Growth sites and growth mechanisms at risk in cleft lip and palate

Hans Friede
Department of Orthodontics, Faculty of Odontology, Göteborg University, Göteborg, Sweden

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A newborn with some kind of facial cleft displays certain characteristics of the nose, upper lip, and jaw caused by abnormal influence on specific growth sites and growth mechanisms. Treatment, particularly surgery, attempts to counteract this aberrant development, for both functional and aesthetic reasons. However, not infrequently, therapy impedes future midfacial growth to a greater or lesser degree. To better understand the varying growth influence, this article aims to review certain aspects of growth of the middle third of the face in both normal and cleft subjects. The normal elongation of the maxilla, to give space for the molars, is usually not affected by lip surgery but rather by scar tissue from palatal repair. The displacement of the upper jaw in relation to the vomer is recognized. Early surgery should therefore avoid affecting the growth of the vomero-(pre)maxillary suture if possible. Periosteal growth, necessary for the development of dentoalveolar structures, might be affected by scar tissue from palatal repair. Different ways to reduce the development of palatal scars and their negative effects on growth are discussed. □ *Maxilla; periosteum; scar tissue; surgery; sutures*

Hans Friede, Department of Orthodontics, Faculty of Odontology, Göteborg University, P.O. Box 450, S-405 30 Göteborg, Sweden

A satisfactory facial appearance and a normalized dental occlusion are among the key factors when the pros and cons of different treatment methods in cleft lip and palate (CLP) habilitation are evaluated. Both appearance and occlusion depend substantially on the facial skeletal growth being as unaffected and normal as possible.

In children born with CLP, there might be different categories of facial growth aberrations: intrinsic, adaptive, and induced changes (1). The intrinsic factors are primarily related to the cleft formation process in the early embryonic period, but might also influence later development. An example of adaptive growth is the premaxillary protrusion seen in bilateral CLP. The varying position and growth of the premaxilla in these patients have a considerable influence, not only on treatment but also on its outcome. The third category, the induced aberration, is related to treatment of the cleft condition, particularly its surgical management. Unfortunately, the literature has often dealt with these induced abnormalities in a nonspecific way with no clear analysis of the mechanisms underlying the observed growth deviations (1).

Normal growth

To be able to discuss growth abnormalities of the facial skeleton, it is necessary to understand normal development. As CLP is under consideration in this review, the focus will be on mid-facial skeletal development, more specifically on maxillary growth. The bony maxilla is developed from different ossification centers. As early as the late embryonic/early fetal period (9th–12th intrauterine week), the premaxillary and maxillary bones on each side of the face fuse at their ectofacial surfaces (2, 3). From that stage on and into adulthood, there is no possibility for separating growth between the two bones even if an incisive suture, though gradually closing, can be seen on the palatal bony surface (4–6). The incisive suture has also been called the incisive fissure, as only minor remodeling growth activity is found in this structure (7–9). Nevertheless, a few clinicians maintain—without presenting solid, unbiased evidence—that it is possible to stimulate growth in the incisive suture (10–13).

Maxillary bone growth

In the anteroposterior dimension, sutural growth of the maxilla takes place in the transverse palatine suture (14, 15) (Fig. 1). The anterior border of the suture might have more bone growth activity than the posterior one (16). Already from early childhood very little periosteal growth on the anterior maxillary surface contributes to the forward development of midfacial structures (4, 17, 18). How much apposition occurs at the posterior border of the palate is more difficult to test and therefore controversial. For instance, while Melsen (19), using histologic evidence, found that the posterior margin of the hard palate showed deposition of bone until adulthood, Sejrsen et al. (16), studying archaeological material, concluded that very little growth occurs at the posterior border of the hard palate.

In the transverse dimension, growth in the midpalatal suture is well demonstrated by metallic implant studies (17, 20, 21). Sutural activity is most pronounced in the posterior part of the suture and continues until adulthood (Fig. 2). Periosteal growth of dentoalveolar structures does not contribute to the increase in width of the maxilla, if measured in the dental arch (17). Finally, in the vertical dimension, the maxilla is relocated downwards through appositional growth in the hard palate as well as the alveolar process. Simultaneously, bone resorption occurs

