The Aponeurotic Tension Model of Craniofacial Growth in Man

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Abstract: Craniofacial growth is a scientific crossroad for the fundamental mechanisms of musculoskeletal physiology. Better understanding of growth and development will provide new insights into repair, regeneration and adaptation to applied loads. Traditional craniofacial growth concepts are insufficient to explain the dynamics of airway/vocal tract development, cranial rotation, basicranial flexion and the role of the cranial base in expression of facial proportions. A testable hypothesis is needed to explore the physiological pressure propelling midface growth and the role of neural factors in expression of musculoskeletal adaptation after the cessation of anterior cranial base growth.

A novel model for craniofacial growth is proposed for: 1. brain growth and craniofacial adaptation up to the age of 20; 2. explaining growth force vectors; 3. defining the role of muscle plasticity as a conduit for craniofacial growth forces; and 4. describing the effect of cranial rotation in the expression of facial form.

Growth of the viscerocranium is believed to be influenced by the superficial musculoaponeurotic systems (SMAS) of the head through residual tension in the occipitofrontalis muscle as a result of cephalad brain growth and cranial rotation. The coordinated effects of the regional SMAS develop a craniofacial musculoaponeurotic system (CFMAS), which is believed to affect maxillary and mandibular development.

Key Words: Brain, Aponeurotic, Airway, Mandible, Rotation, Muscle, SMAS.

INTRODUCTION

The soft tissue matrix, in which skeletal elements are embedded, is the primary determinant of growth, while both the bone and cartilage are secondary growth sites. Growth centers display inherent growth versus growth sites which are reactive [1,2].

This is the fundamental premise of the Functional Matrix Theory of Melvin Moss [3]. The soft tissue matrix (muscles, connective tissue, neural tissue) models the bone, rather than bone morphology being genetically determined.

Proposed is a descriptive model of craniofacial growth based on the principle that late brain growth and cranial rotation create a residual tension in the occipitofrontalis muscles which in turn loads the facial superficial musculoaponeurotic systems (SMAS) *via* connected fascia, muscle and ligaments and reflect a craniofacial musculoaponeurotic system (CFMAS).

PURPOSE

The purpose of this review is to provide a comparative, biologically accurate and clinically effective framework for understanding [4] the coordination of brain and craniofacial growth (CFG), and the relationship of brain growth to cranial rotation, airway and vocal development. There is a strong belief that the musculoaponeurotic system of the skull has a direct effect on maxillary and mandibular development, and forms the underlying theme of the proposed aponeurotic tension model of craniofacial growth.

TRADITIONAL CONCEPTS

Moss introduced the functional matrix theory describing skeletal growth as a secondary, compensatory, and mechanically obligatory response to temporally and morphogenetically prior growth changes in specially related tissues and organs [5-7]. A solitary growth matrix for the entire head is difficult to explain, therefore Moss divided the head into areas (capsular matrices) such as the neurocranial capsular matrix and the orofacial capsular matrix [8], the latter being comprised of the teeth, sinus spaces, muscles and connective tissue (blood vessels, etc...). Neurocranial capsular matrix enlargement resulting from neural growth seems self-evident. However, the nature of the orofacial capsular matrix growth is more elusive. It is believed that the orofacial capsular matrix enlargement is driven by airway enlargement and that the direction of this facial growth is caudad and ventral (Fig. **1**).

The nasal septum model of Scott [9] describes the nasal septum as a growth center forcing the viscerocranium caudad and ventral relative to the cranial base until the facial sutures have become stabilized by dense connective tissue [9,10]. The nasal septum directs prenatal and some postnatal growth to the approximate age of 4 years, [4] but the brain is believed to be the primary growth center for CFG until approximately the age of 8 [11]. After neural growth is complete, the more inferior portions of the anterior cranial base (ACB) are considered to continue "growing" caudad and

