Complementary Role of Pharyngoplasty in Cleft Palate.

Essay

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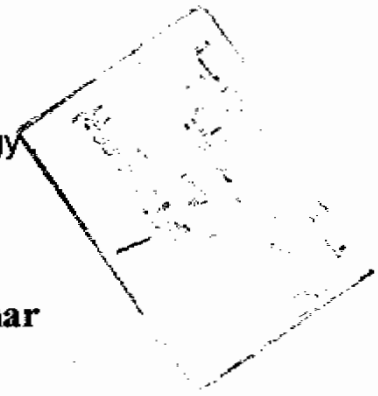

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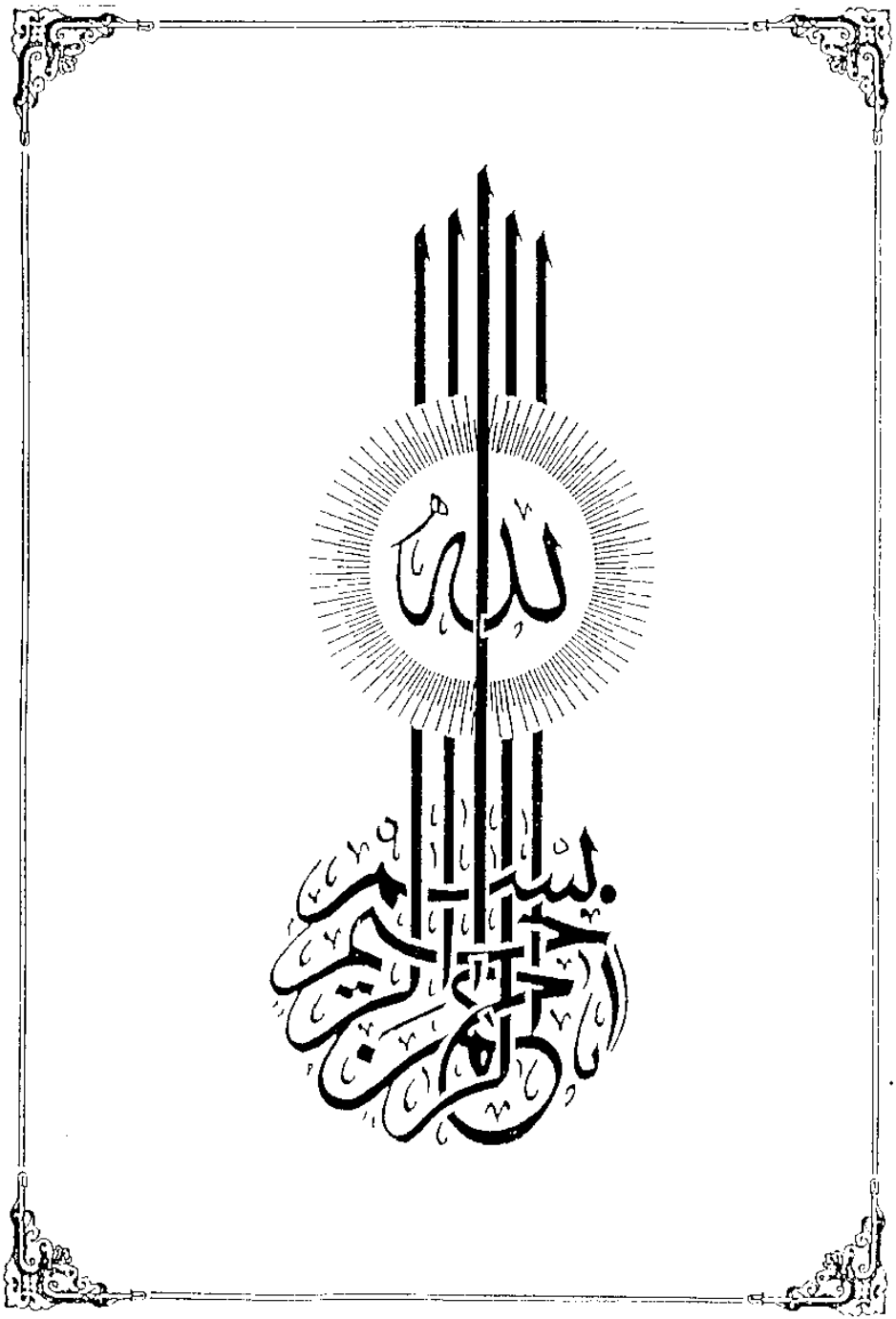

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INTRODUCTION

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The cleft palate is a congenital malformation that disables many children with frequency higher in caucasians (1.3per 1000 live births) than in negros (0.4 per 1000 live births). Heredity and environmental factors influence the incidence of cleft palate. The environmental factors include vitamin deficiencies (Vitamin A and folic acid), nucleic acid antagonists, corticosteriods, alcohol, and smoking.

The cleft can adversely affect feeding, speech development, hearing, nasal breathing and facial appearance.

Although the reasons to repair the clefts may appear obvious, the foremost is to restore the mechanism of normal speech. Normal midfacial growth patterns and decreasing the chances for development of middle ear disease are secondary objectives of the repair.

The optimum timing for palatal repair is controversial. Early closure is associated with defective midfacial growth in spite of the better speech, whileas late closure does not usually jeopardize the dentoskeletal growth but is usually associated with impaired speech.