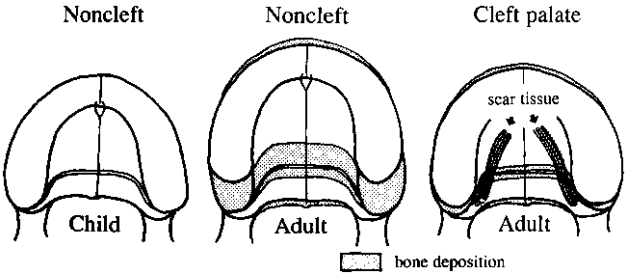

Fig. 1. Schematic illustration of anteroposterior growth of the maxillary complex in noncleft as well as cleft palate situations. Normally, the maxilla is elongated, particularly in the transverse palatine suture and at the tuberosities. Only sparse bone is deposited on the anterior (alveolar) and posterior (palatal bones) surfaces. If abundant scar tissue has developed after repair of the cleft palate, it restricts forward displacement of the maxilla, resulting in a shorter bone complex. Modified from Ross (50).

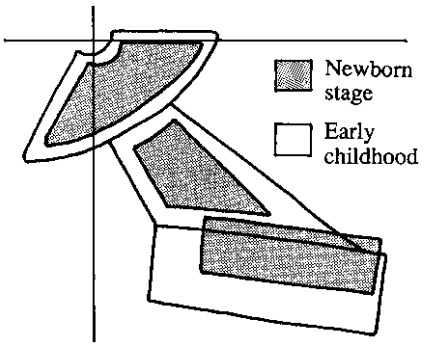

Fig. 3. Schematic drawing of the sphenoid bone, vomer, and maxilla as seen in the lateral projection. During growth up to early childhood, the latter two bones have been displaced in relation to the sphenoid. Note also the sliding growth between the vomer and the maxilla. Modified from Friede (22).

on the nasal floor, while bone is relocated upwards toward the orbital floor (17).

Normal midfacial growth also involves displacement of the maxillary/palatine bone complex relative to neighboring bones, but in this respect only growth in the vomero-premaxillary/maxillary/palatine suture will be mentioned. It has not been possible to obtain actual measurements of growth of this structure in normal subjects, but indirect evidence from cleft children is available (22). During childhood it has been found, in both unilateral and bilateral cleft lip and palate subjects, that the maxillary complex is displaced forwards–downwards in relation to the vomer (22, 23) (Fig. 3). This movement might be generated by the septo-premaxillary ligament (24) or some other functional mechanism.

Surrounding soft tissue matrix

Before turning to the pathologic state, we should also briefly consider the soft tissue matrix that surrounds the lower part of the maxillary complex. Besides the tongue there are two different muscle systems that influence maxillary growth (12). In the front there is the anterior facial muscle chain consisting of the nasolabial muscle ring and the upper part of the orbicularis oris muscle (Fig. 4). Both of these are said to transform growth forces to the maxilla and its anterior surface. Posteriorly, there are the deep facial and cervical muscle chains (Fig. 5). The upper part of the latter ring supports the soft palate and the tongue. Like the anterior chain, these posterior chains are thought to provide impetus for growth of the upper jaw, especially to the posterior and lateral parts of the maxilla.

The surface of the palatal mucoperiosteum has three different characteristics (25) (Fig. 6). The midline mucosa covering the midpalatal suture and the palatal shelves is thin compared with the fibromucosa of the maxillary bone, which is thick and vascular. It includes the neurovascular bundle and is partly covered with rugae. Then there is the thinner gingival fibromucosa covering the alveolar process of the maxilla and surrounding the necks of the teeth. The three types of palatal fibromucosa with different characteristics are related to their different functions.

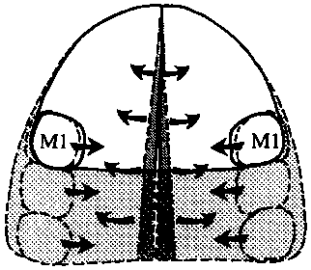

Fig. 2. Drawing illustrating transverse maxillary growth in the midpalatal suture. Note that most growth is posterior. The dental arch is also widened, but generally slightly less than the bones. Therefore, the teeth might have to relocate medially. At the level of the first molars (M1), the mean of this relocation is about 1–2 mm from early school age to adulthood (17).

