4 Facial and Palatal Growth

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4.1 Maxillary and Mandibular Growth Concepts

It is not the author's intent to write a definitive treatise on facial growth and its control processes because there are better sources for such information. However, because the history of cleft palate treatment has been influenced by what clinicians think is the correct facial growth process, it behooves the author to support or refute the various facial-palatal growth concepts based on his own clinical findings.

4.1.1 Newborn Palate with a Cleft of the Lip or Palate

Is bone missing, adequate, or in excess? What is the geometric palatal relation of the palatal segments at birth? With complete clefts of the lip and palate, are the palatal segments collapsed or expanded? Can the palatal segments be stimulated to develop to a larger size by neonatal orthopedic appliances? A number of studies have attempted to determine whether the cleft palate was deficient or adequate in osteogenic tissue; unfortunately, the investigators were limited by paucity of data, lack of homogeneity in their samples, and the hazards of estimating growth from cross-sectional data.

4.1.2 Genetic Control Theory: Craniofacial Growth is Entirely Predetermined

Enlow [1] writes that, in the past, it was thought that all bones having cartilage growth plates were regulated entirely and directly by the intrinsic genetic programming within the cartilage cells. Intramembranous bone (maxillary) growth, however, was believed to have a different source of control. This type of osteogenic process is particularly sensitive to biomechanical stresses and strains, and it responds to tensions and pressure by either bone deposition or resorption.

Tension, as traditionally believed, specifically induces bone formation. According to the traditional wisdom, when tension is placed on a bone, the bone grows locally in response. Pressure, on the other hand, if it exceeds a relatively sensitive threshold limit, specifically triggers resorption. According to this theory when muscle and overall body growth are complete, the bone attains biomechanical equilibrium, that is, the forces of the muscles are then in balance with the physical properties of the bone. This turns off osteoblastic activity, and skeletal growth ceases.

Unfortunately for traditional schools of thought, growth control in the human body is more complex than this. Moreover, it is now known that there is not a direct, one-to-one correlation between tension-deposition and pressure-resorption.

4.1.3 Functional Matrix Theory [2, 3] (Figs. 4.1, 4.2)

Enlow [1] goes on to explain that, with the development of the Functional Matrix Principle, a number of important hypotheses began to receive attention. One of these is that the "bone" does not regulate its own growth. The genetic and epigenetic determinants of skeletal developments are in the functional tissue matrix, that is, muscle, nerve, glands, teeth, neurocranial fossa, and nasal, orbital, oral, and pharyngeal cavities. This is primary while the growth of the skeletal unit is secondary. However, although the Functional Matrix Principle describes what happens during growth, it does not account for how it happens. Experiments have shown that mechanical forces are not the principal factor controlling bone growth.

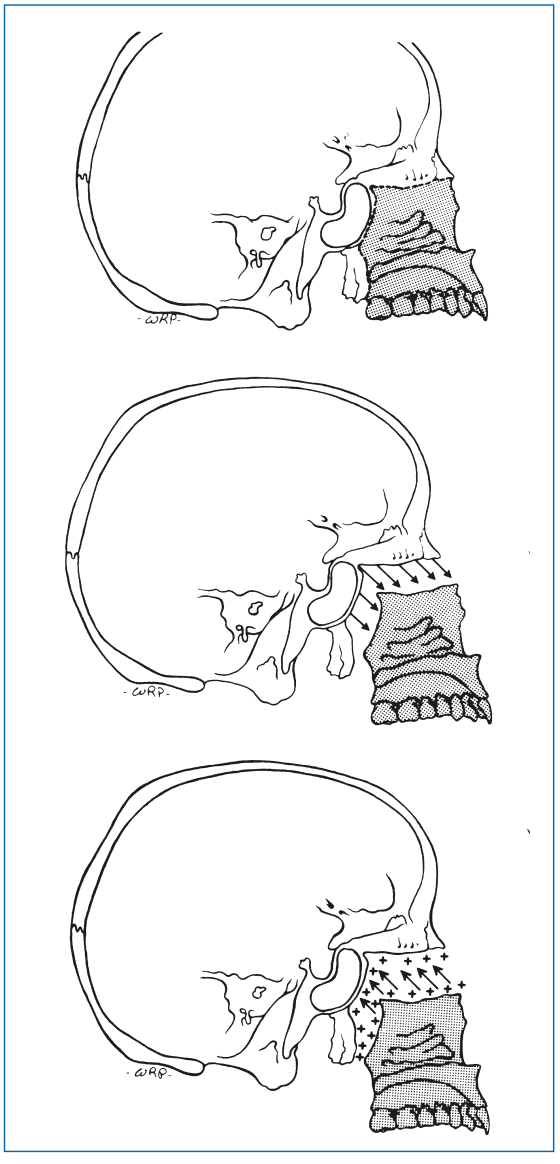


Fig. 4.1. The process of new bone deposition does not cause displacement by pushing against the articular contact surface of another bone. Rather, the bone is carried away by the expansive force of all the growing soft tissues surrounding it. As this takes place, new bone is added immediately onto the contact surface, and the two separate bones thereby remain in constant articular junction. The nasomaxillary complex, for example, is in contact with floor of the cranium (*top*). The whole maxillary region, in toto, is displaced downward and forward away from the cranium by the expansive growth of the soft tissues in the midfacial region (*center*). This then triggers new bone growth at the various sutural contact surfaces between the nasomaxillary composite and the cranial floor (*bottom*). Displacement thus proceeds downward and forward as growth by bone deposition simultaneously takes place in an opposite upward and backward direction (i.e., toward its contact with the cranial floor). (From [1])

