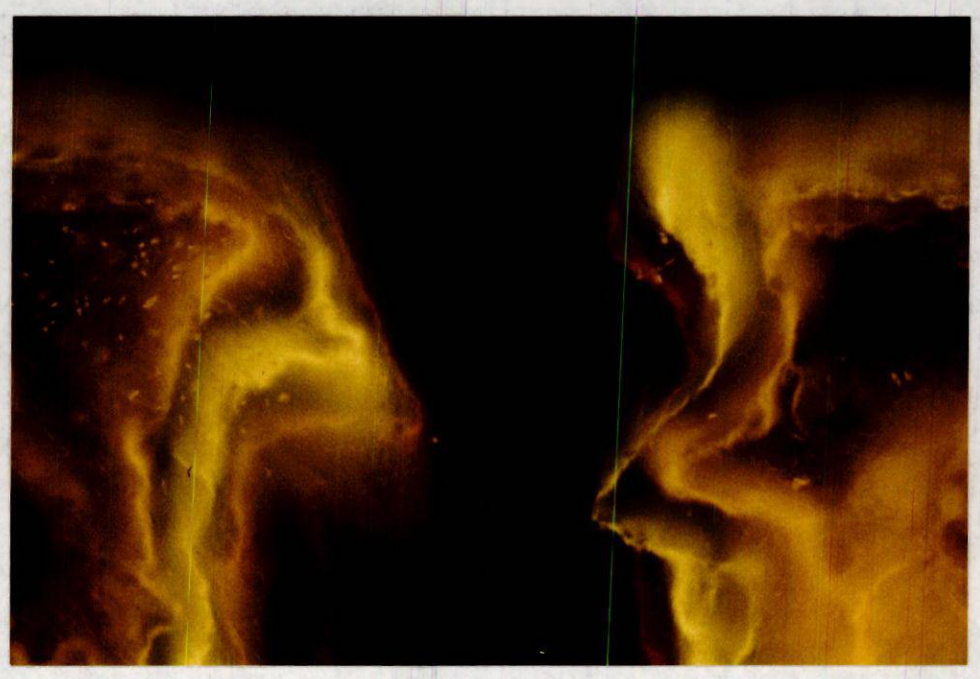


PLATE XII

The bone deposition pattern at the midnasal suture.



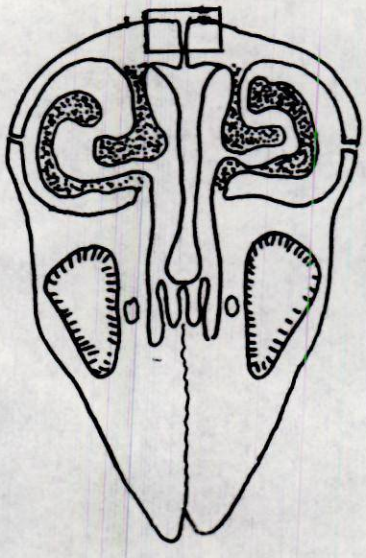


PLATE XIII

Remodeling pattern on the nasal floor, just above the incisal roots.



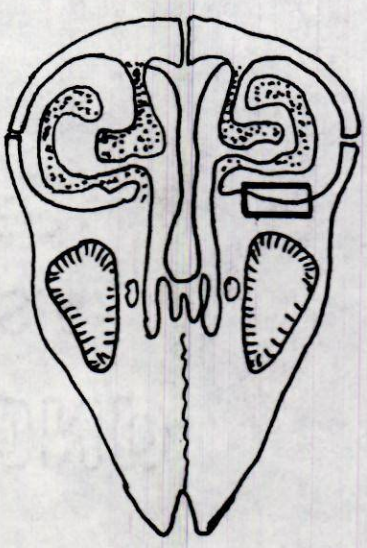


PLATE XIV

Remodeling pattern of the nasal septum.

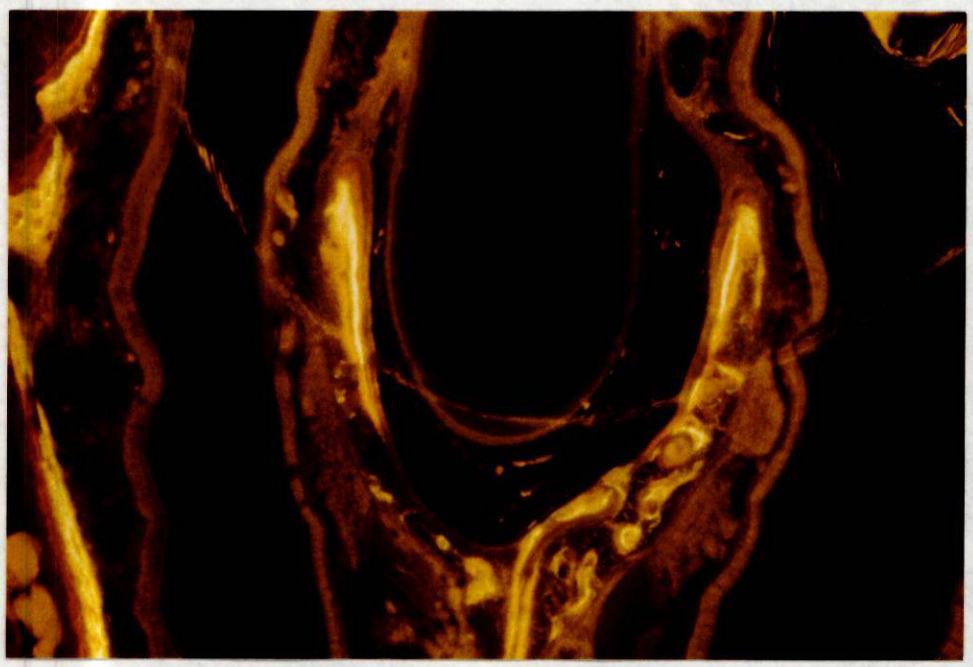
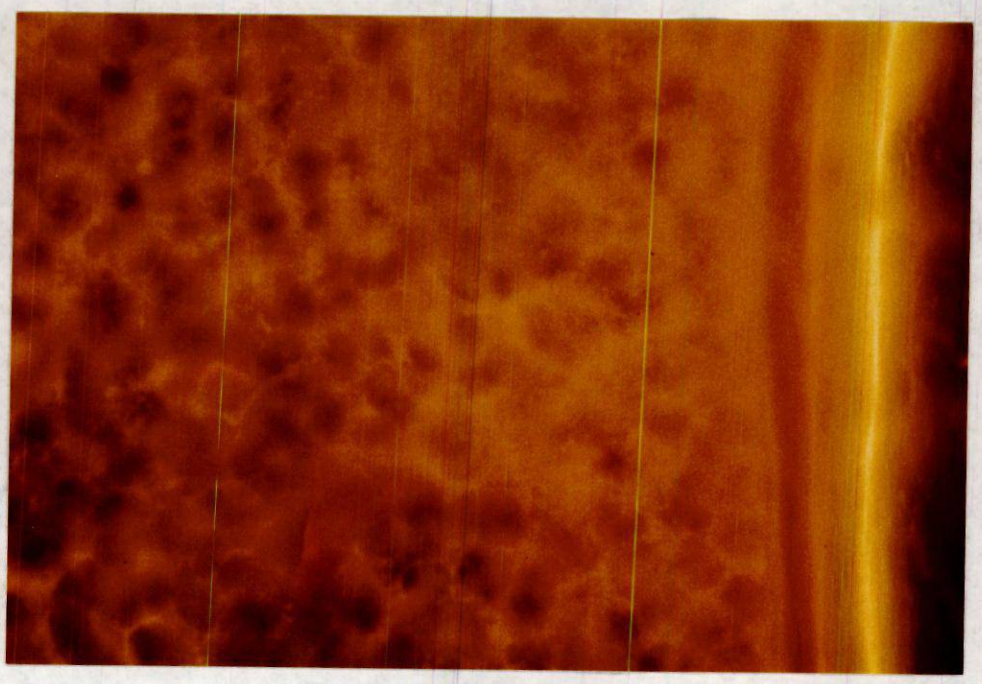
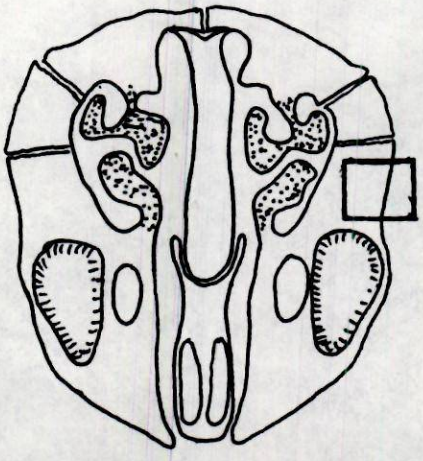


