The anatomical studies of Veau laid the foundation for our present understanding of cleft lip and palate (Veau, 1926, 1931, 1935). His embryologic research into the pathogenesis of cleft lip and palate in the human is a classic study (Veau and Politzer, 1936). Veau recognized that unilateral and bilateral cleft conditions showed a characteristic skeletal deformity that was consistently uniform in each cleft type, and he regarded the skeletal deformity as a product of abnormal growth subsequent to the initial embryonic failure of palate formation. It was his opinion that the protrusion of the anterior premaxillary segment seen in the unilateral and bilateral conditions was caused by uninhibited growth of the bony part of the septal stem, consisting of the vomer and premaxillary bones. The premaxillovomeral suture was regarded as having growth potential similar to that of long bone epiphyseal plates. Consideration of numerous details of the anatomical analysis presented in the chapter may render untenable the hypothesis of growth thrust from the bony septal stem. A synthesis of the available material, in keeping with present concepts of growth and development, is proposed to which scrutiny is invited, both to test the facts and to prompt appraisal of treatment principles.

In man, except for a brief period in the embryo, the premaxilla does not exist as a separate entity. The term has been retained with reference to cleft lip and palate conditions because of its descriptive convenience and homology with experimental animals. In man, the term premaxilla is used to define that part of the upper jaw anterior to the incisive suture and the canine teeth. A cleft of the primary palate is usually thought to divide the premaxillary bone from that of the maxilla.

**Bilateral Cleft Lip and Palate**

**Premaxillary Malformation.** The complete bilateral cleft at birth shows a distinct premaxillary malformation characterized by a protrusion of the entire premaxillary bone with respect to the cartilaginous nasal septum and a protrusion of the tooth-bearing alveolar process. The protrusive premaxillary bone obliterates the columellar area of the nose so that the lip attaches directly to the nasal tip. The total protrusion seen clinically is the summation of three factors: the abnormal forward position of the premaxillary alveolar bone; an abnormally advanced position of the premaxillary basal bone; and possible underdevelopment of the maxillary segments as a whole, including some degree of anteriorly localized hypoplasia at the site of the canine tooth.

Alveolar bone supports or contains the incisor teeth, while the basal bone has a skeletal function. The basal bone articulates with the cartilaginous septum superiorly and the vomer posteriorly. It is normally continuous with the body of the maxilla laterally. In normal structure the alveolar process is directly inferior to the nasal bone, but in the bilateral cleft condition the alveolar bone is anterior to the basal bone in horizontal arrangement. The basal bone and the anterior nasal spine normally lie posterior to the nasal septum's anteroinferior
point, whereas the basal bone of the bilateral cleft premaxillae is anteriorly advanced and adapted around this septal point, and the anterior nasal spine ascends the anterior septal border. The contributions of the alveolar and basal protrusions to the overall premaxillary protrusion appear to be approximately equal.

In profile view, the primary central incisor teeth lie anterior to the cartilaginous nasal septum. Part of the profile of the nasal septum may be seen in a lateral radiograph of the premaxillary segment, where it corresponds to the posterior slope of the anterior nasal spine. The incisors are not rotated forward and upward, as might be thought, but have a relatively normal vertical orientation. They are supported by a thin protruding alveolar process, which commences at the level of the anterior nasal spine, passes forward over the developing incisor roots, inferior to the medial crura of the alar cartilages, and turns inferiorly for a short distance in relation to the labial surface of the teeth.

**Bilateral Cleft: The Lip and Columella.** The normal form of the upper lip, in particular the philtrum, philtrocolumellar angle, and Cupid’s bow, is determined mainly by the underlying musculature. Labial muscle fibers insert densely, into the skin lateral to the philtrum, which, not receiving such support, presents as the median philtral dimple. The inferior border of the orbicularis oris muscle inserts closely along the vermilion border and, together with the other labial muscles, appears to give rise to the tubercle which is inferior to the philtrum. The labial musculature inserts thickly into the skin at the base of the columella and on the nostril floors, attaching this skin to the underlying bone. This anatomical arrangement is clearly a main factor in the development of the philtrocolumellar angle.

The medial part of the bilaterally cleft upper lip is conspicuously everted. This gives the erroneous impression that the premaxillary segment is rotated anterosuperiorly on the nasal septum. The eversion of the lip and, to some extent, the hypoplasia of the columellar skin appear to be caused by the premaxillary protrusion. However, it is also possible that the tethered lip induces forward growth of the alveolar process into a protrusive position. Other important factors contributing to the malformation must also be considered. The medial lip moiety contains no muscular tissue (Vean, 1926; Latham, 1973). It is therefore grossly deficient in bulk and lacks features of form normally produced by muscle (see Chapter 41). Extrinsic factors such as the tongue, mandible, and lower lip play a variable role.