Growth in CLP

It was not until the 1950s that it became clear that surgery was the most significant factor influencing maxillary development in cleft patients. Graber (26) pioneered this research by writing several articles on the subject, and in 1954 he summarized his findings on facial morphology in subjects with cleft lip and palate. His main point was that early traumatic surgery, particularly if using the Brophy-type compression closure (27) (Fig. 7), caused severe anteroposterior and vertical deficiencies of the maxilla. The preoperative molding of the maxillary segments by use of intramaxillary wiring interfered not only with the subsequent growth of the maxillary complex proper but also with the development of the upper dentition. He also found that multiple palatal surgery produced the same

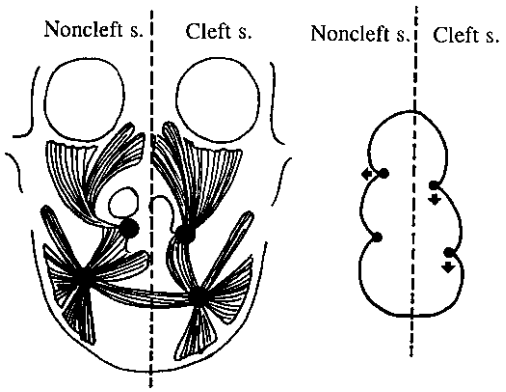

Fig. 4. Drawing of facial muscles in noncleft and cleft lip conditions. The upper (nasolabial) and middle (orbicularis oris) muscle rings have been broken by the cleft of the lip, causing distorted growth of the soft tissue of the nose including its underlying bone. The ruptured chain of muscles also deforms the alar base and the lip morphology. s. = side. Modified from Markus et al. (30).

result, but to a lesser degree. Graber only briefly speculated about the growth sites affected and the mechanisms causing the deformity when mentioning factors such as development of 'cicatricial bands of tissue resulting from surgical manipulation' and 'early and repeated trauma to maxillary growth centers'. Other early writers ascribed the surgical growth interference to 'diminution of blood supply to the maxillary parts concerned' without speculating further about which growth mechanisms were involved (28). However, the idea about the lack of blood supply to the maxillary bone complex after cleft surgery was not generally accepted (29).

In their classic text from 1972, Ross & Johnston (14) pointed out that, in modern surgery, the immediate effect of cleft repair is usually not detrimental but beneficial to midfacial morphology, as the functional continuity of the lip and palatal muscles is restored. The preoperative distortions of the nose and maxilla, caused by the abnormal muscle actions, are then reversed. However, the long-term effects of surgery on facial development might vary considerably. This largely depends on the type of cleft operated on and the method of surgery used. The main thrust of the present article is to review current knowledge regarding these aspects.

Lip repair

If first considering lip surgery in unilateral cleft of the lip, without or with cleft palate (UCL or UCLP), lip closure influences skeletal growth of the maxilla only in rare cases exhibiting extreme tissue deficiency. Pressure from a tense upper lip might cause maxillary anterior teeth to become retroclined, usually resulting in an anterior crossbite situation. However, according to Ross & Johnston (14) and most others, lip tension from 'normal' lip repair has very little effect on the long-term growth of the basal structures of the maxilla. This opinion is supported by the fact that no major growth sites involved in normal forward–downward maxillary growth are part of the surgical field. More recently, some surgeons have speculated about the importance of performing lip surgery that is as meticulous and atraumatic as possible. The reconstruction of perioral and perinasal muscles has been considered especially crucial to allow undisturbed development after surgery (13, 30, 31). Reconstruction of the broken nasolabial muscle ring (Fig. 4) is regarded as a prerequisite for symmetric development of nasal structures as well as a necessary stimulus for further midfacial growth. However, most reports are based on observations and discussions of individual cases, and little objective evidence of improved development is presented. Even a comparative investigation of two groups of patients (32) does not give firm guidance, as palatal surgery had been changed simultaneously with the change in the type of lip repair.

Another recent article, also speculating about the growth effects of lip surgery, studied adult UCLP patients who were 'nonoperated', 'only lip operated', or 'lip as well as palate operated' (33). The authors found that lip repair definitely affected growth; in fact, it was reported to have greater influence on midfacial morphology than the additional palatal surgery. As pointed out in the article, the clear effect of lip surgery might be caused by the fact that this surgery was performed first. The initial operation might have the greatest effect, whether in the lip or in the palate. A further uncertainty is that the quality of the operations might be hard to judge retrospectively, as the surgery was performed a long time ago. Therefore, in the absence of a conclusive study showing the crucial effect from 'modern' lip closure, the conclusion of Ross (34) still appears most well founded: cleft lip repair, if properly executed, has an insignificant long-term effect on facial growth and dentofacial development. Actually, this agrees with the opinion of Brodie almost 50 years ago that the repaired cleft lip has no effect on the 'positioning' of the maxilla; it might influence maxillary 'form' only (28).