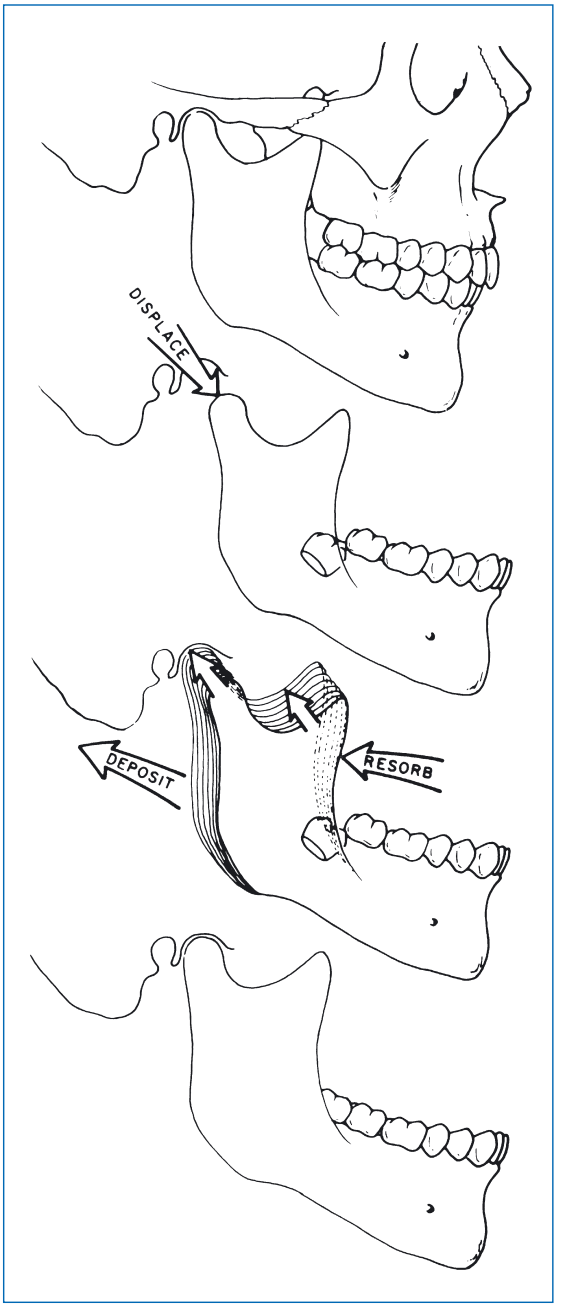


Fig. 4.2. Similarly, the whole mandible is displaced "away" from its articulation in each glenoid fossa by the growth enlargement of the composite of soft tissues in the growing face. As this occurs, the condyle and ramus grow upward and backward into the "space" created by the displacement process. Note that the ramus "remodels" as it relocates posterosuperiorly. It also becomes longer and wider to accommodate (1) the increasing mass of masticatory muscles inserted onto it, (2) the enlarged breadth of the pharyngeal space, and (3) the vertical lengthening of the nasomaxillary part of the growing face. (Reprinted with permission from [1])

Most researchers agree that a notable advance was made with the development of the Functional Matrix Principle introduced by Moss [2, 3]. It deals with what determines bone and cartilage growth in general. The concept states, in brief, that any given bone grows in response to functional relationships established by the sum of all the soft tissues operating in association with that bone. This means that the bone itself does not regulate the rate and direction of its own growth; the functional soft tissue matrix is the actual governing determinant of the skeletal growth process.

The course and extent of bone growth are secondarily dependent on the growth of pace-making soft tissues. Of course, the bone and any cartilage present are also involved in the operation of the functional matrix, because they give essential feedback information to the soft tissues. This causes the soft tissues to inhibit or accelerate the rate and amount of subsequent bone growth, depending on the status of the functional and mechanical equilibrium between the bone and its soft tissue matrix. The genetic determinants of the growth process reside wholly in the soft tissues and not in the hard part of the bone itself.

The Functional Matrix Concept is fundamental to an understanding of the overall process of bone growth control. This concept has had a great impact in the field of facial biology. The concept also comes into play as a source for the mechanical force that carries out the process of displacement. According to this now widely accepted explanation, the facial bones grow in a subordinate relationship with all the surrounding soft tissues. As the tissues continue to grow, the bones are passively (i.e., not of their own doing) carried along (displaced) with the soft tissues attached to the bones by Sharpey's fibers. Thus, for the nasomaxillary complex, the expansion of the facial muscle, the subcutaneous and submucosal connective tissues, the oral and nasal epithelia lining the spaces, the vessels, and the nerves, all combine to move the facial bones passively along with them as they grow. This continuously places each bone and all of its parts in correct anatomic positions to carry out its functions. Indeed, the functional factors are the very agents that cause the bone to develop into its definite shape and size and to occupy the location it does.

Growth control is determined by genetic influences and biomechanical forces, but the nature of the balance between them is still, at best, uncertain. No single agent is directly responsible for the master control of growth; the control process encompasses many factors. It involves a chain of regulatory links. Moreover, not all of the individual links are involved in all types of growth changes.

Enlow [1] identifies the maxillary tuberosity as being a major site of maxillary growth. It does not, however, provide for the growth of the whole maxilla, but rather is responsible for the lengthening of the maxillary arches. The whole maxilla is displaced in an anterior direction as it grows and lengthens posteriorly. However, the nature of the force that produces this forward movement is a subject of great controversy. The idea that additions of new bone on the posterior surface of the elongating maxillary tuberosity "push" the maxilla against the adjacent pterygoid plates has been abandoned.

Bones do not by themselves have the physiological capacity to push away bones. Another theory held that bone growth at the various maxillary sutures produces a pushing apart of the bones, with a resulting thrust of the whole maxilla downward and forward. This theory has also been rejected because bone tissue is not capable of growth in a field that requires the amount of compression needed to produce a pushing type of displacement. The sutural connective tissue is not adapted to a pressure-related growth process. It is believed that the stimulus for sutural bone growth is the tension produced by the displacement of the bone. Thus, the deposition of new bone is a response to displacement rather than the force that causes it. Although the "sutural push theory" is not tenable, Enlow reports that some students of the facial growth control processes are looking anew at growth mechanizing sutures, but not in the old conceptual way.