The columella may be clinically absent, but it is not anatomically absent. By definition the columella is the fleshy external termination of the septum of the nose, supported by the medial crura of the alar cartilages and covered by skin. A preliminary study of the reconstructed nasopremaxillary region of a full term infant with bilateral cleft lip and palate showed that the medial crura of the alar cartilages occupied a normal position in relation to the nose and the cartilaginous nasal septum and were of relatively normal proportions (Latham and Workman, 1974). They were almost totally obscured by the protruding alveolar process, and the columellar skin was correspondingly hypoplastic. Unfortunately, rapid retraction of the premaxillary bones does not serve to uncover the medial crura, because columellar skin is not available to cover them, and the result is a depression of the nasal tip.

**Bilateral Cleft: Nasal Septum, Premaxillae, and Vomer.** In the bilateral cleft condition the inferior border of the cartilaginous nasal septum is reinforced by bone which provides a stemlike support for the premaxillary segment. This stem consists mainly of the
vomer, its anterior part being formed by the premaxillae. The premaxillovomeral joint is located at a point about one-third of the septal length posterior to the premaxillary alveolar process. The premaxillary segment consists of paired premaxillary bones jointed in the midline by the interpremaxillary suture, which represents the anterior third of the normal midpalatal suture. Posteriorly the premaxillary stem consists of paired processes joined by the suture and termed the infravomerine processes of the premaxillae. These overlap the single vomer, whose tapering anterior edge adapts closely to the nasal septum. The premaxillovomerine joint is, therefore, of a tongue-and-groove type, the vomer being the tongue and the oblique groove being formed by the infravomerine processes of the premaxillae.

The vomer adapts to the inferior border of the cartilaginous nasal septum and articulates posteriorly with the sphenoid bone. In cross section the vomer of prenatal specimens is "U"-shaped, but after birth resorption occurs on its lateral surfaces to give a thin "V"-shaped cross section with a notable edge inferiorly.

A slight swelling frequently occurs on the inferior border of the septum just posterior to the alveolar process at a position corresponding with the location of the premaxillovomer suture. The most likely reason for this is the presence of the paraseptal cartilages, bilateral fin like structures which articulate with the inferior border of the septal cartilage and diverge inferiorly in lateral relation to the vomer. The premaxillovomer stem, in the vicinity of the suture, is flanked for a short distance by the paraseptal cartilage plates. It is improbable that the premaxillovomer suture itself produces the swelling.

**Bilateral Cleft: Maxillary Segments.** The gum pads of the maxillary segments of the infant with bilateral cleft are covered with gingival mucosa. They are demarcated from the palatal mucosa on their medial aspect by a groove corresponding with the position of the palatal alveolar process, to which the oral epithelium has fibrous connections. The developing teeth are situated lateral to this groove; the area medial to it corresponds to the horizontal process of the maxilla and palatine bones, which are covered by the thick palatal mucosa.

The shape and size of the maxillary palatal processes occasionally show evidence of intrauterine molding by the tongue. The horizontal process of the palatine bone is deflected superiorly, suggesting that the embryonic palatal processes actually reached a horizontal orientation and that inferior pressure from the tongue then molded the processes superiorly. Despite the presence of the cleft and severed relations with the nasal septum and vomer, the growth pattern of the palatal bone appears to be unaffected in that bone resorption occurs on the nasal aspect and bone formation on the oral aspect. It is the usual finding that the palatal mucosa has been molded or deflected superiorly, and the level of the horizontal process of the maxilla (palatal bone) tends to lie superior to the inferior border of the nasal septum.