However, in bilateral complete cleft lip, without or with cleft palate (C-BCL or C-BCLP), the repaired upper lip provides the main force to mold back the initially protrusive premaxilla into the maxillary dental arch. Usually, this gradual retropositioning of the premaxilla occurs slowly and is completed around or slightly after puberty (35, 36). A few studies have been reported in which several patients have remained premaxillary protrusive even many years postoperatively (37, 38). Lip surgery has, in some of these cases, produced a loose or short upper lip riding above the premaxilla, unable to restrain it and bring it into a more normal position. No doubt, repair of the lip in bilateral clefts influences long-term midfacial development by causing both dentoalveolar and basal changes. Regarding the latter, the vomero-premaxillary suture (VPS) is a crucial structure (39, 40). If a normal profile in adulthood is to be aimed for, growth of the VPS should not be interfered with, particularly during the first years of life. It is therefore hardly surprising that surgical retro-

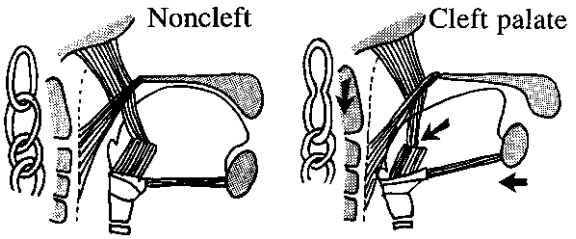

Fig. 5. Schematic diagram of the deep facial and cervical muscle chains in noncleft and cleft palate conditions. A cleft of the soft palate breaks the two upper rings, resulting in a lowering and retraction of the tongue. Reduced growth stimulus for the maxilla and its alveolar structures might be the outcome. Modified from Markus et al. (12).

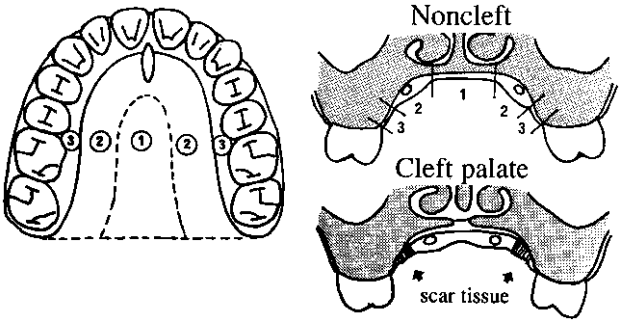

Fig. 6. Three zones of palatal mucosa: 1 = palatal shelves; 2 = maxillary; and 3 = gingival fibromucosa. At repair of the palatal cleft, the maxillary part of the mucosa including its periosteum has been moved medially on each side, causing lateral denudations of maxillary bone. Scar tissue has formed close to the teeth, which has contributed to the constricted dental arch. Modified from Markus et al. (12).

position of the premaxilla—as in early surgical setback (41), or with preoperative, extensive mechanical retrusion of the premaxilla, as in the Latham approach (42)—later results in midfacial deficiency in most bilateral clefts (43).

Repair of alveolus and anterior nasal floor

The VPS is close to or part of the surgical field at lip closure in UCLP, too, and might therefore be interfered with in this cleft type as well. Scarring from simultaneous surgery of the upper alveolar process, including the anterior nasal floor, might restrict the necessary forward–downward sliding of the maxilla in relation to the vomer (22). It might then cause maxillary retrusion and even a tendency toward anterior open bite. If the function of the VPS is completely blocked, as it is after early bone-grafting, then the importance of this suture is strikingly demonstrated. Both forward and downward growth of the anterior maxilla are severely affected (44, 45). Even after only soft tissue repair of the alveolus, with still some chance of scar tissue interference with the growth of the VPS, the anterior maxilla displays less vertical development than in cases where lip repair does not include any alveolar surgery (46).

Palatal surgery

In the midline of the uni- or bilaterally cleft maxilla, the intermaxillary suture is malpositioned or not even present. Relatively normal growth adjustments might still occur in this area, and correct transverse occlusion usually develops in non-operated CLP patients (14). If surgery results in bony union in the midline, either from a bone graft (47) or from the creation of a periosteal envelope promoting bone formation (48), then transverse as well as sagittal growth displacement of the maxillary bones will be impeded. However, as compensatory dentoalveolar growth might occur simultaneously, the adverse effect on transverse occlusion in particular is usually less than expected (49).

