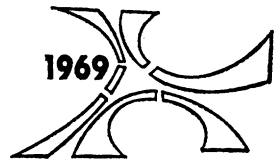
The Clinical Implications of Facial Growth in Cleft Lip and Palate



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It is impossible in a single paper to present a reasonable analysis of the subject of facial growth in cleft lip and palate and the consequent clinical implications. It might be interesting, however, to present a few of the conclusions that have been reached as a result of our research in Toronto and our evaluation of the literature.

What are the facial growth problems encountered clinically? Three major alterations in facial morphology are characteristic of almost all types of clefts. First, there is a retrusion of the mid-face and an inadequate anteroposterior growth of the maxilla (*5, 7, 8, 10, 17, 21, 30, 31, 33*). Second, there is a distortion of the dento-alveolar structures, that is, the teeth and the supporting alveolar bone. Third, there is a difference in the posture and the shape of the mandible (*11, 17, 30, 31, 33*). Figure 1 illustrates these alterations. It is fortunate that the alteration in mandibular posture reduces the protrusion of the chin, and thus tends to disguise the underdevelopment of the mid-face (*30, 33*).

An important feature of these growth aberrations is the progressive or accumulative nature of the defect (*5, 17, 26, 33, 35*). The child at age six years usually appears to have adequate mid-face development, but by the time the pubertal growth "spurt" is completed a deformity is usually apparent and often severe. This accounts for the frequency of orthodontic relapse in adolescence, when facial form can alter due to differential growth (Figure 2).

It should also be stressed that the major growth problem is maxillary retrusion; growth deficiencies in width or height are of much less clinical significance. This is partly because the latter are primarily associated with dento-alveolar changes (which are more amenable to treatment) and partly because retrusion of the maxilla gives the illusion of a deficiency in lateral width due to the tapering shape of the maxilla and mandible. Vertical problems are often related to, or result from, the deficiencies in the other two dimensions (*11*).

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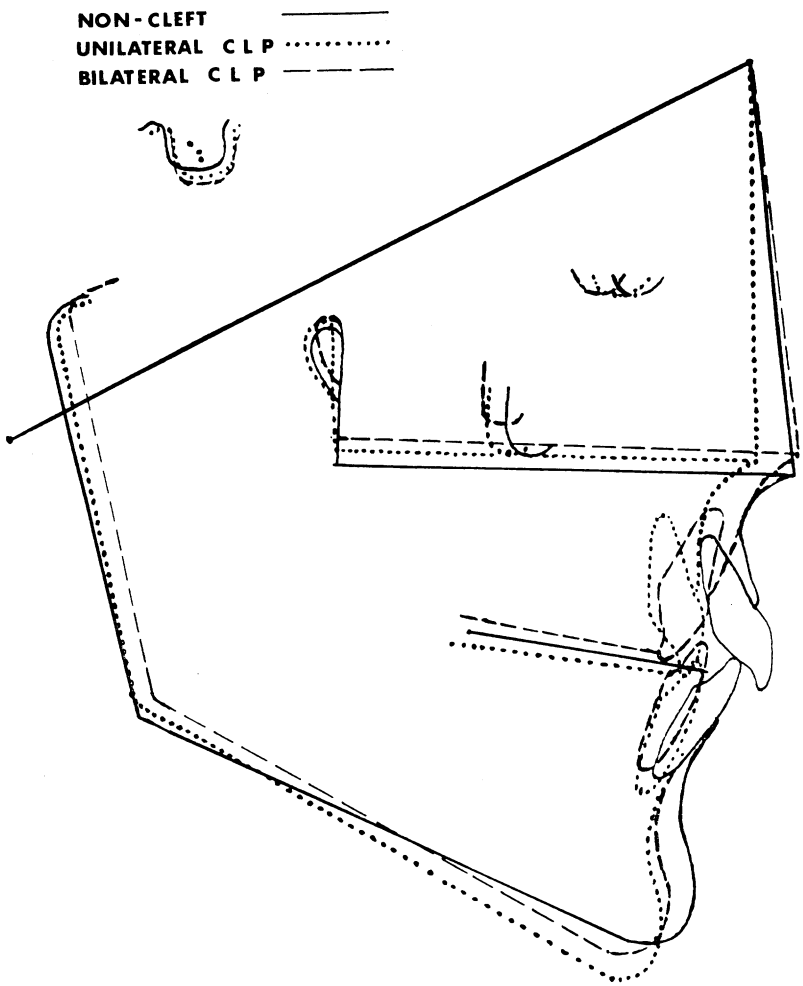

FIGURE 1. Size adjusted facial composites of two types of cleft lip and palate and a noncleft control sample, superimposed on the cranial base line (nasion to basion) (*31*).