4.1.4 Cartilage-Directed Growth: Nasal Septum Theory [4–12]

Cartilages are the leading factor. Synchondrosis, nasal septum, and mandibular condyles are actual growth centers. Sutural growth is compensatory. This theory developed from criticisms of the "sutural theory." Scott [4, 5] believes that cartilage is specifically adapted to certain pressure-related growth sites, because it is a special tissue uniquely structured to provide the capacity for growth as a result of compression. The basis for this theory is that the pressure-accommodating expansion of the cartilage in the nasal septum is the source of the physical force that displaces the maxilla anteriorly and inferiorly. This, accordingly to Scott's hypothesis, sets up fields of tension in all the maxillary sutures. The bones then, while they enlarge at their sutures in response to the tension created by the displacement process, move in relation to each other.

The nasal septum hypothesis was soon adopted by many investigators in cleft palate centers around the world and became more or less the standard explanation, replacing the "sutural theory." Clinicians involved in cleft palate treatment, such as McNeil [13–15] and Burston [16–19] and their followers [20–34] accepted Scott's thesis that cartilage and

periosteum carry an intrinsic genetic message that guides their growth. They believed that the cartilaginous centers, such as the chondrocranium, the associated synchondroses, and the nasal septum, should be viewed as the true centers of skull and facial growth. Scott [4, 5] further suggests that the nasal septum plays more than a secondary role in the downward and forward vector of facial growth.

McNeil [13, 14], following Scott's thesis, describing the embryopathogenesis of complete clefts of the lip and palate and their treatment at the neonatal period, wrote that the palatal processes, being detached from the growing nasal septum, do not receive their growth impetus and, therefore, are not only retruded within the cranium but are also deficient in osteogenic tissue. He goes still further and believes that the deficient palatal processes can be stimulated to increased size through the use of functional orthopedics.

4.1.4.1 Stimulation of Bone Growth – Is it Possible?

As McNeil saw it, pressure forces created by "functional" orthopedic appliances, which are within the limits of tolerance, will act to stimulate bone growth in an anterior direction. This force needs to be applied to particular regions and in particular directions so that it can intensify normal forces. The resulting narrowing of the cleft is due to growth of the underlying bone brought on by such stimulating appliances. Additional growth leads to a reduction in the soft palate cleft as well, thereby increasing the chance of having a long, flexible, well-functioning soft palate after surgical closure.

McNeil [14] goes on to suggest that an obturator alone is unsatisfactory because it will reduce "valuable" tongue space and lead to harmful speech habits. McNeil was correct in stressing that surgery should be reduced to a minimum compatible with sound clinical reasoning and accepted surgical principles.

Whereas McNeil states that his procedure stimulates palatal growth, thereby narrowing the cleft space, Berkowitz's [35] 3D palatal growth studies – using a sample of cases that have not had neonatal maxillary orthopedic treatment and a control sample of noncleft cases – show that growth occurs spontaneously. This is an expression of the palate's inherent growth potential, which can vary among patients. Berkowitz concluded that "catch-up growth" can occur after palatal surgery (with minimum scarring) is performed (see Chap. 16).

4.1.4.2 The Need to Prevent Collapse

McNeil [13–15] further believes that the palatal segments should be manipulated to an ideal relationship prior to lip surgery to prevent them from moving too far medially and becoming collapsed with the buccal segments in crossbite. This, he suspects, will lead to abnormal movements of the tongue and give rise to faulty respiratory, sucking, and swallowing patterns, also causing abnormal growth and development of the palatal structures.

Mestre et al. [36], studying palatal size in a cleft population that had not been operated on, report that the development of the maxilla appears to be normal in unoperated cases. They do conclude that it is the type, quality, and extent of the surgery that determines the effect on maxillary growth and that osteogenic deficiency does exist to varying degrees. Our research on serial palatal growth changes supports this conclusion that palates with clefts are highly variable in size, shape, and osteogenic deficiency.

Unfortunately, McNeil's interpretation of the effects of clefting on the various vegetative functions, and in reducing palatal growth, has not been supported by controlled objective research. The inability of the manipulated arch to remain intact after lip surgery, and not move medially into a collapsed relationship, has led many clinicians to question the accuracy of McNeil's other stated benefits such as reduction of middle ear infections.

McNeil [13–15] made other faulty observations. Among them:

1. He mistakenly believed that the orthopedic appliance will stimulate the underdeveloped cleft segment in unilateral clefts of the lip and palate (UCLP) to move forward, to make contact with the premaxillary portion of the greater segment and both palatal segments in bilateral clefts of the lip and palate (BCLP), after the lip is united. Even as early as the 1960s, many orthodontists found the opposite to be true. In UCLP, the premaxillary portion of the larger segment moves medially and backward to make contact with the lesser segment due to the action of compressive lip muscle forces. If McNeil had had the benefit of serial casts, his interpretation of clinical events would, I am confident, have been totally different.
2. McNeil's claim that the lesser segments in UCLP, and both segments in BCLP, can be stimulated to grow forward is totally erroneous. His conclusions were based on conjecture, not on objective data. The results of Berkowitz's 3D palatal growth studies [37] show marked acceleration in palatal growth during the first 2 years without orthopedic treatment, with most of the growth changes occurring at the area of the maxillary tuberosity and not

at the anterior portion of the palate except for alveolar growth associated with canine development (Fig. 4.2). Movement of the cleft palatal segment anteriorly is only possible as a result of reactive mechanical forces being applied through the use of pinned maxillary orthopedic appliances or from a protraction facial mask.

One last but significant characterization of a newborn cleft of the lip and palate needs to be refuted. McNeil states that "in BCLP lateral segments are collapsed toward the midline before birth." However, he does not explain the dynamics that can make this possible. How can segments be collapsed if there are no inwardly directed forces from the cleft lip-cheek muscle complex, especially when the tongue fits within the cleft space and acts to move the palatal segments apart?