PLATE XV

Remodeling pattern of the maxillary bone above the incisors at the palatal foramen level.





The incisivo-maxillary, the incisivo-nasal and the midnasal sutures were almost equally active on both sides.

In the experimental group the remodeling pattern was virtually identical to that described for the control group. The two investigators in addition to the author who examined the slides as a blind test were unable to distinguish the experimental and control groups.

Cephalometric Evaluation

Descriptive Statistics

Means ($\bar{x}$), standard denations (SD) and standard errors (σ) for the 112 measurements (variables) for both the experimental and control groups are presented in Table III. It should be noted that some of the variables described the same measure. Table IV shows which variables describe the same measure by comparing their mean values between the right and left sides. It can be seen that there is a small difference between the right and left sides, with the left side dominating over the right side. However, this difference occurs in both the experimental and control groups.

By comparing variables 65 to 80, which describe the anterior development of various ancetomic landmarks, it can be seen that they are almost identical. The same observation is also noted when comparing variables 97 to 112, which describes local anteroposterior advancements between the various craniofacial components.

Inferential Statistics

Simple T-test was applied to test any statistical significance at the .05 level between right and left sides for both the experi-

mental and control groups. Table V presents the results of this test. It is noted that there is no statistical significance for any of the variables.

Enlargement Error

To test any enlargement error, a T-test was applied on the variable 113 between experimental and control group. Table VI represents the results of this test.

Error Study

To determine the reliability of the measurements, six animals (4 experimental and 2 control) were chosen at random and their cephalograms were re-enlarged with the cephalometric landmarks repunched. Storage of information and execution of calculations was accomplished with the same way previously used. A t-test at .05 level was applied between the first and second measurements to detect any statisticaly significant difference. Table VII, represent means, standard deviations and standard errors of the variables used and Table VIII, represents the t-test applied.

TABLE III

Means, Standard Deviations, Standard Errors, of Experimental and Control Group

	Experimental Group			Control Group		
Variable	$\bar{x}$	sd.	σ	$\bar{x}$	sd	σ
V_1	12.8350	3.578	1.131	11.8398	2.034	0.830
V_2	14.0020	3.172	1.003	13.9365	1.500	0.612
V_3	16.0570	2.713	0.858	15.8363	1.565	0.639
V_4	17.5699	2.299	0.727	17.527	1.184	0.483
V_5	15.2664	2.241	0.709	15.2710	1.184	0.483
V_6	16.5779	1.714	0.542	17.1683	1.000	0.408
V_7	18.2456	1.963	0.621	18.6343	0.690	0.282
V_8	19.5817	1.695	0.536	19.3812	1.376	0.562
V_9	25.4144	1.637	0.518	25.9465	1.053	0.430
V_{10}	25.6458	1.280	0.405	25.5743	0.749	0.306
V_{11}	35.6054	1.267	0.401	36.1923	1.045	0.426
V12	35.4802	1.089	0.344	35.9688	1.199	0.489
V_{13}	47.1604	1.308	0.414	47.1022	0.383	0.156
V_{14}	46.8541	1.048	0.331	46.5717	1.470	0.600
V_{15}	33.3877	0.16	0.258	37.9105	10.908	4.453
V_{16}	31.9669	5.032	1.591	33.7253	0.247	0.101
V_{17}	13.1693	3.373	1.067	12.4490	2.048	0.836
V_{18}	13.2209	3.149	0.996	13.3267	1.277	0.521
V_{19}	16.6932	2.512	0.794	16.3367	1.328	0.542
V_{20}	16.9437	2.279	0.721	17.0308	0.895	0.365

TABLE III (Cont)

Means, Standard Deviations, Standard Errors of Experimental and Control Group

	Experimental Group			Control Group		
Variable	$\bar{x}$	sd.	σ	$\bar{x}$	sd	σ
V_{21}	15.7511	2.169	0.686	15.6502	0.958	0.391
V_{22}	16.0954	1.691	0.535	16.7913	0.855	0.349
V_{23}	18.6859	1.890	0.598	18.9810	0.739	0.302
V_{24}	19.0991	1.610	0.509	19.0385	1.315	0.537
V_{25}	25.7525	1.524	6.482	26.3133	1.010	0.412
V_{26}	25.3137	1.262	0.399	25.3122	0.749	0.306
V_{27}	35.5390	1.255	0.397	36.1485	1.063	0.434
V_{28}	35.5435	1.053	0.333	36.0117	1.220	0.498
V_{29}	46.9441	1.233	0.390	46.9368	0.491	0.201
V_{30}	47.0743	0.955	0.302	46.7388	1.410	0.576
V_{31}	33.1006	0.781	0.247	37.7055	11.176	4.562
V32	32.2548	4.885	1.545	33.9557	0.494	0.202
V_{33}	12.7446	2.926	0.925	12.0385	1.738	0.710
V_{34}	13.6440	2.281	0.721	13.7385	1.091	0.445
V_{35}	16.3305	1.828	0.578	15.9723	1.120	0.457
V_{36}	17.3116	1.785	0.565	17.3962	0.713	0.291
V_{37}	15.445	1.535	0.485	15.3332	0.850	0.347
V_{38}	16.4050	1.081	0.342	17.1087	0.586	0.239
V_{39}	18.3985	1.152	0.364	18.6768	0.520	0.212
V_{40}	17.3878	1.261	0.399	19.3423	1.091	0.446

TABLE III (Cont)