Could these growth problems be related to a primary deficiency of tissue or to a lack of growth potential? While there is evidence of a mild or moderate embryonic deficiency of tissue in the maxillary complex (*2, 36*), it seems likely that there is normal or compensatory growth during the fetal period (*20*) and that most of the deficiency is eliminated by birth in the majority of cases. It seems likely that infants with clefts have reasonably normal mid-face development, but with occasional local deficiencies of tissue immediately adjacent to the cleft. Figure 3 illustrates an exceptional case with true mid-face deficiency. The severe distortion of the maxillary complex noted at birth is due to muscular imbalance (*28, 35, 37*) or nasal septum growth (*21*) and not

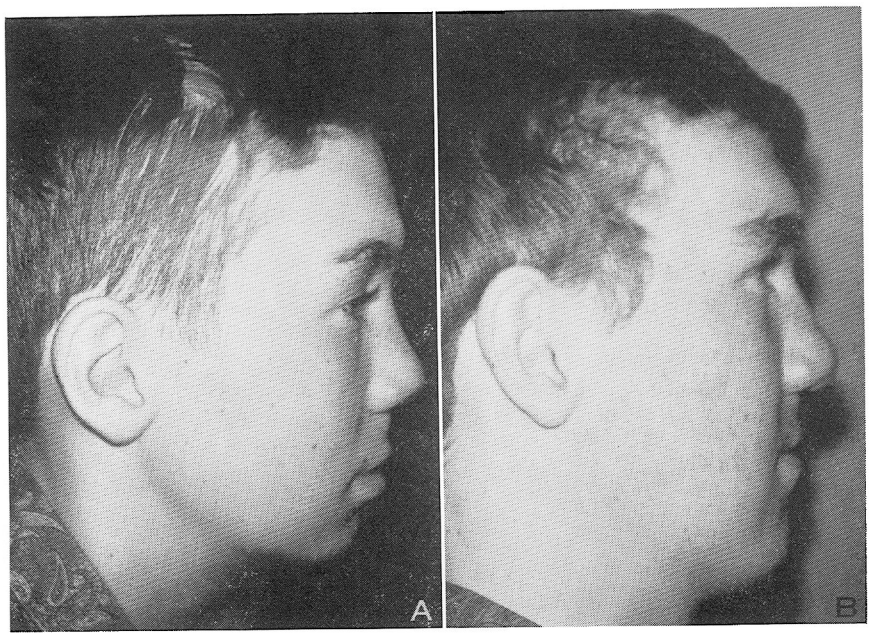

FIGURE 2. Patient M. P. illustrates the dramatic change in jaw relations that may occur during adolescence. A) Age 14.3 years, one year after the completion of orthodontic treatment, and B) age 17.3 years. Despite successful orthodontic treatment and the wearing of a retainer, the mandible continued to grow while the maxilla did not, resulting in a disharmony of jaw relations.

to an intrinsic inadequacy. When a child with cleft lip and palate does not have the palate repaired surgically, there is abundant evidence that the maxillary complex grows adequately (*1, 9, 12, 15, 24, 27, 38*). The dental occlusion in such cases is usually good because the teeth and alveolar bone are capable of compensating for any mild discrepancies in basal jaw relations that might be present. Good surgical repair of the lip does not appear to appreciably affect growth.

The evidence is overwhelming that repair of the cleft palate by almost any of the popular surgical techniques will result in an inhibition of the growth of the maxillary complex (*5, 7, 8, 9, 10, 13, 14, 17, 18, 22, 30, 31, 33, 35*). There are probably two direct surgical effects on the growing maxilla. First, there is a variable degree of *maxillary ankylosis*. Consideration of the normal growth mechanisms will clarify the use of this term.