Enlow's [1] report on current thinking on palatal growth processes delivers McNeil's thesis a mortal blow. Enlow[1] writes that recent research has shown that pressure is detrimental to bone growth.

Bone is necessarily both a traction- and pressure-adapted kind of tissue. The periosteal membranes are constructed to function in a field of tension (as by the pull of a muscle). Covering membranes are quite sensitive to direct compression because any undue amount causes vascular occlusion and interference with osteoblastic formation of new bone. Osteoclasts, conversely, function to "relieve" the degree of pressure by removing bone. Bone is pressure sensitive and high-level pressure induces resorption.

Moss et al. [38], responding to the role of nasal septal cartilage in mid-facial growth as put forth by Scott [4, 12], states that Scott's hypothesis is based on the following assumptions: (1) that in the fetal skull, the original nasal capsule and its derivatives are cartilaginous; (2) that all cranial cartilaginous tissues (septal, condylar, or in synchondroses) are primary growth centers, by virtue of the undoubted ability of all cartilaginous tissues to undergo interstitial expansive growth; and (3) that following the prenatal appearance of the intramembranous vomer (and of the several endochondral ossification centers of the ethmoid sinuses and the turbinates) the remaining unossified portions of the cartilaginous nasal capsule continue to be capable of such interstitial expansion. Moss further suggests that the nasal septal cartilage grows as a secondary, compensatory response to the primary growth of related oro-facial matrices and that mid-facial skeletal growth is not dependent on any prior, or primary, growth "impetus" of the nasal septal cartilages.

In Scott's hypothesis, it is assumed that cartilaginous interstitial growth is the major source of the expansive force that "pushes" on the subjacent mid-facial skeletal structures, causing both vertical and anteroposterior growth. Moss believes that it has been demonstrated repeatedly that growth in size and shape, as well as the changes in spatial position, of all skeletal units is always secondary to primary changes in their functional matrices. This secondary skeletal unit growth comes about in the following manner. All cranial bones and cartilages originate and grow within soft tissue capsules. The splanchnocranial skeleton exists within an oro-facial capsule. The primary growth of the enclosed oro-facial matrices causes the oro-facial capsule to expand responsively. Because the splanchnocranial bones are within this capsule, they are passively translated in space within their expanding capsule. As a result of such spatial displacement, the individual bones will be distracted (or separated) passively from one another.

The increments of growth observed at the sutural edges of these bones, and at the mandibular condylar cartilages, are secondary, compensatory, and mechanically obligatory responses of the skeletal units to such separative movements (i.e., the alterations of size and shape in bones and cartilages are responses to matrix growth, not the cause of it).

The nasal skeleton is characterized by a relatively great normal variation in form. The nasal capsule (and septum), from its inception, serves to protect and support the functional spaces for respiration and olfaction. In human, the olfactory spaces are fully formed at birth. Postnatal cavity growth exclusively increases the respiratory functioning space.

The growth of the upper face is, in part, a response to the functional demands for increased respiratory volume. The nasal cavity is not a space haphazardly left over after the upper facial structures complete their growth. On the contrary, the expansion of the nasal cavity is the primary morphogenetic event; and nasal capsular growth, both osseous and cartilaginous, is secondary. The application of the theory of functional cranial analysis to nasal and mid-facial skeletal growth demonstrates that the growth of each of these two areas is independent of the other and that the nasal septal cartilage plays a secondary compensatory role, rather than a primary morphogenetic one.

At present, the nasal septum theory is somewhat accepted as a reasonable explanation by a number of clinicians who favor presurgical orthopedic treatment, although it is universally realized that much more needs to be understood about facial growth processes [39]. (The use of presurgical orthopedic treatment is covered in greater detail in Chaps. 18–22.)

Clinically, there seems to be more support for the functional matrix theory than the nasal septum theory. Unfortunately, McNeil, in espousing Scott's theory to explain the "retropositioned maxillary complex rel-

ative to the mandible and osteogenically deficient palatal processes" in complete clefts of the lip and palate, did not have access to serial palatal and facial growth records to support such a view. However, Berkowitz's [40] serial casts study of CUCLP and CBCLP cases using the Angle occlusal classification system, which is the most reliable means of judging the geometric relationship of the maxillary to the mandibular arches within the face, showed that at 3–6 years of age, the teeth in the lateral palatal segments were in either a Class I or Class II relationship but were never in a Class III relationship.

On this basis, one can conclude that it is not the lack of a growth impetus from the nasal septum that explains the presence of a small cleft palatal segment at birth. If palatal osteogenic deficiency does exist, it can more accurately be explained in relationship to the embryopathogenesis of facial development: the failure of migrating undifferentiated mesenchymal cells from the neural crest to reach the facial processes [41, 42] (see Chap. 1).

4.1.5 Basion Horizontal Concept: The Direction of Facial Growth (Figs. 4.3–4.5) [43]

No discussion on craniofacial growth is complete without including Coben's Basion Horizontal Concept of the direction of facial growth. Basion Horizontal is a concept based on a plane at the level of the anterior border of foramen magnum parallel to Frankfort horizontal where Basion is the point of reference for the analysis of craniofacial growth. Coben states that the growth concept which Basion Horizontal represents is that craniofacial growth is reflected away from the foramen magnum (Basion) and the vertebral column. The cranio-maxillary complex housing the maxillary dentition is translated upward and forward from Basion by growth of the cranial base. Growth of the mandible is reflected away from Basion, carrying the mandibular dentition downward and forward. The divergence of the two general vectors develops space for vertical facial growth and the eruption of the dentition.

Normal maxillo-mandibular development requires synchronization of the amount, timing, and direction of growth of the cranio-maxillary complex and of the mandible. The cranial base vector represents the upward and forward translation of the upper face by growth of the spheno-occipital synchondrosis, while growth of the spheno-ethmoidal/circumaxillary suture system and the nasal septum increases the depth and height of the upper face.