Means, Standard Deviations, Standard Errors of Experimental and Control Group

	Experimental Group			Control Group		
Variable	$\bar{x}$	sd.	σ	$\bar{x}$	sd	σ
V_{41}	25.5070	0.798	0.252	25.9435	0.838	0.342
V_{42}	25.5628	1.159	0.367	25.5817	0.749	0.306
V_{43}	35.4465	1.232	0.390	36.0052	1.292	0.527
V_{44}	35.6382	1.056	0.334	36.1555	1.099	0.449
V_{45}	46.9067	1.298	0.411	46.8445	0.690	0.282
V_{46}	47.1104	1.024	0.324	46.8340	1.426	0.582
V_{47}	33.0914	0.922	0.291	37.6283	11.226	4.583
V_{48}	32.2576	4.962	1.569	34.0252	0.548	0.224
V_{49}	13.2055	1.426	0.451	12.4269	0.677	0.277
V_{50}	13.1981	1.567	0.495	13.3537	0.935	0.382
V_{51}	16.6889	0.783	0.247	16.2648	0.707	0.289
V52	16.9669	0.641	0.203	17.1107	0.732	0.299
V_{53}	15.6950	0.945	0.299	15.5247	0.540	0.220
V_{54}	16.1579	0.669	0.212	16.9177	1.189	0.485
V_{55}	18.6259	0.698	0.221	18.8398	0.337	0.137
V_{56}	19.1654	0.689	0.218	19.1805	1.114	0.455
V_{57}	25.6519	0.655	0.207	26.0452	0.456	0.186
V_{58}	25.4247	0.731	0.231	25.4798	1.159	0.473
V_{59}	35.2936	1.278	0.404	35.8695	1.518	0.620
V_{60}	35.7833	1.391	0.440	36.2778	0.809	0.330

TABLE III (Cont)

Means, Standard Deviations, Standard Errors of Experimental and Control Group

	Experimental Group			Control Group		
Variable	$\bar{x}$	sd.	σ	$\bar{x}$	sd	σ
V_{61}	46.6386	1.342	0.424	46.6220	1.244	0.508
V_{62}	47.3819	1.712	0.541	47.0557	1.229	0.502
V_{63}	32.7695	0.941	0.298	37.3303	10.854	4.431
V_{64}	32.5590	5.565	1.760	34.2813	1.119	0.457
V_{65}	155.8752	3.790	1.199	156.6670	2.741	1.119
V_{66}	155.6333	4.286	1.355	157.0447	3.356	1.370
V_{67}	135.7747	3.392	1.073	136.1105	2.762	1.127
V_{68}	135.1320	3.682	1.164	136.3682	3.068	1.252
V_{69}	115.7132	2.814	0.890	115.5627	2.447	0.999
V_{70}	115.8266	3.012	0.952	115.9167	2.815	1.149
V_{71}	109.6185	2.473	0.782	109.8177	2.640	1.078
V72	109.1657	2.835	0.896	109.4657	2.941	1.201
V_{73}	95.7585	2.030	0.642	95.6256	2.299	0.939
V_{74}	95.4753	2.468	0.780	95.6620	2.703	1.104
V_{75}	42.6753	1.493	0.472	42.2733	1.011	0.413
V_{76}	42.8445	1.530	0.484	43.2307	1.202	0.491
V_{77}	22.4127	1.139	0.360	12.3780	0.769	0.314
V_{78}	22.2834	1.608	0.509	22.3535	1.483	0.606
V_{79}	13.2307	1.262	0.399	10.4853	5.169	2.110
V_{80}	12.9450	1.540	0.487	12.2648	0.876	0.358

TABLE III (Cont)

Means, Standard Deviations, Standard Errors of Experimental and Control Group

	Experimental Group			Control Group		
Variable	$\bar{x}$	sd.	σ	$\bar{x}$	sd	σ
V_{81}	13.2596	1.415	0.447	12.3253	0.681	0.278
V_{82}	13.1289	1.519	0.480	13.4472	1.053	0.430
V_{83}	16.8008	0.919	0.291	15.9245	1.079	0.441
V_{84}	16.8410	0.838	0.265	17.4435	1.255	0.513
V_{85}	15.8769	1.359	0.430	14.9493	1.233	0.503
V_{86}	15.9685	1.426	0.451	17.4897	2.143	0.875
V_{87}	18.8177	1.209	0.382	18.1983	1.408	0.575
V_{88}	18.9681	1.629	0.515	19.8315	1.838	0.750
V_{89}	25.8961	1.611	0.509	25.2368	1.208	0.493
V_{90}	25.1716	1.816	0.574	26.2947	2.537	1.036
V_{91}	35.7432	2.383	0.754	34.4308	1.879	0.767
V_{92}	35.3452	2.659	0.841	37.7248	3.032	1.238
V_{93}	47.1688	2.810	0.889	44.9308	1.948	0.795
V_{94}	46.8581	2.302	0.728	48.7455	3.133	1.279
V_{95}	33.3360	2.869	0.907	35.5317	11.731	4.789
V_{96}	32.0218	4.529	1.432	36.0995	2.290	0.935
V_{97}	74.3007	1.898	0.600	73.0608	2.066	0.844
V_{98}	73.5442	1.392	0.440	72.0758	3.130	1.278
V_{99}	23.2729	0.770	0.243	23.4003	0.825	0.337
V_{100}	23.5781	1.071	0.339	23.6293	0.843	0.344

TABLE III (Cont)

Means, Standard Deviations, Standard Errors
of Experimental and Control Group

Variable	Experimental Group			Control Group		
	$\bar{x}$	sd.	σ	$\bar{x}$	sd	σ
V_{101}	54.0013	1.374	0.434	54.1565	1.235	0.504
V_{102}	53.6122	1.768	0.559	53.6688	1.743	0.712
V_{103}	22.3589	1.206	0.381	22.4397	0.488	0.199
V_{104}	22.3603	1.257	0.397	22.1033	0.900	0.368
V_{105}	40.2755	2.030	0.642	41.2003	1.593	0.650
V_{106}	39.9418	2.023	0.640	41.3507	1.682	0.687
V_{107}	16.2886	1.841	0.582	15.2538	1.084	0.443
V_{108}	16.3360	1.822	0.576	15.9970	1.404	0.573
V_{109}	74.1750	1.956	0.618	74.4057	1.065	0.435
V_{110}	74.1833	2.036	0.644	74.7588	1.551	0.633
V_{111}	150.4429	2.340	0.740	151.0883	2.040	0.833
V_{112}	150.4502	2.804	0.887	151.3417	3.229	1.318
V_{113}	16.3161	0.151	0.048	16.3290	0.106	0.043

TABLE IV

Variables describing the same measure.