Forward growth of the mid-face occurs through lengthening of the maxilla, since there is usually no contribution to anteroposterior growth by either the pterygoid plates of the sphenoid bone or the pyramidal process of the palatine bone. The normal maxilla increases in length by a forward movement of the entire bone and a concomitant apposition of bone to the posterior tuberosity (Figure 4). The anterior surface

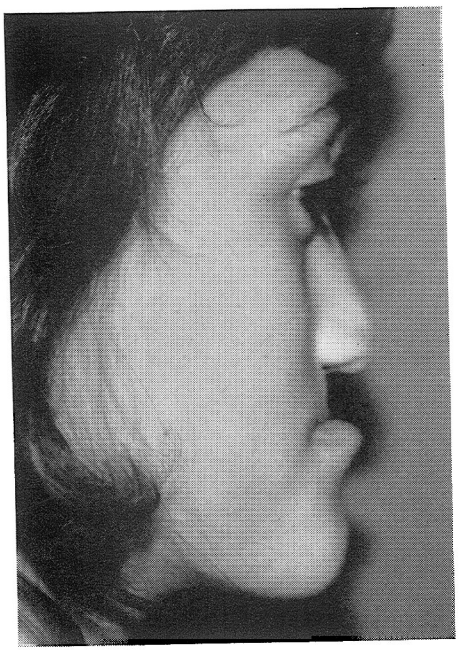

FIGURE 3. A true midface deficiency involving the entire maxillary complex. Overclosure of the mandible causes protrusion of the chin, although the mandible itself is not abnormally large. This is therefore a relative prognathism.

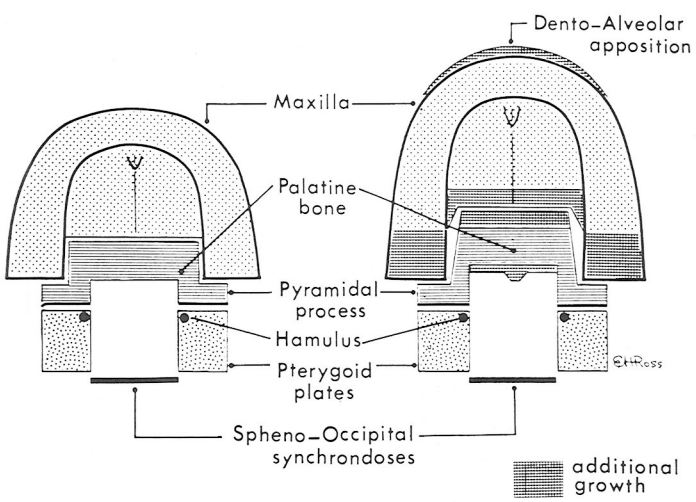

FIGURE 4. Diagrammatic representation of the mechanism of maxillary forward growth. Note that increase in length results almost entirely from apposition to the posterior surface of the tuberosity, and that pharyngeal depth remains the same. The pyramidal process of the palatine bone migrates posteriorly by anterior resorption and posterior apposition (*6*).

of the basal maxilla can be expected to show resorption rather than apposition (*3, 4, 6*) (except in infancy), although there is some dento-alveolar apposition which contributes to over-all length.

The mechanism by which the forward movement of the maxilla occurs is not completely established, but the best evidence suggests that the maxilla is not pushed forward with a positive force such as is generated by an epiphysis (*19*); rather it more or less drifts forward in response to a number of factors including the facial sutures (*3, 34*), the nasal septum (*32*), and the soft tissue functional matrix (*25*). The significance of this is that the maxilla is more readily inhibited than is a true growth center such as an epiphysis.

How does surgery to the palate affect this process? In many surgical techniques, the hamulus is fractured and some dissection of tissues in the area is carried out to release tension on the soft palate rudiments and to alter the direction of the tensor palati muscle. The mucoperiosteum covering the palate is raised and displaced medially and frequently posteriorly. As a result of these procedures, a continuum of scar tissue joins the maxilla, the palatine bone, and the ptyerygoid plates of the sphenoid, inhibiting separation of these bones and thereby creating a condition which could be termed *maxillary ankylosis* (Figure 5). A thick band of tissue frequently appears on either side of the posterior palate after surgery (Figure 6). The posterior insertion of this band is obscure, but, in any event, it is an artificial structure created by surgery and probably contributing to maxillary ankylosis.

A second direct effect of palatal surgery is the distortion of dento-alveolar growth. Most techniques of palatoplasty leave an area of denuded bone close to the alveolar process which is rapidly covered by scar tissue. The initial contraction of the tissue results in a medial movement of the maxillary segments and a medial tipping of the teeth and

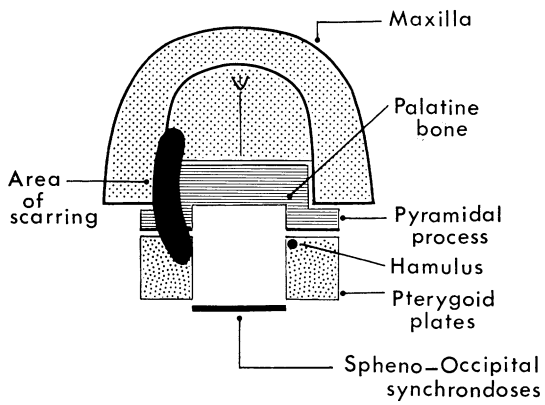

FIGURE 5. Following a typical surgical repair of a cleft palate, an area of scar tissue forms which may unite the growing complex of bones and inhibit further growth to a variable extent.