The arch form of the maxillary segments generally appears normal soon after birth. However, the position of the maxillary segments is subject to intrauterine molding, particularly by the tongue, so that at birth they may be asymmetrical. The tongue may have been wedged superiorly into one nasal cavity or the other, deflecting the septum a little and displacing a maxillary segment considerably in a lateral direction, so as to enlarge that nasal cavity. After birth both maxillary segments tend to collapse medially.
Development of Bilateral Cleft Deformity. Human embryos with bilateral clefts of the primary palate have been illustrated by Kraus, Kitamura, and Latham (1966); two embryos aged 41 and 43 days, showed no sign of premaxillary protrusion. However, in a 47 day old specimen, protrusion of the premaxilla was beginning to appear, and it was conspicuous in a 9-week specimen. Veau and Politzer (1936) illustrated the palatal view of Hochstetter's 41 day old specimen (23 mm CR), which showed protrusion of the premaxillary segment. Stark (1954) illustrated a well-preserved embryo of about 8.5 weeks ovulation age (46 mm CR) with bilateral clefts which showed advanced skeletal deformity. A recently acquired 13 week old specimen demonstrated extreme premaxillo-maxillary malrelation.

From these observations it appears that the premaxillary protrusion of the bilateral condition arises after original cleft formation in the embryo. The primary palate is normally formed by 35 days, and clefts would be apparent by that time. In a matter of days palatal malrelationships would begin to show, subsequently developing rapidly to reach, at 10 weeks, proportions comparable to those seen after birth.

The deformity seen in the 13-week fetus represents the failure and abnormal activity of embryonic growth mechanisms. The maxillary segments lose some of their normal forward displacement because of their isolation from the nasal septum. The premaxillary bones are held at the anteroinferior point of the nasal septum by the septopremaxillary ligament (Latham, 1971, 1973), so that protrusion of the basal premaxillary bone is fully established in the 13-week fetus.

In older fetuses the gradual forward growth of the labial alveolar process as teeth develop has been observed. In a sagittal section through the premaxillary segment of a 17-week fetus, the labial alveolar process shows a little convexity anteriorly, indicative of its early forward growth pattern. The position of the premaxillary segment at the anteroinferior point of the cartilaginous nasal septum is also clearly demonstrated. The septopremaxillary ligament, arising from the anterior septal border, inserts onto the anterior nasal spine and into the interpreamaxillary suture. The anterior nasal spine develops in a superior direction under the influence of its ligamentous attachment. Eversion of the lip is already established and may be attributed, in part, to the protruded position of its mucosal attachment to the labial alveolar bone. This section also demonstrates the absence of muscle in the lip.

The protrusion of the basal premaxillary bone is evidently established by the age of about 10 weeks in utero. The dentoalveolar protrusion is a slowly progressing feature over a period of about 7 months in utero and continues for some months after birth until the crowns of the primary incisor teeth reach a mature size.

Cause of Premaxillary Protrusion. It was Veau's opinion that the premaxillary segment was driven forward by excessive growth of the bony premaxillary stem (Veau, 1934). However, his concept of a forward growth force generated within the developing vomeropremaxillary stem is no longer in keeping with concepts of facial growth and bone maturation. Bone cannot grow interstitially, so the presumed growth force would have to be a function of the premaxillolovomerine suture. Since this suture still has not formed by the tenth week of fetal life, when protrusion of the premaxillary basal bone is almost complete, it is clear that another explanation must be sought.
The evidence indicates that the septopremaxillary ligament plays a key role. Normally, premaxillary bone is kept in place by its early fusion with the maxilla to form one bone, and the developing dental arch is controlled by continuity of the mucogingival arch. The alveolar process develops inferiorly from the basal bone in relation to the developing teeth, which in turn are affixed to the gingiva by their fibrous follicles. The lip also influences dentoalveolar form.

The complete cleft condition never develops continuity of bony, gingival, or labial structure between the premaxillary and maxillary regions, so that the developing premaxillary segment is under no lateral restraint from any of these structures. Consequently, its attachment to the nasal septum by the septopremaxillary ligament becomes a dominant factor. Commencing as early as the sixth week of embryonic life, the ligament tends to shorten, drawing the premaxillary segment into the protrusive position with respect to the nasal septum and position in which it remains.

Normally there is differential growth between the nasal septum and the upper jaw; the cartilaginous septum slides forward relative to the bone, overshooting to the extent shown in the figure. The bilateral cleft premaxillae are carried forward by the growing nasal septum at an identical rate, since the ligament prevents their relative posterior movement. Septal growth stimulates equal elongation at the premaxillovomeral suture, so that the premaxillovomeral stem becomes much longer than normal. The additional protrusive growth of the alveolar process increases the deformity considerably, and by growing forward, it may give the erroneous impression that the entire segment is being pushed by growth of the premaxillary stem.

Given the absence of normal relationships due to clefts, the causative factors contributing to the deformity may be summarized thus: protrusion of the premaxillary basal bone is determined by the septopremaxillary ligament; elongation or excessive growth of the premaxillovomeral stem derives its motivation from the nasal septum; and the alveolar process becomes protrusive by growing in the direction of least resistance.