Variables	Measure
V_1, V_{17}, V_{33}, V_{49}, V_{81}	Right lateral nasal bone development
V_2, V_{18}, V_{34}, V_{50}, V_{82}	Left lateral nasal bone development
V_3, V_{19}, V_{35}, V_{51}, V_{83}	Right lateral development of incisivomaxillary suture
V_4, V_{20}, V_{36}, V_{52}, V_{84}	Left lateral development of incisivomaxillary suture
V_5, V_{21}, V_{37}, V_{53}, V_{85}	Right lateral development of 1st molar
V_6, V_{22}, V_{38}, V_{54}, V_{86}	Left lateral development of 1st molar
V_7, V_{23}, V_{39}, V_{55}, V_{87}	Right lateral development of zygomatic process of maxilla anterior
V_8, V_{24}, V_{40}, V_{56}, V_{88}	Left lateral development of zygomatic process of maxilla surface
V_9, V_{25}, V_{41}, V_{57}, V_{89}	Right lateral development of zygomatic process of maxilla
V_{10}, V_{26}, V_{42}, V_{58}, V_{90}	Left lateral development of zygomatic process of maxilla
V_{11}, V_{27}, V_{43}, V_{59}, V_{91}	Right lateral development of the anterior part of the mandibular fossa
V_{12}, V_{28}, V_{44}, V_{60}, V_{92}	Left lateral development of the anterior part of the mandibular fossa
V_{13}, V_{29}, V_{45}, V_{61}, V_{93}	Right lateral development of the posterior part of the mandibular fossa
V_{14}, V_{30}, V_{46}, V_{62}, V_{94}	Left lateral development of the posterior part of the mandibular fossa
V_{15}, V_{31}, V_{47}, V_{63}, V_{95}	Right lateral development of tympanic bulla
V_{16}, V_{32}, V_{48}, V_{64}, V_{96}	Left lateral development of tympanic bulla

TABLE V

T-test comparison between right and left side on both experimental and control group.

Variable	Experimental Group			Control Group		
	T-Value	Degrees of Freedom	2-tail probability	T-Value	Degrees of Freedom	2-Tail probability
V_1 vrs V_2	0.82	9	0.433	1.50	5	0.194
V_3 vrs V_4	0.97	9	0.355	1.52	5	0.190
V_5 vrs V_6	1.10	9	0.302	2.70	5	0.043
V_7 vrs V_8	1.17	9	0.273	0.96	5	0.838
V_9 vrs V_{10}	0.27	9	0.791	-0.97	5	0.378
V_{11} vrs V_{12}	-0.28	9	0.787	-0.58	5	0.585
V_{13} vrs V_{14}	-0.67	9	0.522	-0.99	5	0.367
V_{15} vrs V_{16}	-0.89	9	0.394	-0.93	5	0.397
V_{17} vrs V_{18}	0.03	9	0.979	0.66	5	0.539
V_{19} vrs V_{20}	0.17	9	0.869	0.77	5	0.475
V_{21} vrs V_{22}	0.30	9	0.774	1.96	5	0.107
V_{23} vrs V_{24}	0.39	9	0.703	0.08	5	0.943
V_{25} vrs V_{26}	-0.55	9	0.597	-2.61	5	0.048
V_{27} vrs V_{28}	0.01	9	0.992	-0.32	5	0.760
V_{29} vrs V_{30}	0.35	9	0.733	-0.39	5	0.714
V_{31} vrs V_{32}	-0.57	9	0.585	-0.79	5	0.464
V_{33} vrs V_{34}	0.63	9	0.547	1.53	5	0.186
V_{35} vrs V_{36}	0.93	9	0.392	1.93	5	0.111
V_{37} vrs V_{38}	1.30	9	0.226	4.71	5	0.105
V_{39} vrs V_{40}	1.47	9	0.176	1.20	5	0.284

TABLE V (Cont)

Variable	Experimental Group			Control Group		
	T-Value	Degrees of Freedom	2-tail probability	T-Value	Degrees of Freedom	2-Tail probability
V_{41} vrs V_{42}	0.11	9	0.915	-3.20	5	0.027
V_{43} vrs V_{44}	0.47	9	0.650	0.29	5	0.782
V_{45} vrs V_{46}	0.46	9	0.565	-0.02	5	0.987
V_{47} vrs V_{48}	-0.54	9	0.605	-0.76	5	0.484
V_{49} vrs V_{50}	-0.03	9	0.977	1.78	5	0.135
V_{51} vrs V_{52}	0.93	9	0.376	1.54	5	0.184
V_{53} vrs V_{54}	1.41	9	0.193	2.33	5	0.067
V_{55} vrs V_{56}	2.16	9	0.059	0.65	5	0.543
V_{57} vrs V_{58}	-1.00	9	0.345	-1.66	5	0.157
V_{59} vrs V_{60}	0.83	9	0.430	0.75	5	0.487
V_{61} vrs V_{62}	0.97	9	0.359	0.59	5	0.580
V_{63} vrs V_{64}	-0.11	9	0.915	-0.67	5	0.531
V_{65} vrs V_{66}	0.71	9	0.493	-1.23	5	0.274
V_{67} vrs V_{68}	1.41	9	0.193	-0.58	5	0.590
V_{69} vrs V_{70}	-0.37	9	0.723	-1.59	5	0.174
V_{71} vrs V_{72}	1.81	9	0.104	1.63	5	0.163
V_{73} vrs V_{74}	0.79	9	0.449	-0.19	5	0.858
V_{75} vrs V_{76}	-0.69	9	0.505	-5.93	5	0.202
V_{77} vrs V_{78}	0.33	9	0.748	-1.63	5	0.164
V_{79} vrs V_{80}	0.91	9	0.385	-0.87	5	0.422

TABLE V (Cont)

Variable	Experimental Group			Control Group		
	T-Value	Degrees of Freedom	2-tail probability	T-Value	Degrees of Freedom	2-Tail probability
V_{81} vrs V_{82}	-0.31	9	0.764	1.89	5	0.117
V_{83} vrs V_{84}	0.09	9	0.929	1.63	5	0.164
V_{85} vrs V_{86}	0.02	9	0.910	1.88	5	0.119
V_{87} vrs V_{88}	-0.54	9	0.605	-0.76	5	0.484
V_{89} vrs V_{90}	-0.71	9	0.494	0.71	5	0.510
V_{91} vrs V_{92}	-0.27	9	0.794	1.75	5	0.141
V_{93} vrs V_{94}	-0.21	9	0.842	1.89	5	0.117
V_{95} vrs V_{96}	-0.75	9	0.473	0.11	5	0.920
V_{97} vrs V_{98}	1.34	9	0.212	1.52	5	0.189
V_{99} vrs V_{100}	-1.05	9	0.321	-0.39	5	0.713
V_{101} vrs V_{102}	0.95	9	0.368	1.76	5	0.138
V_{103} vrs V_{104}	-0.01	9	0.994	1.11	5	0.319
V_{105} vrs V_{106}	1.20	9	0.260	-0.62	5	0.562
V_{107} vrs V_{108}	-0.11	9	0.915	-1.54	5	0.183
V_{109} vrs V_{110}	-0.04	9	0.965	-0.86	5	0.429
V_{111} vrs V_{112}	-0.02	9	0.986	-0.38	5	0.917

TABLE VI

Mean ($\bar{x}$), Standard deviation (sd), Standard error ( ) and T-test comparison between control and experimental enlargement variables.

Group	Number of cases	$\bar{x}$	sd	σ	T-value	Degrees of Freedom	2-tail Probability
Experimental	10	16.3161	0.151	0.048	-0.18	14	0.858
Control	6	16.3290	0.106	0.043			

TABLE VII

Means, Standard deviations and standard errors for error study.