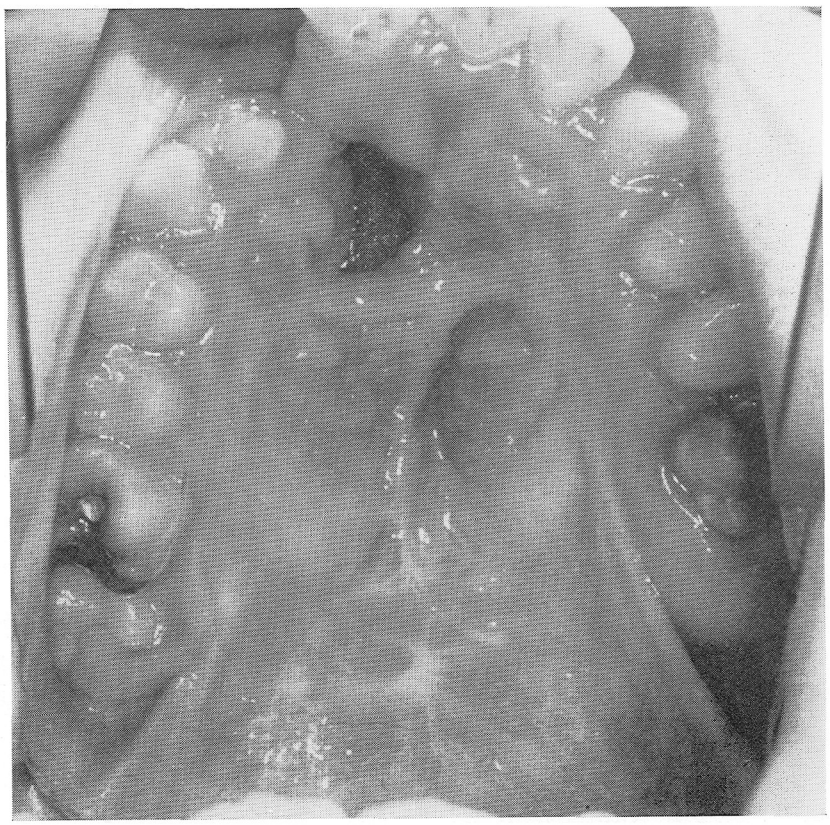

FIGURE 6. A thick, taut band of tissue frequently inserts into the maxillary tuberosity and continues forward into the palate. This may have an inhibiting effect on forward movement of the maxilla.

alveolar bone. The latter occurs due to the insertion into the scar tissue of periodontal fibers from the teeth. As further tooth eruption and vertical development of the alveolar process occurs, the area of scar tissue adjacent to the alveolar process resists growth and thereby induces a medial and posterior deflection of the dento-alveolar structures and a collapse of the dental arch. Basal bone appears to be relatively unaffected after the initial medial movement (Figures 7 and 8), unless extremely traumatic or multiple surgical procedures are performed.

The secondary effects of palatal surgery are associated with a large number of other factors of abnormal physiology which together cause a dramatic change in the posture and form of the mandible. Many of these factors are illustrated in Figure 9.

If these conclusions are valid then obviously the surgical procedures customarily practiced, at least in North America, should be modified. There is some indication that the posterior ankylosis may not be seriously handicapping if the dento-alveolar structures remain free

to adapt to and to accommodate moderate discrepancies in jaw relations (*12, 29*). To prevent dento-alveolar distortion and to permit dento-alveolar adaptation, no operative procedures should be performed which leave an area of denuded bone adjacent to the alveolar process. To minimize ankylosis of the maxilla, great care should be practiced to reduce scarring across the pterygoid-palatine-maxillary junction.