Unilateral Cleft Lip and Palate

Unilateral conditions of cleft lip and palate consistently show an associated skeletal deformity, of which the prominent features are lateral displacement of the noncleft maxillopremaxillary part of the upper jaw, malformation of the nose, and lateral distortion of the nasal septum.

Premaxillary Segment and Nasal Septum. The premaxillary segment, in frontal view, tilts upward into the cleft. The intermaxillary suture is also rotated markedly, as seen in coronal sections, a finding which indicates that the upturning of the premaxillary segment is due to bodily rotation of the entire segment and not solely to a local alveolar deficiency. The cartilaginous nasal septum is very much bent laterally and upward with the noncleft segment, to which it is attached in the region of the anterior nasal spine. The incisor teeth within the uptilted premaxillary segment later erupt with their crowns tilted and their occlusal plane sloping upward into the cleft. In an older patient this malocclusion indicates persistence of the original skeletal deformity present at birth.
The deviated nasal septum and displaced premaxillary region have significant implications with regard to the height of the middle third of the face. Normally the full height of the upper face is realized when the nasal septum is straight and located in the median plane. If one regards the nasal septum of the infant with unilateral cleft as being of normal size and proportions, the fact that it is bent means that it must also be shorter vertically. Thus the premaxillary segment to which it is connected suffers a decreased vertical dimension as long as the cartilaginous nasal septum remains bent. The same observation may be made for the anteroposterior dimension when the nasal septum is considerably deviated. If straightened, the nasal septum would extend further anteriorly, and a corresponding advance of the premaxillary segment would be necessary due to the ligamentous connection between these two structures. The depressed middle third of the face sometimes seen in the 10 to 12 year old may represent a residuum of the original skeletal deformity.

The nostril on the noncleft side is constricted and may be functionally occluded. The constriction results from deviation of the cartilaginous nasal septum into the floor, with the effect of raising the skin, and from the approximation of the alar base and columella. The ala nasi of the cleft side is usually stretched and flattened.

Unilateral Cleft: The Lip and Columella. If it is presumed that the labial muscle on the noncleft side is normal in bulk and attachments, there would be reason to expect that part of Cupid's bow and the philtral ridge on that side might be identifiable. However, the lip over the premaxillary segment is subjected to a unilateral muscle pull, which tends to retract it from the gingival pad over the incisor tooth of the cleft side, thus contributing to lip distortion. The latter finding can be attributed to the fact that the muscle band of the orbicularis oris inserts at the border of the cleft along the vermilion border, which turns superiorly at the cleft (see Chapter 41).

A columella may be identified in relation to the noncleft nostril, but on the cleft side it is merged with the stretched ala nasi. The columellar skin is more developed than in the bilateral cleft condition, but the deviated nasal septum and asymmetrical alar cartilages jeopardize the prospects of normal development of a symmetrical columella and adequate support for the nose.

Unilateral Cleft: Vomer and Palatal Process. In the secondary palate the cleft may be unilateral or bilateral with respect to the nasal septum. When the cleft condition is bilateral, the vomer will have a structure like that described above for the complete bilateral cleft lip and palate. It will be symmetrically attached to the inferior border of the septal cartilage, tending to thin laterally in later infancy to develop a sharp inferior edge. In either case, since the deformity of the primary palate is similar, the middle third of the nasal septum distends into the nasal cavity corresponding to the side of the cleft lip.

In the event of unilateral union between the nasal septum and a secondary palatal process, the nasal floor thus formed is stretched laterally owing to the two factors dilating the nasal cavity. The noncleft maxilla is displaced away from the cleft; the cartilaginous septum is distended into the nasal cavity on the cleft side; and the horizontal palatal process is stretched so that the vomer is pulled into the nasal floor. The suture between the vomer and the palatal process of the maxilla is thus located in the center of the floor of the nasal cavity. The mucosa covering the oral aspect of the vomer where it contributes to the palate in this
way is lined by ciliated columnar epithelium. The vomer makes a remarkable right angle
junction with the nasal septum, which remains upright.

The intact side of the secondary palate, where the vomer is joined to the palatal
process, always corresponds with the side of the intact primary palate. In the 6- to 7-week
embryo, in the developmental stage at which palate formation normally takes place, the
unilateral cleft of the primary palate favors secondary palate fusion on the noncleft side
because the septum is bent and displaced towards that side. It antagonizes fusion on the cleft
side when conditions for fusion are unfavorable because of the increased distance between
the palatal process and both the nasal septum and fellow palatal process.