The Basion–Articulare dimension is essentially stable postnatally, indicating that the mandible maintains a constant sagittal spatial relation to the foramen magnum as the reflection of mandibular growth carries the lower teeth downward and forward, away from the cranial base.

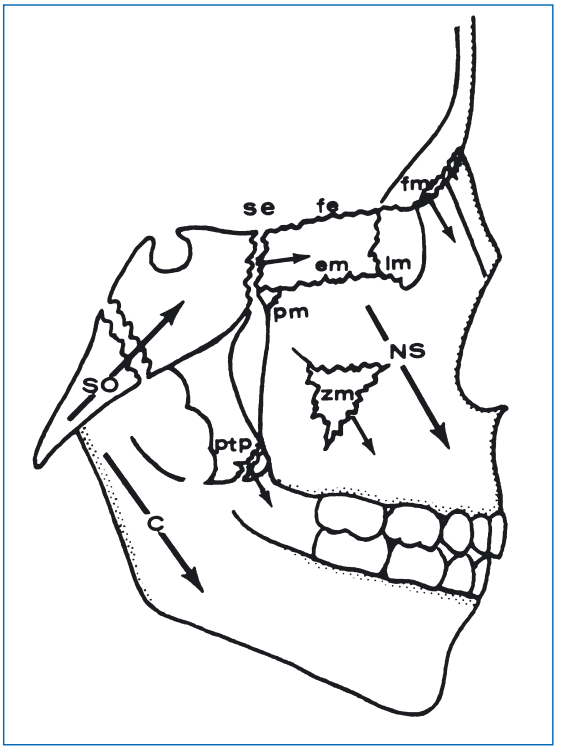


Fig. 4.3. Postnatal craniofacial growth systems to the age of 7 years (first decade). Cartilaginous growth: *SO*, Spheno-occipital synchondrosis; *C*, reflection of condylar mandibular growth; *NS*, nasal septum. Spheno-ethmoidal circumaxillary suture system: *se*, Spheno-ethmoidal; *ptp*, pterygopalatine; *pm*, palatomaxillary; *fe*, fronto-ethmoidal; *em*, ethmoidal-maxillary; *lm*, lacrimal-maxillary; *fm*, frontomaxillary; *zm*, zygomaticomaxillary; *zt*, zygomaticotemporal (not shown). Surface apposition-modeling resorption development (*stippled area*): minor contribution. (Reprinted from [43])

There are two distinct phases of craniofacial growth because of a change in the system of upper facial development after the approximate age of 7 years. Before age 7, growth of the upper face is dominated by the nasal septum, the eyeballs, and the spheno-ethmoidal/circumaxillary suture system (Fig. 4.4). At this age, the growth in this suture system produces space for the eruption of the maxillary first molars. Longitudinal cephalometric findings of a continuous increase in the Sella–Frontale dimension with little increase in the thickness of the frontal bone before age 7 support the concept that bone apposition and remodeling resorption are minor factors in these early years.

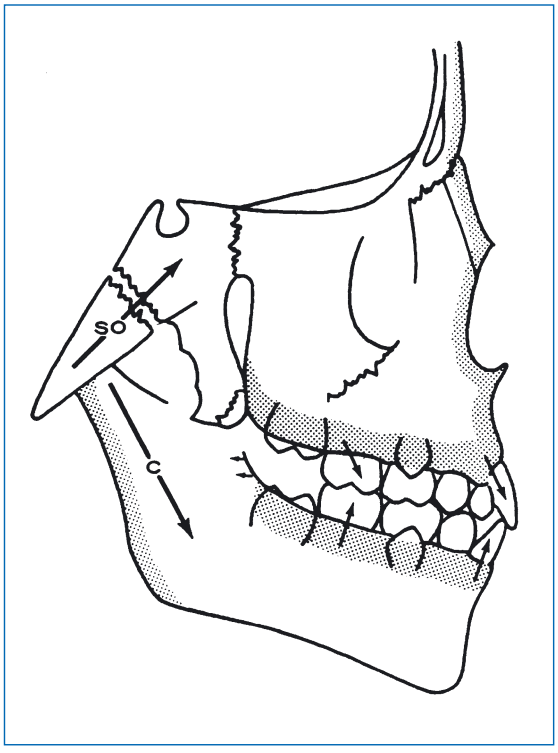
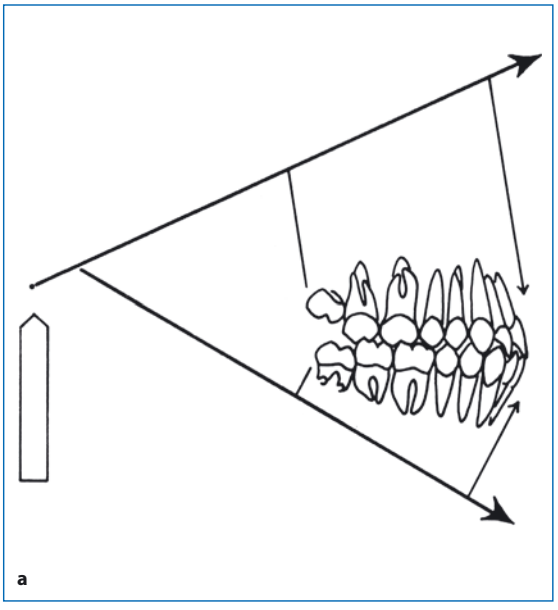


Fig. 4.4. Postnatal craniofacial growth systems from age 7 years (second decade). Cartilaginous growth: *SO*, Spheno-occipital synchondrosis-active through puberty; *C*, reflection of condylar mandibular growth – active to facial maturity; nasal septum – growth completed. Spheno-ethmoidal circumaxillary suture system: Sutural growth no longer primary system of upper facial development. Surface apposition-modeling resorption development (*stippled area*): Now major method of upper facial development and alveolar growth [43]