It would therefore seem desirable to use such procedures as the Schweckendiek method of managing palatal clefts where only the soft palate is repaired in infancy. Non-inhibiting obturators may be inserted until the hard palate is spontaneously closed to a large extent by growth of the palatal shelves, at which time a relatively minor midline surgical procedure completes the closure with a minimum of residual scar tissue. The results are reported to be excellent (*12, 29*). As to the timing of palatal surgery, from a theoretical point of view it would be preferable to postpone surgery until maxillary growth is almost completed, that is, until the age of twelve years or later. Clinical studies support this hypothesis (*12, 23*). The theory that there will be less interference with maxillary growth if surgery is postponed until the age of four to six years is of little clinical value, since it assumes that the growth deficiency is in maxillary width. As stated previously, with current surgical procedures, width of the maxillary basal bone is rarely a problem.

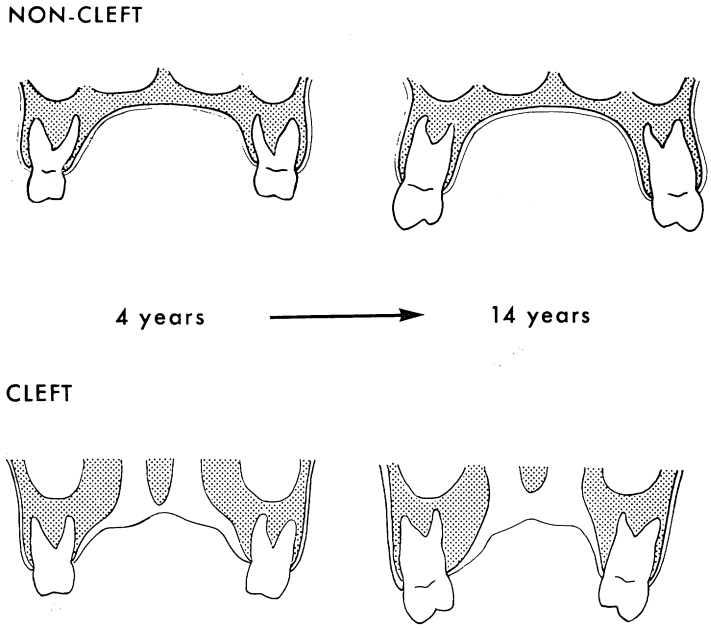

FIGURE 7. Dento-alveolar development in cleft and noncleft cases. Note that normally the alveolar bone bulges beyond the lateral extent of its basal bone, but in cleft palate cases the alveolar bone is narrower than the basal bone.

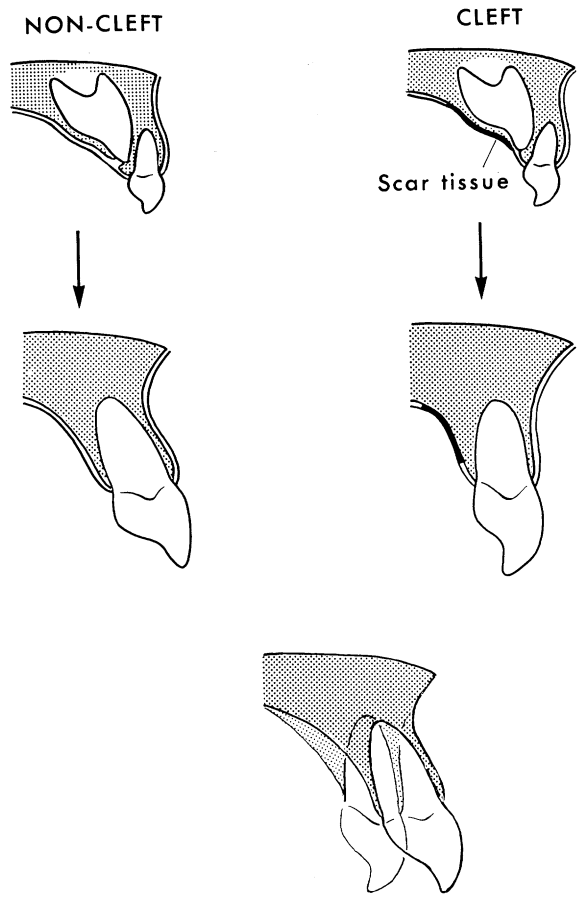

FIGURE 8. Incisor eruption in cleft and noncleft cases showing the differences relative to the basal bone (anterior nasal spine).

Postponing palatal surgery may create a number of additional difficulties, particularly in the area of speech development, so that it would seem advisable to develop methods of palatal surgery which could be performed at an early age, yet would not interfere with mid-facial development. There should no longer exist an either-or situation where it is necessary to choose between excellent speech or excellent facial development: both should result from properly designed palatal surgery.