At birth, both nasal cavities are functionally obstructed - the noncleft side anteriorly
at the nostril, and the cleft side posteriorly at the conchal level.

Development of Unilateral Cleft Skeletal Deformity. Interest in the cause of the
skeletal deformity has been prominently featured in some of the principles on which surgical
treatment has been based. Related information such as the time of onset and the rate of
development of the skeletal deformity has had important clinical applications in the past. The
following account is based on the anatomical study of six human specimens personally
collected and reported in the literature, in which five additional fetal specimens have been
illustrated (Veau and Politzer, 1936; Kraus and coworkers, 1966; Atherton, 1967; Latham,
1969).

Two distinct phases were identified in the development of the deformity, as seen in
coronal sections through the premaxillary region. Illustrations of the youngest known human
embryos with complete unilateral cleft of the primary palate (6 weeks) were published by
Veau and Politzer (1936). These specimens showed a deformity different from that seen at
birth in some important details. Deviation of the nasal septum toward the noncleft side is of
mild degree and similar in nature to that found later; however, the interpremaxillary suture
is inclined toward the cleft, and there is inferior displacement of the cleft-side premaxilla. At
about 12 weeks in fetal life, the direction of rotation of the premaxillary region is reversed.
The interpremaxillary suture becomes inclined toward the noncleft side, and the premaxillary
segment is rotated upward into the cleft. In the horizontal plane, lateral displacement of the
premaxillary segment toward the noncleft side is well established by the age of 8.5 weeks
(Atherton, 1967). Such displacement is accompanied by bending of the anterior part of the
nasal septum to the noncleft side. Once the nasal septum becomes angulated anteriorly, its
middle third begins to distend into the nasal cavity on the cleft side. This is seen in specimens
of 12 weeks and may occur earlier. In this manner the septal bending results in a narrowing
of the nasal cavity on the cleft side and a widening of the noncleft nasal cavity. The
distention of the nasal septum also disrupts its articulation with the vomer and, anteriorly,
with the interpremaxillary suture. Normally the nasal septum is situated directly superior to
the midpalatal suture. However, since the suture is displaced toward the noncleft side and the
septum is distended in the opposite direction except in the vicinity of the anterior nasal spine,
where the septum and bone have a strong attachment, a dislocation of the septal keel away
from the interpremaxillary suture occurs.

Onset and Rate of Development. Evidence from the youngest known human embryos
with unilateral cleft supports the view that, at the crucial time of initial cleft formation, the
primordial face is symmetrical (at 33 to 35 days) and that the deformity arises in the period immediately following, when the skeletal structures of the face begin to appear. Either the deformity is present right at the beginning in miniature and simply enlarges, or it is not part of the original affliction but rather is superimposed upon the facial structure during subsequent development. In embryos of 41 days, a mild degree of deformity was noted, consistent with the view that the skeletal deformity was present at a very early stage of formation. It may be said, therefore, that the deformity arises in the latter part of the sixth week, soon after initial cleft formation.

The skeletal deformity is well established by 12 weeks of fetal life, although the fetus does not exhibit the upturned premaxillary segment at this stage. The latter feature is seen in all specimens older than 12 weeks. Apparently the skeletal deformity develops rapidly in the embryonic and early fetal periods and subsequently increases slowly in severity as the fetus approaches full term.

**Simonart's Bar.** It is frequently found that a cleft of the primary palate is bridged by a bar of lip tissue, referred to as Simonart's bar or band. Depending upon its size, such a connection of soft tissue across a cleft may prevent the development of much of the skeletal deformity present in complete clefts. Substantial bridging bars usually pass from lip tissue to lip tissue. Narrow bridging bars may pass from the lip laterally to the alveolar mucosa medially, and this is associated with some protrusion of the premaxillary segment with malalignment of the alveolar arch.

Histologic examination of Simonart's bar shows that it may be composed of muscle fibers and a substantial number of arterioles and nerves (see also Chapter 41). These vessels may be of such size as to warrant a surgical effort to preserve them.

Simonart's bar has been the focus of attention in the discussions of the pathogenesis of the cleft primary palate. Maurer (1936) regarded Simonart's bar as the result of a healing process after breakdown has occurred. Veau and Politzer (1936), in a well-documented study, explained that the connecting bridge was the result of only partial penetration of the epithelial wall or nasal fin, which at first separates the maxillary and frontonasal processes and which is then normally penetrated by mesoderm to establish the primary palate. This subject is reviewed briefly by Töndury (1961), who suggested that Simonart's bar may result from only partial formation of the epithelial wall in the first instance.