	Initial			Second		
Variable	$\bar{x}$	sd	σ	$\bar{x}$	sd	σ
V_1	11.6507	2.692	1.099	11.7765	2.420	0.988
V_2	13.7952	1.713	0.699	14.0822	1.423	0.581
V_3	15.9143	1.280	0.522	15.8650	1.161	0.474
V_4	17.4212	1.535	0.627	17.6347	1.663	0.679
V_5	15.2040	1.510	0.617	14.8230	1.236	0.505
V_6	16.4985	0.704	0.287	16.6280	1.412	0.576
V_7	18.4653	0.908	0.371	18.1718	0.799	0.326
V_8	19.3092	1.371	0.560	19.3928	1.483	0.606
V_9	25.7663	1.121	0.458	25.5317	1.286	0.525
V_{10}	25.6150	0.736	0.300	25.8508	0.654	0.267
V_{11}	35.2773	0.712	0.291	35.2667	0.708	0.289
V_{12}	35.4207	0.971	0.396	35.5608	1.167	0.477
V_{13}	46.7808	1.039	0.424	47.0300	1.058	0.432
V_{14}	46.4055	1.212	0.495	46.4537	1.622	0.662
V_{15}	33.3760	0.729	0.297	33.3397	0.824	0.336
V_{16}	33.6813	0.970	0.396	33.8500	0.772	0.315
V_{17}	12.1520	2.570	1.049	12.5307	2.806	1.145
V_{18}	13.2993	2.160	0.882	13.3220	2.071	0.846
V_{19}	16.3165	1.202	0.491	16.4752	1.473	0.601
V_{20}	17.0250	1.821	0.744	17.0293	2.186	0.892

TABLE VII (Cont)

Variable	Initial			Second		
	$\bar{x}$	sd	σ	$\bar{x}$	sd	σ
V_{21}	15.5057	1.502	0.613	15.2845	1.459	0.595
V_{22}	16.1967	1.124	0.459	16.1652	1.859	0.759
V_{23}	18.7373	1.164	0.468	18.5905	1.051	0.429
V_{24}	19.0427	1.426	0.582	18.9763	1.724	0.704
V_{25}	25.9772	0.984	0.420	25.8487	1.322	0.540
V_{26}	25.4090	1.000	0.408	25.5358	0.978	0.399
V_{27}	35.2392	0.722	0.295	35.2045	0.750	0.306
V_{28}	35.4567	0.927	0.379	35.6203	1.116	0.456
V_{29}	46.6533	1.093	0.446	46.8285	1.239	0.506
V_{30}	46.5427	1.039	0.424	46.6662	1.417	0.578
V_{31}	33.2043	0.802	0.327	33.0713	1.039	0.424
V_{32}	33.8595	0.855	0.349	34.1263	0.489	0.200
V_{33}	11.4900	2.588	1.057	12.0795	2.636	1.076
V_{34}	13.9537	2.279	0.931	13.7753	1.745	0.713
V_{35}	15.7435	1.182	0.483	16.0915	1.351	0.551
V_{36}	17.5943	1.920	0.784	17.4098	1.866	0.762
V_{37}	15.0207	1.168	0.477	14.9725	1.280	0.523
V_{38}	16.6788	1.053	0.430	16.4778	1.375	0.561
V_{39}	18.2777	0.906	0.370	18.3000	0.982	0.401
V_{40}	19.4980	1.279	0.522	19.2655	1.354	0.553

TABLE VII (Cont)

Variable	Initial			Second		
	$\bar{x}$	sd	σ	$\bar{x}$	sd	σ
V_{41}	25.5787	0.595	0.243	25.6093	1.278	0.522
V_{42}	25.8020	0.954	0.390	25.7775	0.561	0.229
V_{43}	35.0695	0.758	0.309	35.1425	0.913	0.373
V_{44}	35.6255	1.039	0.424	35.6845	1.143	0.467
V_{45}	46.5695	1.152	0.470	46.8325	1.395	0.569
V_{46}	46.6207	1.008	0.412	46.6582	1.400	0.572
V_{47}	33.1600	0.899	0.367	33.1072	1.231	0.503
V_{48}	33.9012	0.811	0.331	34.0880	0.589	0.240
V_{49}	12.4483	1.380	0.564	12.6335	1.204	0.492
V_{50}	13.0077	1.399	0.571	13.2348	1.077	0.440
V_{51}	16.4817	0.510	0.208	16.4988	0.599	0.245
V_{52}	16.8692	0.625	0.255	17.0067	0.523	0.213
V_{53}	15.5323	0.824	0.336	15.2428	0.778	0.317
V_{54}	16.1727	0.962	0.393	16.2022	1.151	0.470
V_{55}	18.7320	0.583	0.238	18.5325	0.745	0.304
V_{56}	19.0493	0.949	0.387	19.0362	0.985	0.402
V_{57}	25.8757	0.624	0.255	25.7498	0.986	0.402
V_{58}	25.5145	0.683	0.279	25.6390	0.585	0.239
V_{59}	34.7698	0.299	0.122	34.9280	0.731	0.299
V_{60}	35.9240	1.483	0.605	35.9005	1.382	0.564

TABLE VII (Cont)

Variable	Initial			Second		
	$\bar{x}$	sd	σ	$\bar{x}$	sd	σ
V_{61}	46.0422	0.548	0.224	46.4822	1.011	0.413
V_{62}	47.1583	1.931	0.788	47.0140	2.296	0.937
V_{63}	32.5250	0.528	0.216	32.6815	1.025	0.419
V_{64}	34.5365	1.899	0.775	34.5085	1.535	0.626
V_{65}	157.3025	3.705	1.513	165.7490	4.610	1.882
V_{66}	157.0107	3.846	1.570	157.2258	5.176	2.113
V_{67}	136.2035	4.041	1.650	136.0405	4.520	1.845
V_{68}	136.0190	3.859	1.575	135.9540	4.675	1.908
V_{69}	115.7002	3.372	1.377	115.6772	3.840	1.568
V_{70}	115.9578	3.093	1.263	116.2258	3.967	1.620
V_{71}	109.5988	3.084	1.259	109.5992	2.927	1.195
V_{72}	109.2187	3.335	1.361	109.6138	3.321	1.356
V_{73}	95.3308	2.380	0.972	95.3923	2.686	1.097
V_{74}	95.3020	2.557	1.044	95.4793	2.834	1.157
V_{75}	42.6238	1.904	0.777	42.7102	2.231	0.911
V_{76}	43.2008	1.671	0.682	43.0870	1.927	0.787
V_{77}	22.1698	1.377	0.562	22.4008	1.525	0.623
V_{78}	22.0173	1.385	0.565	21.9795	1.598	0.652
V_{79}	13.0242	1.288	0.526	13.1113	1.639	0.669
V_{80}	12.8630	1.557	0.636	12.7687	1.484	0.606

TABLE VII (Cont)