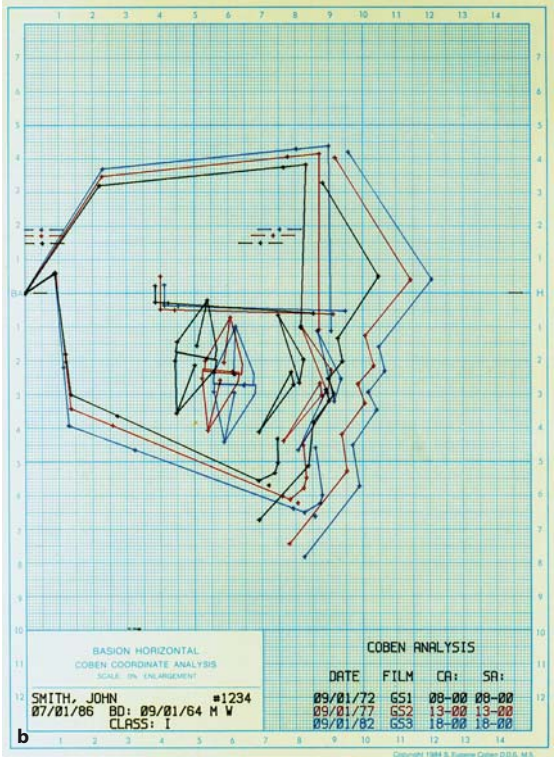


Fig. 4.5. **a** Basion Horizontal. General vectors of craniofacial growth. Growth of the cranial base translates the upper face and the maxillary dentition upward and forward away from the foramen magnum. Growth of the mandible translates the lower dentition downward and forward. The two diverging vectors create space for vertical facial development and tooth eruption. [43] **b** Basion Horizontal. Basion Horizontal Coordinate computer Craniofacial serial schematic line graph of Fig. 4.5a

At about age 7, the growth system of the upper face changes with the closure of the spheno-ethmoidal suture. The Sella–Frontale dimension stabilizes, and the thickness of the frontal bone begins to increase by surface apposition and remodeling until maturity. The interpretation is that after age 7, the initial primary system of spheno-ethmoidal/circumaxillary sutural growth of the upper face is replaced by surface apposition and remodeling resorption (Fig. 4.4). It is significant that, before age 7, space for the erupting upper first molars results from growth of the spheno-ethmoidal/circumaxillary suture system. After age 7, space for the upper second and third molars is produced by maxillary alveolar apposition as the maxillary dentition erupts downward and forward. This concept was supported by Scott [12], who reasoned that the spheno-ethmoidal suture must be viewed as

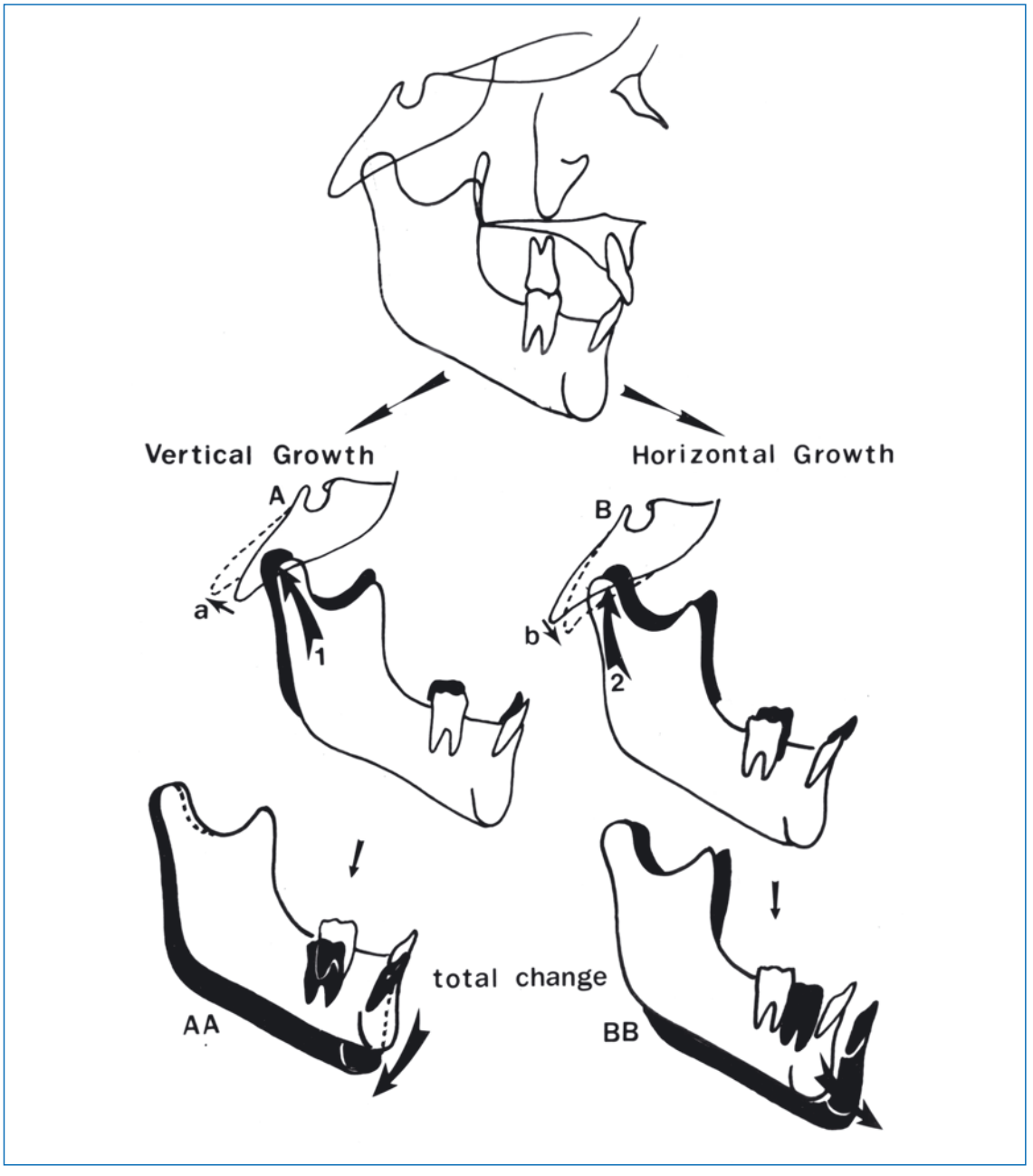


Fig. 4.6. Various growth changes that occur, the condylar head determine the direction and extend of mandibular growth

part of the major circumaxillary suture system, and that once part of the suture closes, there is no further growth in that suture system. Longitudinal cephalometric growth studies confirm this interpretation (Fig. 4.5).