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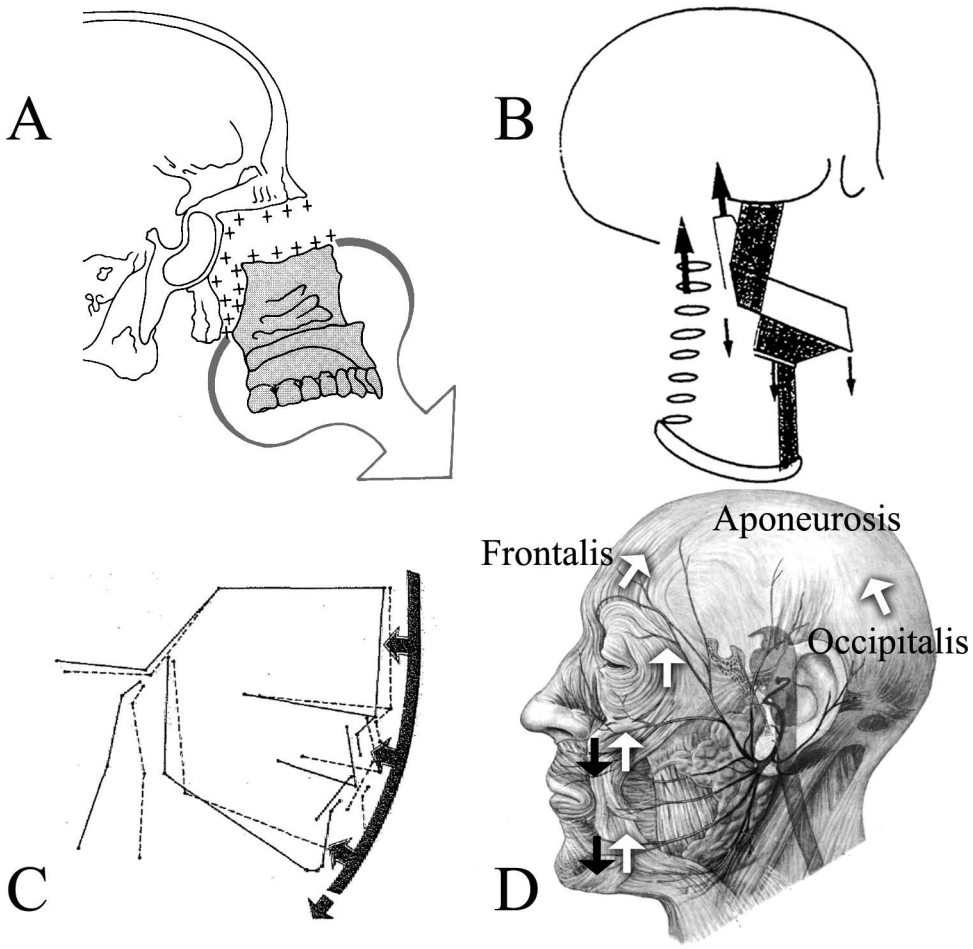

Fig. (1). (A) Displayed is the ventral and caudad growth direction of the ethmomaxillary complex Illustration from [1]. **(B)** Houston [16] combined his CFG model based on cervical vertebrae growth, with **(C)** the soft-tissue stretching of Solow [17] and cranial posture changes [114]. Illustrations from [16, 17]. Houston's model cannot explain forward/counter-clockwise mandibular rotation. **(D)** An anatomical drawing of the aponeurotic tension model of craniofacial growth. Shown are: the force of gravity (black arrows) and CFMAS tension (white arrows); the frontalis muscle (frontalis), the occipitalis muscle (occipitalis) and the area between is the location of the galea aponeurotica (aponeurosis). The modiolus (muscular confluence joining the upper portion of the muscle mask with the lower portion) is found vertically between the black arrows overlying the cheek and chin. Illustration adapted from [115].

ventral, causing drift and displacement of the ethmomaxillary (midface) complex [11-13] in conjunction with growth at the sphenooccipital synchondrosis (SOS: a hyaline cartilage growth center between the clivus of the occipital bone and sphenoid bone) [13]. The anteroposterior force of the SOS displacement is believed by some to be transmitted through the nasal septum acting as a strut connecting to the midface [4,13,14]. Alternatively, the advancement of the midface could be a result of physical growth forces of enclosing soft tissues. For example, displacement of facial sutures could result from the enlargement of muscles, [13] or from growth of the brain temporal lobe, infraorbital and retromaxillary fat pads, and infratemporal fossa contents (pterygoid muscles, fat pads) creating a laterally transmitted force to the midface [11,15].

The facial tissues have previously been described by Houston [16] and Solow [17] (Fig. **1**), as influencing facial growth but from the aspect of growth restriction and postural change through soft tissue forces by facial application of a dorsally directed force to the underlying skeletal structures. The Servosystem model of Petrovic [18] assumes the displacement of the midface through nasal septum growth in conjunction with direct thrust of labionarinary muscles, and through the superior labial frenum and septopremaxillary ligament [19]. As the maxilla is moved ventrally there is believed to be compensation maintaining the mandibular relationship to the maxilla.