Variable	Initial			Second		
	$\bar{x}$	sd	σ	$\bar{x}$	sd	σ
V_{81}	12.4170	1.355	0.553	12.5828	1.138	0.465
V_{82}	13.0387	1.410	0.576	13.2745	1.171	0.478
V_{83}	16.3720	0.669	0.273	16.3627	0.814	0.332
V_{84}	16.9768	0.547	0.223	17.1413	0.564	0.230
V_{85}	15.3532	0.971	0.396	15.0208	1.055	0.431
V_{86}	16.3508	1.297	0.522	16.4248	1.688	0.689
V_{87}	18.5282	0.912	0.372	18.2827	1.422	0.581
V_{88}	19.2570	1.095	0.447	19.2852	1.162	0.474
V_{89}	25.6217	1.018	0.415	25.4338	1.145	0.467
V_{90}	25.7693	0.946	0.386	25.9525	1.586	0.648
V_{91}	34.3408	1.301	0.531	34.3925	1.983	0.810
V_{92}	36.3557	0.915	0.373	36.4387	2.011	0.821
V_{93}	45.5430	1.370	0.559	45.8578	2.278	0.930
V_{94}	47.6617	1.012	0.413	47.6460	1.746	0.713
V_{95}	31.9956	1.036	0.423	32.0245	2.190	0.894
V_{96}	35.0717	0.826	0.337	35.1767	1.669	0.681
V_{97}	74.8632	1.361	0.556	75.3513	1.425	0.582
V_{98}	73.4383	1.861	0.760	73.6060	2.029	0.828
V_{99}	23.3273	0.973	0.397	23.3280	1.457	0.595
V_{100}	24.0220	0.781	0.319	23.9463	0.880	0.359

TABLE VII (Cont)

Variable	Initial			Second		
	$\bar{x}$	sd	σ	$\bar{x}$	sd	σ
V_{101}	53.4300	1.372	0.560	53.4562	0.998	0.407
V_{102}	53.1740	1.295	0.496	53.4397	1.409	0.575
V_{103}	22.8453	1.337	0.546	22.8493	1.194	0.487
V_{104}	22.7268	1.178	0.481	22.8588	1.367	0.558
V_{105}	41.7223	1.274	0.520	41.1603	1.426	0.582
V_{106}	41.2003	1.416	0.578	41.1365	2.023	0.826
V_{107}	15.1945	1.661	0.678	15.6882	1.190	0.486
V_{108}	15.8660	1.322	0.540	16.0167	1.307	0.534
V_{109}	75.1337	2.198	0.897	75.1393	2.007	0.819
V_{110}	75.2027	2.094	0.855	75.2205	2.068	0.844
V_{111}	150.8262	2.765	2.129	150.8282	3.556	1.452
V_{112}	151.6190	2.938	1.200	151.7697	3.560	1.453

TABLE VIII

T-test between first and second calculations for reliability of measurements.

Variables	T Value	Degrees of Freedom	2-tail Prob.	Variables	T Value	Degree of Freedom	2-tail Prob.
V_1 vrs V_1	0.09	10	0.934	V_{23} vrs V_{23}	0.23	10	0.822
V_2 vrs V_2	0.32	10	0.759	V_{24} vrs V_{24}	0.07	10	0.944
V_3 vrs V_3	0.07	10	0.946	V_{25} vrs V_{25}	0.19	10	0.852
V_4 vrs V_4	0.23	10	0.822	V_{26} vrs V_{26}	0.22	10	0.829
V_5 vrs V_5	0.48	10	0.643	V_{27} vrs V_{27}	0.08	10	0.937
V_6 vrs V_6	0.20	10	0.845	V_{28} vrs V_{28}	0.28	10	0.788
V_7 vrs V_7	0.49	10	0.565	V_{29} vrs V_{29}	0.26	10	0.800
V_8 vrs V_8	0.10	10	0.921	V_{30} vrs V_{30}	0.17	10	0.867
V_9 vrs V_9	0.34	10	0.743	V_{31} vrs V_{31}	0.25	10	0.809
V_{10} vrs V_{10}	0.59	10	0.570	V_{32} vrs V_{32}	0.66	10	0.522
V_{11} vrs V_{11}	0.03	10	0.980	V_{33} vrs V_{33}	0.39	10	0.704
V_{12} vrs V_{12}	0.23	10	0.826	V_{34} vrs V_{34}	0.15	10	0.882
V_{13} vrs V_{13}	0.41	10	0.689	V_{35} vrs V_{35}	0.47	10	0.645
V_{14} vrs V_{14}	0.06	10	0.955	V_{36} vrs V_{36}	0.17	10	0.869
V_{15} vrs V_{15}	0.08	10	0.937	V_{37} vrs V_{37}	0.07	10	0.947
V_{16} vrs V_{16}	0.33	10	0.746	V_{38} vrs V_{38}	0.28	10	0.782
V_{17} vrs V_{17}	0.24	10	0.812	V_{39} vrs V_{39}	0.04	10	0.968
V_{18} vrs V_{18}	0.02	10	0.986	V_{40} vrs V_{40}	0.31	10	0.766
V_{19} vrs V_{19}	0.20	10	0.842	V_{41} vrs V_{41}	0.05	10	0.964
V_{20} vrs V_{20}	0.00	10	0.997	V_{42} vrs V_{42}	0.05	10	0.958
V_{21} vrs V_{21}	0.26	10	0.801	V_{43} vrs V_{43}	0.15	10	0.883
V_{22} vrs V_{22}	0.04	10	0.972	V_{44} vrs V_{44}	0.09	10	0.927

TABLE VIII (Cont)

Variables	T Value	Degrees of Freedom	2-tail Prob.	Variables	T Value	Degree of Freedom	2-tail Prob.
V_{45} vrs V_{45}	0.36	10	0.729	V_{69} vrs V_{69}	0.10	10	0.991
V_{46} vrs V_{46}	0.05	10	0.959	V_{70} vrs V_{70}	0.13	10	0.899
V_{47} vrs V_{47}	0.08	10	0.934	V_{71} vrs V_{71}	0.00	10	1.000
V_{48} vrs V_{48}	0.46	10	0.658	V_{72} vrs V_{72}	0.21	10	0.841
V_{49} vrs V_{49}	0.25	10	0.809	V_{73} vrs V_{73}	0.04	10	0.967
V_{50} vrs V_{50}	0.32	10	0.759	V_{74} vrs V_{74}	0.11	10	0.912
V_{51} vrs V_{51}	0.05	10	0.958	V_{75} vrs V_{75}	0.07	10	0.944
V_{52} vrs V_{52}	0.41	10	0.688	V_{76} vrs V_{76}	0.11	10	0.915
V_{53} vrs V_{53}	0.63	10	0.545	V_{77} vrs V_{77}	0.28	10	0.789
V_{54} vrs V_{54}	0.05	10	0.963	V_{78} vrs V_{78}	0.04	10	0.966
V_{55} vrs V_{55}	0.52	10	0.617	V_{79} vrs V_{79}	0.10	10	0.920
V_{56} vrs V_{56}	0.02	10	0.982	V_{80} vrs V_{80}	0.11	10	0.917
V_{57} vrs V_{57}	0.26	10	0.797	V_{81} vrs V_{81}	0.23	10	0.823
V_{58} vrs V_{58}	0.34	10	0.742	V_{82} vrs V_{82}	0.32	10	0.759
V_{59} vrs V_{59}	0.49	10	0.634	V_{83} vrs V_{83}	0.02	10	0.983
V_{60} vrs V_{60}	0.03	10	0.978	V_{84} vrs V_{84}	0.51	10	0.619
V_{61} vrs V_{61}	0.94	10	0.371	V_{85} vrs V_{85}	0.57	10	0.583
V_{62} vrs V_{62}	0.12	10	0.909	V_{86} vrs V_{86}	0.09	10	0.934
V_{63} vrs V_{63}	0.33	10	0.746	V_{87} vrs V_{87}	0.36	10	0.729
V_{64} vrs V_{64}	0.03	10	0.978	V_{88} vrs V_{88}	0.04	10	0.966
V_{65} vrs V_{65}	0.23	10	0.823	V_{89} vrs V_{89}	0.30	10	0.770
V_{66} vrs V_{66}	0.08	10	0.936	V_{90} vrs V_{90}	0.24	10	0.813
V_{67} vrs V_{67}	0.07	10	0.949	V_{91} vrs V_{91}	0.05	10	0.958
V_{68} vrs V_{68}	0.03	10	0.980	V_{92} vrs V_{92}	0.09	10	0.928