Only in rare cases is growth of the transverse palatine suture blocked by bony fusion across the suture line from treatment (47). However, separating growth in this structure might still be interfered with, and then it is caused by severe palatal scar tissue from the soft tissue repair (50) (Fig. 1). In most surgical techniques muco-periosteal flaps are raised and displaced medially and frequently posteriorly. At healing the denuded palatal bone becomes covered by scar tissue, which, if extensive, might join the maxilla, the palatal bones, and the pterygoid plates of the sphenoid. Ross (50) termed this condition maxillary ankylosis, though it was thought to be caused by fibrous tissue and not by bone.

Another effect of the palatal scar tissue is, according to this view, the influence on periosteal maxillary growth, causing distortion of dentoalveolar structures. The contractive forces from the fibrous scars might result in medial movements of maxillary segments and medial tipping of teeth and alveolar bone. In the sagittal plane the incisors respond to the palatal scar tissue by becoming retroclined. Even maxillary tooth eruption and vertical development of the alveolar process might be reduced owing to growth interference of the scar tissue, if it is severe. It should be remembered, though, that many other factors might also be at play, adding to the dentoalveolar distortion in cleft palate patients. Abnormally low tongue posture deteriorates maxillary dental arch development both horizontally and vertically (14) (Fig. 5).

To reduce the amount of scar tissue and move its position further away from sensitive growth structures, different types of palatal surgery can be advocated. For instance, two-stage palatal closure has been suggested because the first surgery, soft palate repair, usually considerably reduces the width of the cleft in the hard palate (51). According to Markus et al. (25), then only the central mucosa of the vault covering the palatal shelves can be used at the second stage; in their experience, this lessens the negative growth influence from surgery. If, on the other hand, fibromucoperiosteal flaps covering the maxillary part of the palate are raised and advanced medially, the results are denudation and scars more laterally, closer to the teeth. This will increase the risk of adverse effects on

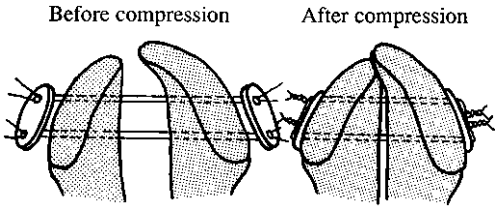

Fig. 7. Illustration of the traumatic Brophy method of palatal compression to narrow the cleft (27).

dentoalveolar maxillary growth in particular. After a follow-up study of pushback repair in cleft palate patients, my co-workers and I have come to a similar conclusion. We found that the closer the scar line was to the teeth, the higher the risk was of developing a narrow upper dental arch (52). Similarly, in another study we found better development of maxillary intercanine width in UCLP patients who had been treated with the von Langenbeck method of palatal surgery (less scar tissue) than in those treated with the pushback technique (risk of more scar tissue) (53).

Other ways to reduce negative effects from scars from palatal repair are reported. One method is to use supraperiosteal instead of mucoperiosteal flaps to cover the cleft area; that is, the palatal bone left exposed after surgery is not stripped from its periosteum (54). Compared with scar tissue formation over denuded bone, scars after wounds where the deeper layer of the mucoperiosteum was left in situ were found to differ in structure, composition, and outline (55). Even if the patients were followed up to only 5 years of age, this more beneficial healing after surgery resulted in greater maxillary arch length than in a group of patients in whom palatal bone was left completely denuded at repair. A further way to lessen the risk of palatal scar tissue formation at repair might be to cover the exposed periosteum, for example after a vomer flap, with a skin graft (56, 57).

Final comments

No doubt, the dentofacial growth results after habilitation of cleft lip and palate patients have improved considerably over the years since Graber (26) described the poor outcome of the aggressive surgery (Fig. 7) used on his patient sample. This change has been possible only through close cooperation between surgeons and orthodontists, working together in teams. By use of longitudinal follow-up studies, it has been possible to demonstrate the weaknesses and advantages of certain surgical methods, and this has resulted in changes in treatment routines (e.g. 58). The interpretation of why a certain growth site or growth mechanism has been affected by surgery is based on a thorough understanding of the specific techniques used for cleft patients. It is equally necessary to have a full understanding of how maxillary growth takes place, both in normal and in cleft lip and palate subjects. In this review particular attention has been paid to the growth issues.

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