Although each of the models has added to our attempt at understanding CFG, none of the CFG models seem to directly neither address nor provide a model for the differing patterns of maxillomandibular rotation in hypo- and hyperdivergent individuals as described by Bjork [20,21]. There are inconsistencies found in earlier theories that are believed more effectively addressed with the proposed CFG model. The proposed model explains why the airway enlarges sagittally, despite a backward slide of the vomer between infancy and adulthood [11]. If it were the only issue, the relative descent of the larynx would be plausible with traditional caudad and ventral CFG model as there is significant growth of the cervical vertebrae, for which the heights of the vertebrae roughly double by growth at their respective epiphyseal plates [22]. However, Houston's cervical growth

model [16] cannot explain forward mandibular rotation as the tissue are oriented to provide a dorso-caudad force.

Also, the maxilla is believed to display "growth" at the maxillary tuberosity causing pressure against the pterygoid plate of the sphenoid bone. This pressure is believed to displace the maxilla forward. However, between the pterygoid plate and maxilla there is a fibrous suture rather than synchondrosis. Compression in sutures has been shown to display resorption [23-25] and the bone (pterygoid plates) in the area is much too pliable in that environment to withstand the pressures [26]. The lack of inherent growth potential of sutures negates any ventral thrust of midface growth by the circumaxillary suture system [2,4,14]. The viscerocranial units (maxilla, mandible) seem suspended in CFMAS relative to cranial rotation concomitant with allometric brain growth. Further investigation of the degree of viscerocranial suspension by the CFMAS relative to elastic fiber and collagen fiber resistance within the suture itself seems warranted.

The relative fixed position of the zygomatic processes as the remainder of the maxilla is thrust forward is assumed to be a result of maxillary resorption anteriorly and deposition posteriorly [26]. Problematic is that the anterior resorption is superficial as the anterior surface displays stability relative to endosseous implants. Essentially, all of the increase in maxillary length occurs posteriorly [27-29]. Considering the limitations of microscopic histology, this process requires further study with intravital markers [30]. Cranial rotation better explains the observed pattern of surface resorption anteriorly due to pressure applied by CFMAS weight to the anterior region during the rotation. Surface resorption is created as bone advances into the drape of the CFMAS pressure which is created on the anterior bone leading to collapse of the vasculature, stimulating compensatory modeling. Deposition posteriorly is stimulated by tension created within the suture by cranial rotation and the associated force of the CFMAS. Cranial rotation and the facial block concept [12, 31] would rectify the conflict of Bjork's observations displaying a stable zygomatic surface relative to implants [27], while Enlow believed the zygoma relocated dorsally [13]. Both were correct. The rotation of the maxilla relative to the zygoma would display a stable zygoma surface relative to an implant, while slight surface resorption of the zygoma would be observed due to pressure of the facial muscle mask. The rotation of the maxilla would be displaced forward relative to the zygoma leading to the conclusion that the zygoma must be posteriorly displaced with growth. This also explains the observation that the mandible growth rotation is greater that maxillary growth rotation; at the same time, the mandible is also rotating around the maxilla [32].

ALTERNATIVE MODEL INTRODUCED TO SUPPLEMENT THE CURRENT UNDERSTANDING OF CRANIOFACIAL GROWTH IN MAN

Proposed is an alternative model of CFG based on musculoaponeurotic tension enveloping the head. This CFG model describes the affect of late cephalad growth of the brain pushing on the occipitofrontalis muscle (Figs. **1** and **2**), which places the muscles in tension, the peak in temporal and occipital lobe gray matter being at 16-20 of years age [33]. The tension force is transmitted from the occipitofrontalis muscles down through the mask of muscles overlying the face due to individual muscle fiber blending with adjacent muscles and the associated superficial musculoaponeurotic systems (SMAS; investing connective tissues) [34-40]. This facial muscle mask and associated regional SMAS are believed to be part of a CFMAS important in directing craniofacial development and jaw rotation by acting as a conduit for the brain derived force. Cranial rotation [12, 41] is also believed to occur sagittally around the atlantooccipital joint as a result of allometric brain growth and progressive facial bone pneumatization with sinus development.

Brain extension consists of uprighting of the cerebral portion of the brain, and therefore also the head, relative to the body axis during growth and development. This pattern is mimicked by cranial base (basicranial) flexure [12, 41] and airorhynchy (posterior and upper portions of the face rotate dorsally relative to the posterior cranial base by extension of the ACB relative to the posterior cranial base (PCB), analogous with the "facial block") [12] (Fig. **3**). Cranial rotation being intimately integrated with the latter, the term cranial rotation will be used collectively for them from this point. It is possible that the proposed model applies to only humans as it seems coordinated with cranial base flexion during growth which is unique to humans [12].