TABLE VIII (Cont).

Variables	T Value	Degrees of Freedom	2-tail Prob.
V_{93} vrs V_{93}	0.29	10	0.778
V_{94} vrs V_{94}	0.02	10	0.985
V_{95} vrs V_{95}	0.03	10	0.978
V_{96} vrs V_{96}	0.14	10	0.893
V_{97} vrs V_{97}	0.61	10	0.557
V_{98} vrs V_{98}	0.15	10	0.884
V_{99} vrs V_{99}	0.00	10	0.999
V_{100} vrs V_{100}	0.16	10	0.878
V_{101} vrs V_{101}	0.04	10	0.971
V_{102} vrs V_{102}	0.35	10	0.734
V_{103} vrs V_{103}	0.01	10	0.996
V_{104} vrs V_{104}	0.18	10	0.861
V_{105} vrs V_{105}	0.72	10	0.488
V_{106} vrs V_{106}	0.06	10	0.951
V_{107} vrs V_{107}	0.59	10	0.567
V_{108} vrs V_{108}	0.20	10	0.847
V_{109} vrs V_{109}	0.00	10	0.997
V_{110} vrs V_{110}	0.01	10	0.998
V_{111} vrs V_{111}	0.00	10	0.999
V_{112} vrs V_{112}	0.08	10	0.938

DISCUSSION

The purpose of the present study was to investigate the effects of altered nasal function on craniofacial growth. More specifically, it was intended to investigate if there is any relationship between imbalanced nasal function and regional craniofacial asymmetries. Furthermore, it was also intended to determine if nasal function is a significant factor controlling midfacial growth.

The most important function of the nasal system include breathing, olfaction, filtration and vocal resonance. One of these functions needing clarification is nasal breathing. Four parameters, including airflow, resistance, differential pressure and nasal cycle must be taken into account.

The nasal cycle, a phenomenon in which alternating changes in patency of the two nasal cavities occur, has been found to occur in about 80% of human population (Lillie, 1923; Heetderks, 1927; Hasegawa and Kern, 1977; Stoksted, 1976; Arbour and Kern, 1975; Stoksted, 1952). If the nasal cycle also exists in animals, and specifically in guinea pigs, it is not presently known.

Nasal airflow and differential pressure (the pressure difference between nasopharynx and nasal openings) up to a certain point are linearly related. If this point is reached the nasal airflow is not increased even if the differential pressure is increased.

Nasal airflow is also dependent on nasal resistance. The higher the nasal resistance the lower the nasal airflow, but this

relationship is not a linear one. Some investigators (Kern, 1983; Sarver, 1979; Warren, 1970; hershey et al., 1976) relate airflow (v), resistance (R) and differential pressure (P) by applying the Ohm's Law, in which $R = \frac{P}{V}$. However, such a relationship is over-simplified and measurements taken by this method are invalid for the simple reason that the nasal airflow is not laminar.

Human beings are obligatory nasal breathers for the first six months of life and then prefer to breath through the nose thereafter (Stool & Houlihan, 1977). Although not clear, it seems that this preference is vital for the organism and has to do with the fact that inspired air should be properly warmed and moistened. A man who inspires dry air at 0°C will loose (evaporate) about 420 gr water per 24 hours in order to sature all inspired air with water vapor. Since this requires about 2200 calories/24 hr., he would use nearly all of his metabolic energy just to warm and humidify the inspired air (Bouhuys, 1974). Experimental work done by Ogura et al. (1966, 1968, 1973) and Ohnishi et al., (1969, 1972) added more information as to why nasal breathing is preferred. According to him nasal obstruction of whatever origin causes an increase of lower airway (trachea, bronchi) resistance.

On the other hand dogs forced to breath through their mouth develop a decreased PO_2 level and an increased PCO_2 level of their arterial blood. This in turn causes tissue dyspnea and increased pH, which is just the beginning of a vicious cycle, leading finally

to increased pulmonary vascular resistance and pulmonary artery hypertension.

The reasons for the existence of two nasal airways are unclear. The existence of the nasal cycle is also an enigma. However, it seems that their existence surely must serve some important role. The idea that a compartmented nasal airway can provide more surface so that the inspired air is better "serviced" conflicts with the nasal cycle.

Accepting the fact that two nasal airways exist for some important reason, it follows that if one does not function adequately, the other should be properly adjusted in order to compensate for whatever is lost. Such compensating changes have been reported in other parts of the body, e.g. the kidneys. Another approach leading to the same final point is the use-disuse growth theory (Goss, 1964). According to this theory disuse of an organ leads to atrophy while overuse results in hypertrophy. In the nose, compensating changes would be expected to occur in the epithelium, the underlying connective tissue, the turbinates and the bones surrounding the nasal cavities. Since the nose is mostly hosted by maxillae it follows that bone changes will likely occur within them resulting to various degrees of facial asymmetries.

In our experiment involving the suturing of one nasal opening, we removed some functions from the corresponding nasal airway. Specifically, inspiration and airconditioning were no longer performed on this side. If the organism needs the same amount of

oxygen during a given period, it follows that the airflow of the unblocked nasal airway should be doubled. This theoretically could happen by increasing the differential pressure or by decreasing the nasal resistance. There is strong evidence, at least for humans, that the second mechanism is more effective. Decreased nasal resistance could occur by decreasing the size of turbinates or by expanding the nasal walls. To what extent the decrease of turbinate size or the expansion of nasal walls can contribute and which factors control it is not presently known.

Contrary to this logical sequence, our findings based on histological, ground section and cephalometric evaluation clearly demonstrate that there is not any significant difference between the blocked and unblocked sides regarding epithelial type, gland type and distribution, nasolacrimal duct size and shape, turbinate size and shape, bone deposition pattern and nasal cavity form.

Before we try to interpret these findings it may be helpful to discuss briefly the relationship between form and function.