Let us briefly consider infant orthopedic procedures, which may be very beneficial to the child with a cleft lip and palate. The proponents of these procedures, however, sometimes claim that the manipulation of the maxillary segments in infancy will decrease the need for future orthodontic treatment and promote maxillary growth. Since no long-term evidence to support this contention has appeared,

we should examine the rationale. There are three reasons why I cannot presently accept their hypothesis, at least as applied to unilateral clefts. First, it is difficult to understand how infant orthopedic procedures can alter growth, since they appear to merely tip the maxillary segments slightly. Second, the benefits that are obtained seem to be limited to the lateral or width dimension of the maxilla, and this is prevention of a relatively minor problem. Anteroposterior growth is the major problem and is probably not affected by infant orthopedics. Third, these procedures are carried out at an inappropriate time: growth problems are insignificant before palate surgery and thereafter gradually increase in severity to become worse in adolescence. There is no doubt that a continuous program of orthopedic procedures can produce an excellent dental occlusion in the primary dentition, but the minor problems usually found in the primary dentition can be treated quite easily and efficiently by conventional orthodontic means, and probably with equal long-term effect.

Bone grafting is a procedure which aids the orthodontic placement of teeth and provides greater maxillary stability. Theoretically, however,

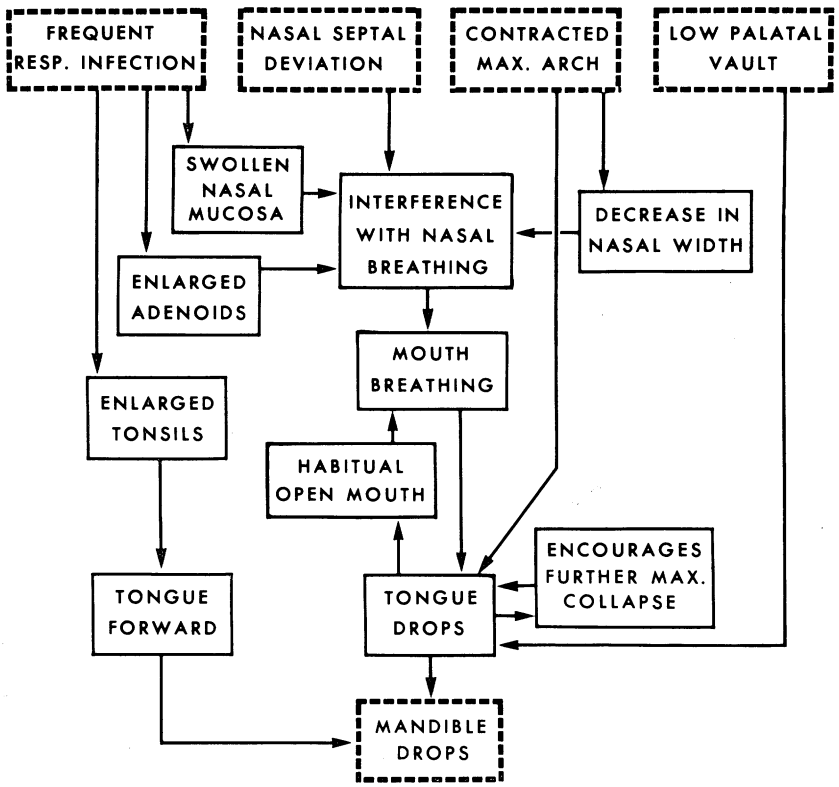

FIGURE 9. Diagram illustrating many of the factors which contribute to the alteration in mandibular posture.

this procedure should have almost no effect on long-term maxillary growth, since the graft is placed in an area where growth does not usually occur. Primary bone grafting could prevent the initial medial movement of the maxillary segments, but would have no effect on the secondary dento-alveolar distortion related to palatal surgery. This probably accounts for the conflicting reports on the results of bone grafting; the critical variable may be the surgical procedure used in repairing the palate, not the placement of the bone graft.

Summary

There are many clinical implications suggested from growth studies and these, as well as supporting evidence, will be presented in more detail elsewhere. Modern palate surgery is excellent in most respects, but from the point of view of facial growth, many of the procedures used are unsatisfactory. Excellent methods are apparently available and new methods could probably be developed. Another conclusion drawn from a great deal of indirect evidence is that infant orthopedic procedures are essentially irrelevant to the long-term facial growth of the child with a cleft lip and palate.

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