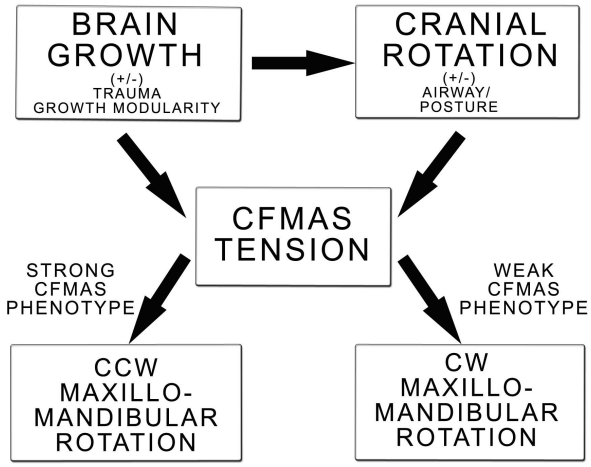

Fig. (2). Algorithmic adaptation of the proposed CFG model. Displayed is brain growth modulation of cranial rotation and CFMAS tension. Brain growth can display temporal regional growth and mylenization with normal development and as a result of trauma (concussion, drug use). Cranial rotation modulates CFMAS tension and itself is influenced by brain development and postural control. CFMAS tension manifests as as a strong or weak phenotype. A strong CFMAS phenotype will be expected to develop a counterclockwise/forward maxillomandibular rotation, while a weak CFMAS phenotype will be expected to develop a clockwise/backward maxillomandibular rotation pattern.

Brain Growth and Craniofacial Adaptation up to the Age of 20

Lateral cranial expansion is limited after approximately 1 year of age [10,14] and ACB anteroposterior growth is limited by 8 years of age with the fusion of sphenoethmoid, frontoethmoid and intersphenoid synchondroses [11]. The

mocranial capsule (dura) is anisotropic, being thicker at its base which grows slowly and resists the enlargement of the brain in the developing cranial base. Over the calvarial region it is thinner and less resistant, allowing the cerebral hemispheres, and to a lesser extent, the cerebellar hemispheres to expand more rapidly [10, 53]. Also, cephalad brain growth better explains why the entire cranial base surface is not resorptive, but displays areas of bone deposition: the petrous portion of the temporal bone, crista galli/foramen cecum, between the occipital lobes, and sella turcica [13, 48] (Fig. **4**). Continued relative growth of the PCB into the adolescent years is an important factor that has not been adequately appreciated. The relative effects of cephalad brain growth warrant further study.

Explaining Growth Force Vectors

Mitz and Peyronie (Mitz and Peyronie, 1976) originally coined the term SMAS, yet despite numerous publications on this subject, there remain significant variations in the anatomic descriptions of facial fascial anatomy, and descriptions of the relationship between the superficial and deep facial fascia remain imprecise [54]. The SMAS is a composite fibro-fatty layer comprising collagen and elastic fibers interspersed with fat cells. It microscopically displays a considerable amount of elastic fibers in close relationship to the collagen fibers, and the collagen fibers display a convoluted appearance similar to that found in the dermis [55]. The SMAS invests the superficially lying mimetic muscles (muscles of facial expression; e.g. platysma, orbicularis oculi, zygomaticus major, and risorius) and forms a continuous sheath throughout the head and neck, extending into the temporal region, forehead, scalp, malar areas, nose and upper lip. Thus the superficial facial fascia is intimately associated with the mimetic muscles [54, 56]. The mimetic muscles and SMAS function as a single anatomic unit in producing movement of facial skin [54] and the low viscoelastic properties of the SMAS are the reason for incorporation of the SMAS as a standard part of the rhytidectomy (facial lift) procedure [55, 57]. Also deserving further investigation and consideration are the relatively thick osteocutaneous retaining ligaments that anchor periosteum to dermis, notable being the zygomatic and mandibular ligaments [54] (Fig. **3**).

The coordinated effect of facial muscle and regional SMAS blending develops a CFMAS. The proposed model provides a more biologically correct explanation for observations that earlier models have been unable to provide satisfactorily. The tension conducted through the CFMAS explains the enlargement of airway and maxillomandibular rotation patterns consistent with the observations of implant studies [28, 58] and cephalometric superimposition referenced at I-point on the occipital condyles in *norma lateralis* [46, 47].