4.2 Mandibular Development in Cleft Palate (Figs. 4.6, 4.7)

Recent studies have revealed a series of often subtle differences in the morphology of the mandible in persons with cleft lip and/or palate. Dahl [44] and Chierici and associates [45] found that, in persons with

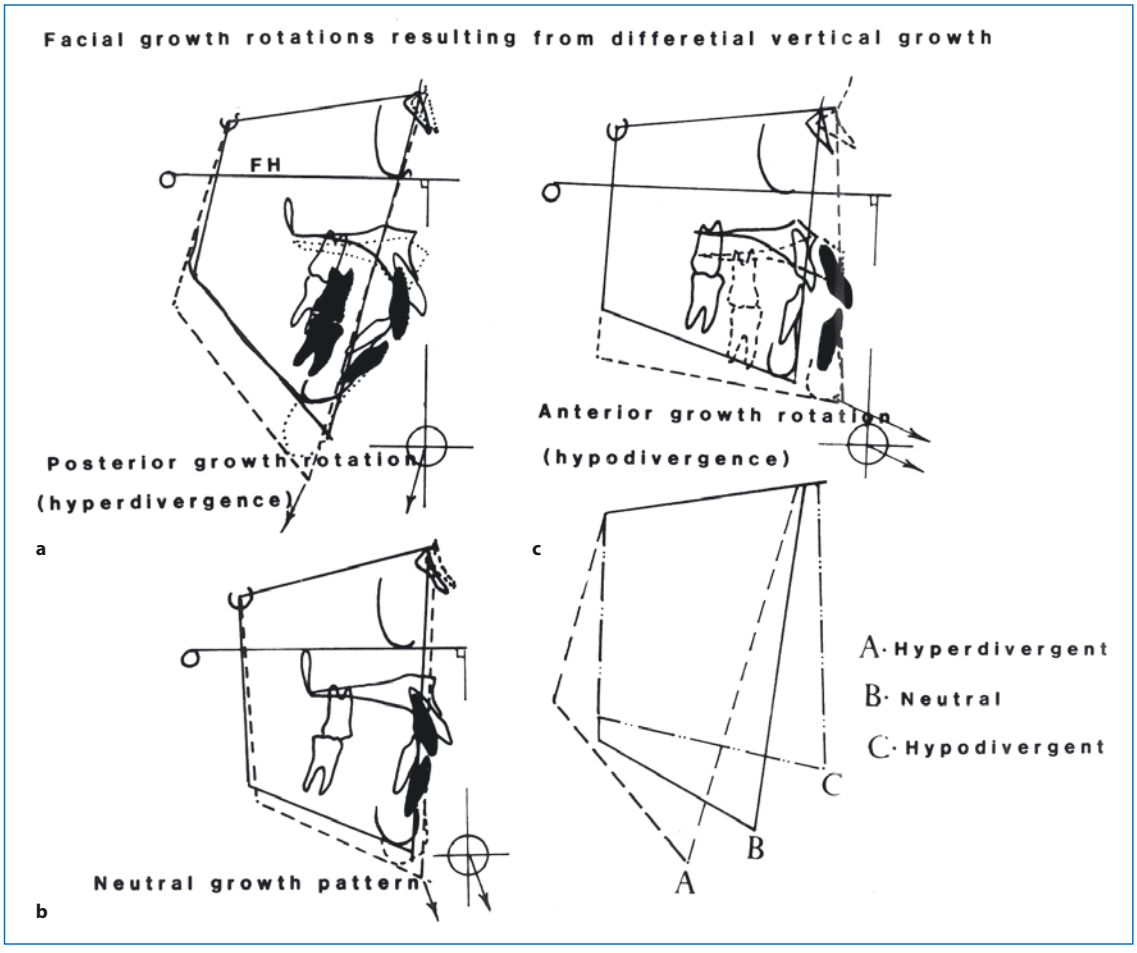


Fig. 4.7 a–c. Facial growth rotations resulting form differential vertical growth. **a** Hyperdivergent pattern with posterior growth rotation. **b** Neutral growth pattern. **c** Hypodivergent growth pattern with anterior growth rotation.*Comment: This series is not a true reflection of the growth of various components of the face. See Coben's Basion Horizontal, Coordinate Craniofacial Analysis system for this (Fig. 4.5)*

clefts of the hard palate only, the mandibular plane was steeper and the gonial angle more obtuse than in a normal population. Mazaheri and coauthors [46] noted that the length and width of the mandible were significantly less in persons with cleft palate only than in those with cleft lip and palate (CLP) and normal groups. Aduss [47] observed that the mandibular gonial angle in patients with unilateral CLP was more obtuse, and that the anterior cranial base appeared to be elevated. Rosenstein [48] also found the mandibles to be smaller, with steeper mandibular plane angles. Bishara [49] studied Danish children with repaired cleft palates only. In that study, and again in a later study of patients with CUCLP [50], he noted that the mandible was significantly more posterior in relation to the cranial base and that its mandibular plane was steeper than normal.

Krogman and colleagues [51] found no difference in mandibular dimensions in the BCLP population, other than a more obtuse gonial angle. They also found the temporomandibular joint to be positioned farther back, so that its effective length was less than in the normal population. Robertson and Fish [52], comparing mandibular arch dimensions, found no significant differences between normal and cleft children either at birth or at 3 years of age.