**Cleft Palate**

A cleft of the secondary palate involves both the hard palate and the soft palate from the uvular processes posteriorly to the junction with the primary palate anteriorly; the junction corresponds with the position of the incisive foramen on an intact skull. Normal fusion of the palatal processes first occurs in the anterior third at about 47 days and progresses posteriorly to complete uvula fusion by about 54 days. The variation in severity of palatal clefts reflects the anteroposterior progress of development, and the cleft invariably affects the uvula area, which is the last to fuse. Deficiency of the bony palate varies from a midline notch in the posterior border at the normal site of the posterior nasal spine to a "V"-shaped defect extending throughout the hard palate to the anterior limit of the secondary palate. At birth the uvular processes are usually shortened and distorted in an anterior direction, presumably as
a result of the contraction of the musculus uvulae, which originates in part from the posterior border of the horizontal process of the palatine bone.

The tongue exercises great influence over the size and shape of the cleft palatal shelves and does so most obviously in some patients with the Pierre Robin anomaly (Latham, 1966). The syndrome comprises mandibular hypoplasia, paroxysmal respiratory obstruction due to glossoptosis, and cleft palate (see Chapter 50). The cleft palate is not necessary for recognition of the Pierre Robin anomaly, and occasionally there is a cleft of the entire secondary palate with impaction of the tongue into the nasal cavities. In this case the palatal shelves tend to slope inferiorly; this finding is strongly suggestive of the mode of failure of palatal formation. Underdevelopment of the lower jaw may have forced the tongue to remain between the secondary palatal processes at the time when the latter normally elevate to a horizontal position and subsequently fuse in the midline (Davis and Dunn, 1933). It is thought that intrauterine pressure, possibly due to oligohydramnios, could cause such developmental embarrassment of both the tongue and the lower jaw. The mandibular hypoplasia is frequently severe, and in such cases it is more likely to have an intrinsic cause, residing primarily in the mandibular arch cartilage (Meckel's cartilage). In the pathogenesis of the hypoplasia of the mandibular arch cartilage, the origin may lie in the cells of the neural crest. Whatever its cause, an underdeveloped mandibular arch cartilage must result in an underdeveloped mandible. The resulting mandible, while initially small, need have no impairment of growth rate at the mandibular condyles, which later develop at about 12 weeks' ovulation age.

Deficiency of mucosal tissue and bone is the main characteristic of the cleft hard palate. In the soft palate, deficiency of mucosal tissue is combined with shortening of the velar musculature, which has abnormal insertion sites (see Chapter 41). Deficiency of palatal tissue entails two factors. There may have been a deficiency of palatal mesenchyme at the time of normal palatal formation. On the other hand, a significant deficiency evidently results from the fact that the palatal processes failed to fuse. If it is postulated that normal growth of the palate is dependent to some extent on the continuity of palatal tissue across the midline, then a cleft palate will always incur some degree of subsequent underdevelopment of the palatal processes.

Absence of Midpalatal Suture. In normal development the midpalatal suture between the horizontal processes of the maxillae is established at about 12 weeks in embryonic life; the intermaxillary part of the midpalatal suture forms in the primary palate at about 6.5 weeks (Latham, 1971). In regard to the role of the midpalatal suture in the growth in width of the upper jaw, it must first be recalled that present teaching does not support the concept that these sutures generate the kind of growth force which would result in growth in palatal width. Bone formation in the midpalatal suture must be seen as a response to the tendency of the maxillae to move apart secondary to forces originating elsewhere and not in the midpalatal suture. The 5-week time lag between the development of bony support in the primary and secondary palates is another indication that growth of the hard palate in the midpalatal suture represents a secondary fill-in process rather than a mechanism of basic importance to overall jaw growth.

Therefore, a cleft of the secondary palate does not disrupt a basic growth mechanism of the upper jaw. The main skeletal function of the secondary hard palate appears to be one
of mechanical support for the partition between the oral and nasal cavities with regard to masticatory function. A second function involves bracing the molar segments against medial and lateral forces. The latter aspect becomes understandable when the continuity of the primary palate is breached by a cleft and a tendency toward maxillary collapse is observed. In such an event, an intact secondary palate counters the collapse tendency.