Growth associated force transmittance through mimetic muscles has previously been described by Delaire [59, 60]. CFMAS tension may also be related to resting muscle tonus, [61] which opposes gravity effects resulting from increase in tissue mass (e.g. muscle, fascia, skin, bone, connective tissue, associated hydration of these structures and any cantilever developed). During normal growth it is postulated that the tension through the CFMAS resists the effects of gravity until CFMAS attenuation with late aging [54]. Hemifacial paralysis, (e.g. Bell's Palsy) conveniently displays effect of muscular atonicity resulting in uncompensated gravitational forces upon tissue mass, which is tissue sag (Fig. **5**). CFMAS tension transmittance is amplified with muscular growth and development with puberty, along with densification and increased crosslinking of connective tissue component [62] (Fig. **5**). An increase in muscle size and fat deposit deep to the SMAS may also have a tendency to place the overlying associated SMAS in tension which exhibits some similarity to the work of Solow [17, 54, 56, 63].

Defining the Role of Muscle Plasticity as a Conduit for Craniofacial Growth Forces

The CMFAS is an unstable conduit for force transmittance and its development is age dependant, reactive and inherent (genetic).

The traditional view is that bone reacts to muscle forces but that muscle does not react to bone modeling. However, there is increasing evidence that the muscle response is also adaptive to underlying skeletal development [64]. The effect of gravity with increase in bone and muscle mass may stimulate muscle lengthening. In addition to functional muscle development, genetic properties may determine the number of muscle myofibrils, myofibers and myotubes [65, 66] and quality of the supporting connective tissue, thereby displaying individual variation.

During muscle growth, there is an increase in length by addition of sarcomeres at the muscle tendon junction for which the rate of sarcomeres addition may vary temporally depending on the individual muscle [67]. At a certain point, the addition of sarcomeres and associated increase in muscle fiber length with growth ceases. Any further increase in muscle belly length is presumed to be a reorganization of muscle fibers as insertion of myofibers into the tendon are not uniform but instead stagger [67]. However, the muscle continues to increase in girth due to myofibril splitting as a result of oblique forces within the sarcomeres when a critical diameter is reached [68]. Immobilization of limbs in both extended or contracted muscular positions displays a decrease in the number of sarcomeres relative to controls, presumably due to the restriction of function [67, 69] because the bone length in immobilized specimens is not significantly different than controls. This is assumed to be a result of increase in tendon length [67]. Immobilization is obviously not physiologic as it overpowers the normal function of the Golgi tendon apparatus and occult muscle tonus, thereby stressing ligaments and tendons beyond the normal viscoelastic limits [70]. Therefore, certain observations from immobilization experiments may not be representative of the normal growth process.

As muscle ages, there is a rapid increase in quantity and quality of muscle associated connective tissue and therefore CFMAS; [62, 71-73] this increase may be a phenomenon throughout the connective tissue in the body. Skeletal muscle growth is believed to be rate limited by connective tissue growth which controls myofiber diameter and length [73-75] increasingly as the intramuscular connective tissue arrangement becomes thicker and increasingly cross-linked with age [62] (Fig. **5**). The latter is resistant to lengthening compared to the rather compliant muscle fibers [71, 73] and relatively small increases in the muscle collagen content increases

[71, 73] until the age at which brain growth and cranial rotation cease and CFMAS attenuation [54] becomes an issue.

Tension is Transmitted from the Galea Aponeurotica/ Occipitofrontalis to the Cranial Base

It is postulated that the anteroposterior tension within the occipitofrontalis muscles is the cause of the asymmetric separation of the SOS. The weight of the brain on the desmocranial capsule (dural slings traverse the SOS; Fig. **3**) and cranial base, and anteroposterior tension muscle tension within the CFMAS, cause a pivot point at the superior aspect of the SOS creating a greater relative separation of the pharyngeal side of the SOS relative to the endocranial aspect [45, 79]. A biomechanical lever system would allow smaller increments of brain growth a greater significance in directing facial tension, (Fig. **3**) however, a finite element analysis is needed to adequately describe the stresses and distinguish effects.

Asymmetric growth at the SOS has been demonstrated with and without implants as radiologic markers [12, 41, 45, 79-81]. Hyaline cartilage becomes anabolic when induced by tensile stress, which also accelerates endochondral ossification at the pharyngeal surface of the SOS [80, 82-86]. Therefore, it is difficult to demonstrate the asymmetric growth of the SOS radiographically because of concomitant bone modeling. Consideration must be given to the SOS as a reactive site of growth rather than a primary growth center during the adolescent and early adult years [87]. Premature fusion of a cranial suture, craniosynostosis, causes flattening of the basicranium, [88] while inhibition of SOS growth creates a more flexed cranial base [12].