4.3 Patterns of Postnatal Growth

Based on the serial studies, three general patterns of postnatal growth have been demonstrated. In the Pierre Robin sequence, and in complete bilateral clefts of the lip and palate, most cases demonstrate substan-

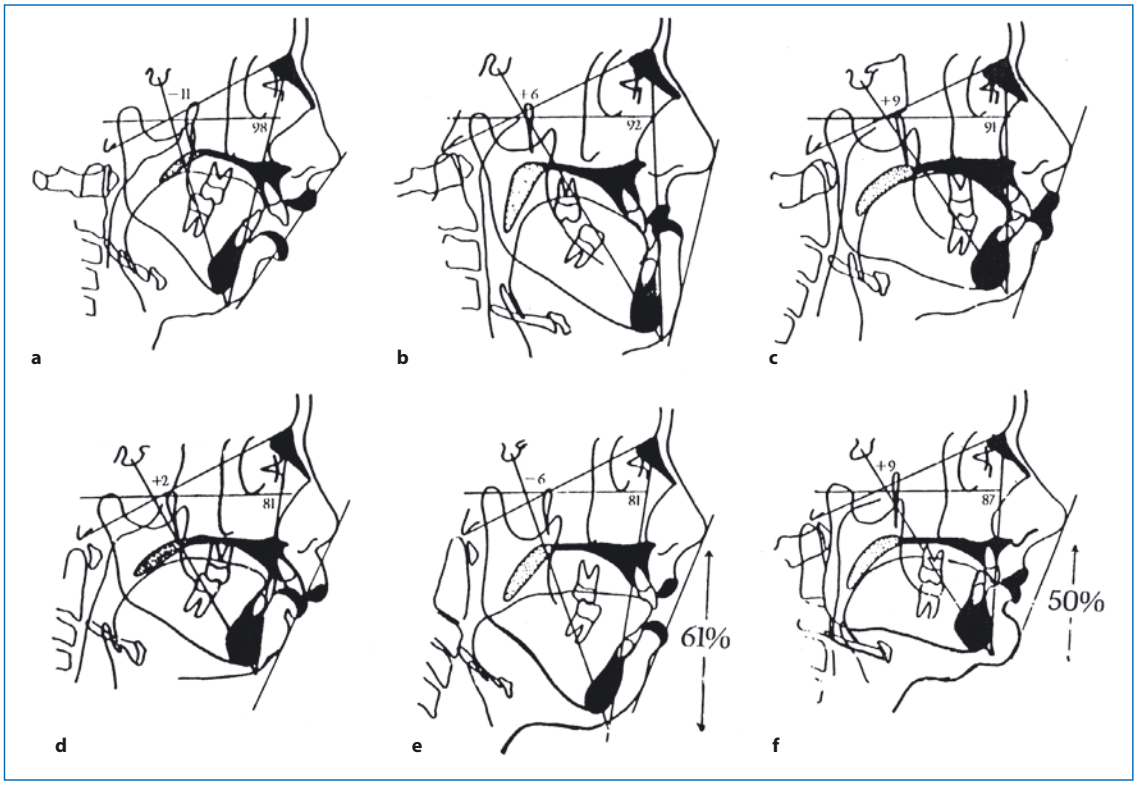


Fig. 4.8 a–f. Not all faces are the same, therefore treatment most vary according to the facial growth pattern. Various types of facial patterns. **a** Retrognathic mandible with steep mandibular plane angle. Severe overbite and overjet. Chronic mouth breather. **b** Prognathic mandible with recessive maxilla. **c** Brachyfacial type with dental protrusion. **d** Slightly retrognathic type with protrusive maxillary denture and severe deep bite. **e** Long shallow face with severe tongue problem, extremely wide openbite, and an inability to close the lips. **f** Extremely closed bite with short denture height. (Courtesy of R. Ricketts. The Biology of Occlusion and the Temporomandibular Joint in Modern Man, 1972)

tial improvement through "catch-up" in the growth of the mandible. In the second pattern, mandibulofacial dysostosis, the pattern of growth is such that the deformity observed in infancy or early childhood is maintained throughout the growth period. The deformity of the mandible neither improves nor worsens in the course of time. The third pattern is one in which the growth process is so deranged that the severity of the deformity increases with age. This has been observed in some instances of unilateral agenesis of the mandibular ramus (e.g., hemifacial microsomia) and in the growth of the maxilla and neurocranium in some forms of premature craniofacial synostosis.

4.3.1 Bone Remodeling During Growth (Fig. 4.8)

Enlow [1] states that remodeling is a basic part of the growth process. The reason why a bone must remodel during growth is because its regional parts become moved; "drift" moves each part from one location to another as the whole bone enlarges. This calls for sequential remodeling changes in the shape and size of each region. The ramus, for example, moves progressively posteriorly by a combination of deposition and resorption. As it does so, the anterior part of the ramus becomes remodeled into a new addition for the mandibular corpus. This produces a growth elongation of the corpus. This progressive, sequential movement of component parts as a bone enlarges is termed relocation. Relocation is the basis for remodeling. The whole ramus is thus relocated posteriorly, and the posterior part of the lengthening corpus becomes relocated into the area previously occupied by the ra-

mus. Structural remodeling from what used to be part of the ramus into what then becomes a new part of the corpus takes place. The corpus grows longer as a result.

4.3.2 Maxillary Growth

The maxilla grows downward and forward from the cranial base with growth occurring at the articulations with other bones (i.e., the sutures). Björk [53] stated that during growth the maxilla is displaced in a rotational manner relative to the cranial base; however, this rotational aspect is small, which results in the downward and forward effect. Furthermore, he emphasized that there is little variation in the upper facial height between groups. Therefore, because of the small variation, it is likely that individual difference in facial form results from growth in other facial areas where there is more variation.

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