The relationship between form and function has long been a question for biological scientists including of course orthodontists. In particular the question of which comes first, the form or the function, has provoked many observations and speculations. It is believed that growth of a tissue is stimulated by the physiological demands for increased functional activity. For example, lymphatic organs enlarge when challenged antigenically, renal hyperplasia follows the need to secrete excess sodium, and muscles become

hypertrophic when called upon to work harder. However, few tissues perform only single functions despite their specializations. The problem arises, therefore, as to which of several functions is the one or ones that control growth. Do all of the specialized physiological activities of a tissue play equal roles in regulating growth or are the interests of all represented by just one, and if so, what considerations could have dictated its selection? Another interesting aspect is whether or not there is a limit in the size to which an organ or tissue can be stimulated to grow. It appears that many tissues are capable of considerably more growth than they ordinarily manifest, but whether or not the maximum size of a tissue or organ is really relatively unlimited is still unknown.

Functional demand and functional activity are so inseparably associated that it is difficult to study one in the absence of the other. Yet it is essential to know if growth is stimulated by the enhanced activity of a tissue or cell or by factors that initiated this activity in the first place.

There are compelling reasons to attribute growth stimulation to those physiological factors which promote increased functional activity and which are themselves activated by functional insufficiencies. The physiological feedbacks by which proper functional levels are maintained serve also to regulate organ and tissue growth as an accessory mechanism for controlling function. Although nerves and hormones are the main means of maintaining or stimulating functional output by effector organs, there are many other factors, each

related to a specific function, by which growth can be controlled. For example (Goss, 1964) sodium blood level controls the growth and function of kidneys, the calcium blood level regulates the size and activity of parathyroid glands and the body's demands. Oxygen and its availability affect the entire respiratory mechanisms including the rate of erythropoiesis.

There seem to be as many growth-controlling mechanisms as there are physiological means for initiating functional activity. How functional demands are translated to the target cells responsible for growth is still unknown.

Returning to the nasal region, possible factors which could affect or determine its growth pattern are, in general, heredity, environment and functional activities.

Functional activities include nasal functions, forces coming from teeth (occlusal forces) and forces from muscles acting directly on the nasomaxillary complex.

The role of nasal functions as described in current literature (Moss, 1968(a), 1975; Moss and Salentijn, 1969(a), 1969(b); Rickets, 1979; Sarner, 1979; Freng, 1980) is not only oversimplified but also incompletely documented. As already stated, in our experiment two nasal functions inspiration and aircondition were exluded from one nasal airway. Contrary to common assumption, it was shown that these two functions do not have apparent effects on the naso-maxillary growth pattern.

Rickets (1979), however, stated that:

> "When we observe a lack of function in the nose, we know there can be growth inhibition. Harvold proved this in monkeys experimentally! It may be seen on only one side of the nose as the other serves as a normal control. If you study frontal headfilms, frequently you can see a small nasal cavity on one side and a larger one on the other. You may find that the patient had a unilateral obstruction and see the whole maxilla sucked inward and upward on that side."

It should be mentioned that the above quotation comes from a journal interview, that documentation is lacking, and that even if true, "function in the nose", is a very broad term and does not specify which function.

Sarner (1979), using a human sample, tried to evaluate the effect of airflow on facial morphology. His conclusion was that airflow does not have any relationship to facial morphology. However, his approach which was to relate final facial morphology with only one possible case (airflow) questions the reliability of his results.

Freng (1979) using models and lateral cephalometric x-rays on a sample of 16 people with congenital unilateral choanal atresia, concluded that neither dental occlusion nor the vertical facial growth diverged from normal. It is obvious that frontal cephalometric x-rays, which could provide valuable information in this study, were not used.

The manner of conventional application of the functional matrix theory as it relates to nasal functions and nasal form (Moss, 1962; Moss, 1968; Moss& Salentijn, 1969(a), 1969(b)) seems to be

imcomplete and oversimplified since two functions related to nasal breathing proved to have no effect on the nasomaxillary morphology.

Cracovaner and Goodman (1956) reported that unilateral choanal atresia produced "atrophy and underdevelopment of the turbinates, narrowing of the nasal passages and a high-arched palate." The difference between our results and these findings can be explained by the fact that choanal atresia blocked off not only inspiration and airconditioning, as in our experiment, but also expiration. It seems possible that expiration provides a necessary functional stimulus for nasomaxillary growth. Another possibility for this discrepancy could originate from the structural differences between human nose and the guinea pig nose.

Chierici et al. (1973) reported:

> "The findings reported here suggest that the mechanism controlling the position of the lateral walls of the nasal cavity is relatively independent of the shape and position of the tooth carrying part of the maxilla. The lateral walls are more likely responsive to the demands of the respiratory system."

The first part of the above report deals with a very controversial point since other investigators have shown a positive relationship between occlusal forces and nasomaxillary form. The second point, considering the new information coming from the present study, needs to be more specific. However, if Chierici et al. (1973) are correct in regards to the lateral nasal expansion and respiratory demands relationship, then it is apparent that expiration should play an important role in determining nasomaxillary growth pattern.

Additional information regarding the role of nasal functions in determining nasomaxillary growth pattern can be derived from certain craniofacial pathological conditions, such as cebocephalia and cyclopia, in which some nasal components are totally or partially lacking (Mahindra et al., 1973; Kukreja, 1973; Gifford et al., 1972; Latham, 1971(b); Currarino and Silverman, 1970; Ackerman et al., 1965).

A problem encountered in these circumstances is the difficulty in distinguishing those aspects of the deformity which relate to inadequate cell potential as part of a developmental malformation from those which are specific adaptational responses of otherwise normal structures.

In conclusion, to relate nasal functions in general to craniofacial form is seen as misleading approach. The findings of the present investigation show that, at least for the guinea pig craniofacial complex, two functions of the nasal breathing cycle (inspiration and airconditioning) have little if any apparent effect on the nasomaxillary growth pattern. This points to the need for detailed studies of all other nasal functions as they relate to final nasal and facial form.

SUMMARY AND CONCLUSIONS

While the effects of breathing mode on craniofacial growth have received great interest, no studies exist on the effects of imbalanced nasal function. Deviated nasal septum, hypertrophied turbinates, unilateral choanal atresia, and foreign objects are some of the common causes underlying imbalanced nasal function.

It was the purpose of the present study to examine experimentally any possible effects of imbalanced nasal function on craniofacial growth. Nineteen, 3 week old guinea pigs were used for this study. Six of them were used as control group and the remaining 13 as experimentals. In the experimental group the right nasal airway was blocked by suturing the anterior nasal opening. The animals were kept for four months after which they were sacrificed. Histological, ground sectioning and cephalometric data were prepared to evaluate any effect.

The right and left sides were compared in regards to size and shape of nasal cavities, type and distribution of epithelia, type and distribution of glands, size and shape of the turbinates, bone deposition pattern, size of the nasolacrimal duct and shape of nasal septum.

Histological and ground section data did not reveal any significant difference between the blocked and unblocked sides. Cephalometric analysis at the .05 level did not show also any significant difference between the blocked and unblocked sides.

The conclusions drawn from the present investigation are as follows:

1. The function of an organ or tissue does not always affect its growth pattern.
2. Inspiration and airconditioning do not have, at least for the guinea pig nasal complex, any apparent affect on nasomaxillary form.
3. The view that nasal function in general affects nasomaxillary form is oversimplified and imcomplete. Future research needs to make clear which other functions, if any, affects nasomaxillary growth pattern.

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