Implants demonstrate that growth of the face does not follow straight lines, but rather curves in association with sutural plane rotation [58]. Rotation of the cranium [12] seems related to allometric brain development [12, 33, 89] and possibly the progression of brain myelination patterns (the water content of fat being less than grey matter), brain capillary blood volume, [90] and progressive pneumatization of the facial bones [46] which collectively allow the head to remain balanced as facial tissues enlarge with growth. The physiologic result is a circular growth pattern around the basioccipital portion of the occipital condyles. The occipital condyles in *norma lateralis* have been observed slightly dorsal to the calculated center of mass for preserved head specimens, [91] however this is due to artifacts [90, 92]. MRI displays a 4% greater brain volume and weight resulting from the volume of blood in gray matter capillaries in the living brain, [90] which may cause the center of mass to be located directly over the occipital condyles. Maintenance of head balance is important as it seems that CFG emanates from the occipital condyles [93] which are along the central growth axis of the body and proximate the brainstem, around which the brain grows centripetally. Balance of the head would reduce any unnecessary metabolic demand required of the musculature for an upright posture; conservation of energy from an evolutionary standpoint.

The proposed CFG model is able to explain the developmental and functional observations of airorhynchy and the facial block hypothesis [12, 31]. The concept of brain temporal lobe growth displacing the ACB forward must deal with the asymmetric growth of the SOS, lack of brain temporal lobe/middle cranial fossa elongation, [11] and lack of a direct articulation between the MCB and maxilla due to separation by the infraorbital fissure. Additionally, current consensus seems to be that the nasal septum functions to support the roof of the nasal chamber rather than actively participate in the displacement of the palate itself by approximately 4 years of age [4, 13, 14, 94]. On the whole, the tissue(s) that displaces the maxilla downward with craniofacial growth postnatally have not yet been satisfactorily defined relative to the competing nasal septum theory and functional matrix theory [26].

Airway and Speech Development Through CFMAS Tension

Sagittal rotation of the cranium at the occipital condyles and asymmetric growth at the SOS are proposed to cause the face to rotate cephalad and ventral, opening the airway with normal jaw rotation and extension of tissues (lingual tonsil, velum) [95, 96] (Fig. **6**). The resultant relative descent of the larynx develops the hyolaryngeal complex, [12] creating a resonance chamber to allow voice production for speech.

Transverse development of the maxilla and nasal airway is believed to be a result of muscle mass increases resulting in greater CFMAS tension which causes the teeth to be compressed (keeping in mind the relative separation of the jaws with growth). Developed is a greater posterior relative to anterior transverse increase of the maxilla and nasal airway with growth (molar force is greater due to the Class 3 lever system of the jaws) [27]. The functional occlusion of the teeth displaces the maxillary halves, thereby increasing the transverse airway and providing room for the tongue. The tongue has been believed to play a major role in transverse airway enlargement, however the apposition observed on the bony palate during growth [13, 97] seems dismissive of a pressure large enough to displace the maxillary shelves. Also, the comparable posterior face height of individuals with hyperdivergent/leptoprosopic (simply, long faced) profiles to those with the hypodivergent/europrosopic (simply, short faced) profiles also raises questions of relative posterior tongue posture [98].

Lateral midfacial muscle attachments may also play a minor role in developing transverse nasal dimension. Craniofacial muscles are bilateral and, through lateral attachment relative to the midfacial bone centroids (center rotation), may cause lateral rotation below the centroid and medial rotation above the centroids. This may contribute to the inverted "V" shape [13] of the nasal aperture, in conjunction with occlusal forces transmitted across the palate and mid-palatal suture. This must be weighed against the reciprocal forces from the muscles of mastication.

SUPPORT FOR THE PROPOSED MODEL

The Effect of Cranial Rotation in the Expression of Facial form

Maxillomandibular rotation with craniofacial growth (CFG) had not been apparent until the Bjork implant studies of the mid-fifties [20]. Yet, as a whole, the interrelationship between the growth of the maxilla and mandible is still not fully understood, and remains one of the great challenges of craniofacial biologists [14].

lar resorption, post-surgical relapse, temporomandibular joint dynamics/growth, understanding of airway, future beneficial surgical procedures and age specific plasticity of tissues.

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