# Cleft growth after primary palatoplasy. factors such

Design

Cleftpalate is among the most common deformities of the craniofacial region, with aprevalence of clefts being somewhere between 27, 000 and 33, 000 clefts/year inIndia9. The presence of a cleft palate leads to feedingdifficulties, hearing impairment, impaired speech development, and thepossibility of decreased facial growth. Cleftpalate patients develop an impaired midfacial growth after primary palatoplasy.

Factorssuch as intrinsic developmental deficiencies and functional distortions areinitially involved but palatal surgery seems to be a main factor in these growthproblems 10-16. A strong indication for the involvement of iatrogenic factorsis the largely undisturbed maxillary growth in untreated patients 17, 18, 19. In treated patients, the healing of surgical wounds after palatoplasty isprobably responsible for the growth disturbances 20. Woundhealing process is quicker in intraoral wounds than in the skin andproduces less scar tissue.

21. Decreased levels of pro-inflammatory andpro-fibrotic cytokines have been found in mucosal wounds 22. The presence ofsaliva and large numbers of bacteria also have an influence on intraoral woundhealing 23. Saliva contains many growth factors such as epidermal growthfactor(EGF).

Also, phenotypic differences between skin and mucosal fibroblasts may beinvolved 24). In palatal mucosa, themucosa and periosteum are merged and attached to the palatal bone 25. The palatalmucoperiosteum is much more rigid than buccal mucosa and it contains fewerelastin fiberes26. The palatal mucosal epithelium is generally thicker than the buccal mucosa.

Therefore, thephysiological and mechanical characteristics of the palatal tissue is differentfrom buccal mucosa, which explains the differences in the outcome of the woundhealing process 27 Enlow 28states that: Growth is not “ programmed” within the calcified part of the bone itself. The “ blueprint” for the design, construction, and growth of a bone lies in the functional matrix: the compositeof the muscles, tongue, lips, cheeks, integument, mucosa, connective tissue, nerves, blood vessel, airways, pharynx, the brain as an organ mass, tonsils, adenoids, and so on. Growth fields (growth sites) for example, a suture and thealveolar bone housing teeth, throughout a bone do not have thesame rate of growth activity. Some” fields” grow much more rapidly or to a greater extent than others. The same istrue for resorptive fields. All surfaces are sites of growth; relocation of thebone, going from one location to another, is the basis for remodeling. In themaxilla, the palatal growth is in the downward direction by periostealresorption on the nasal side andperiosteal deposition on the oral side. The nasal mucosa provides the periosteum onone side and the oral mucosa provides it on the other side.

This results in adownward location of the palatal maxillary arch . Depending on the extent of palatal surgery and the resulting scar tissue, the stability ofa region can be disrupted if theresults negatively affect the pattern of resorptiveand depository fields on bone and at the suture; that is, scarring can work against growth. As the midface grows, bone is laid down in the sutures surroundingthe maxillary complex.

Any damage to oneof these sutures can interfere with the direction and amount of growth. Forexample, an excessive amount ofscarring at the pterygomaxillary suture, (PTM) or at the premaxillary vomerinesuture (PVS),

will interfere withanteroposterior and vertical maxillary and premaxillary growth. Growthmovement of the premaxilla is produced by the growth expansion of all the bonesbehind andabove itand by growth in other parts of the maxilla, especially at the premaxillaryvomerine suture (PVS). The premaxilla itself contributes a major part of its ownforward growth movement through changes at the PVS.

These displacement growth movementsare a result of the “ carry effect,” as Enlow calls it, which is produced by theexpansion of the soft tissues associated with the bones, not a “ pushing effect” of bones against bones. Scarring of the palatal mucoperiosteum, therefore, actsto interfere with the “ carry effect,” thus preventing the change in position ofthe maxilla within the face. In the lastcentury, surgeons involved in cleft palate treatment usually performed surgicalprocedures whose sole purpose was to “ close the hole” as early as possiblewithout considering the ultimate effect of the surgery on palatal, facial, orspeech development. These procedures, Millard 29 reports, fall into threecategories: 1.

The useof various kinds of flaps from other parts of the body to cover the cleftspace. 2. Treating the edges of the cleft so that they could be sutured together bypulling the mucoperiosteumover thecleft. Failure of a lasting union led to the use of laterally positionedrelaxation incisions by Dieffenback in 1826 and von Langenbeck in 1862 30. 3. Stagedsurgical treatment.