Several techniques have been described to repair a cleft but non is invariably accepted as a solution to all the functional deficits; normal speech, hearing, and midfacial growth which are not always corrected to the same degree by a specific procedure.

A considerable percentage of patients with repaired palates suffers from velopharyngeal incompetence (VPI) to some degree due to failure of the soft palate to achieve adequate contact with the posterior pharyngeal wall. This affects the intelligibility of speech.

The aim of surgical correction of VPI is to prevent nasal coupling to the oropharyngeal cavity during speech . Several techniques are described for lengthening of the soft palate or narrowing of the aperture of the nasopharynx .

As there is no absolutely satisfactory technique , surgical procedures for palate repair and VP correction have been evolving .

The aim of this study is to review the problems associated with VPI in relation to cleft palate and its repair. The surgical procedures for cleft palate, main complications, and evaluation of the effects of these procedures on speech, dentoskeletal growth and hearing are emphasized .

ANATOMICAL CONSIDERATIONS

Embryology of the Palate

The embryogensis of the palate can be divided into two separate phases : The formation of the primary palate followed by the formation of the secondary palate i.e the part of palate posterior to the incisive foramen.

Palatal development begins approximately at day 35 of gestation with the emergence of facial processes. These are the medial nasal processes, lateral nasal processes and the maxillary processes. ***(Nguyen and Sullivan., 1993)***

As a result of the medial growth of the maxillary prominences, the two medial nasal prominences merge to form intermaxillary segment ***(Patten., 1961)***. Among the 3 components of this segment, the palatal component forms the triangular primary palate. The primary palate is completely formed at the 7th week. ***(Delaire and Precious 1985).***

The main part of the palate is formed by two shelf-like growths from the maxillary prominences. These out growths, the palatine shelves, appear in the 6th week of development and are directed obliquely downward on either side of the tongue.

In the 7th week, the palatine shelves ascend to attain a horizontal position above the tongue and fuse with each other, thus forming the secondary palate. Anteriorly, the shelves fuse

with the triangular primary palate, and at the same time of fusion of palatine shelves, the nasal septum grows down and joins with the cephalic aspect of the newly formed palate.**(Sadler.,1990)**

The soft palate is formed by sequential merging of the growth centers in the caudal end of the palate shelves. At the beginning of this process, pqithelial cells of the opposing palatal shelves adhere to each other through formation of cell adhesion molecules and desmosomes ***(Ferguson, 1988).*** Even before contact is made, the epithelial cells appear to be programmed to self destruction by signals received from the underlying mesenchyme.**(Jones., 1993)**

The complete development of the palate is at the end of the ninth week ***(Delaire and Precious, 1985).***

The clefts of the primary palate are due to partial or complete lack of fusion of the maxillary prominence with the medial nasal prominence on one or both sides. The secondary palate cleft are caused by failure of the palatal shelves to fuse (or fuse incompletely) resulting in a velopalatine cleft which can be total (the entire secondary palate), partial, or limited to the uvula. A third category is formed by combination of clefts lying anterior and posterior to the incisive foramen.

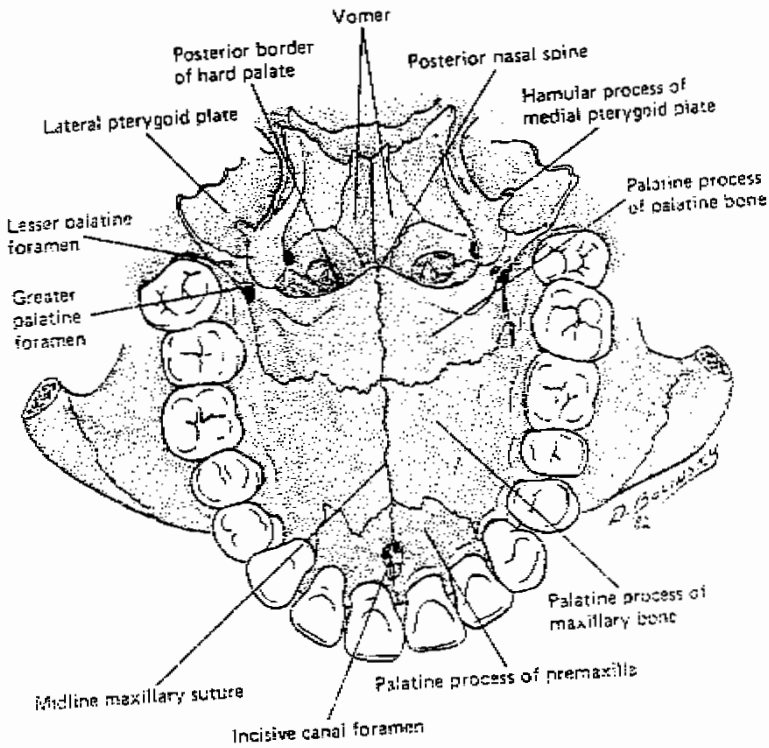

Fig .1 Anatomy of the hard palate.

Anatomy of the palate

(After Smith . 1983).

Anatomy of the palate

The palate is a complicated composite structure that forms the roof of the mouth, it separates the mouth from the nasal cavity and the isthmus of the fauces from the nasal part of the pharynx, and it extends into the cavity of the pharynx, forming a partial division between the oral and nasal parts of the pharynx. The palate is divided into the bony anterior hard palate and the mobile posterior soft palate

The hard palate :

The hard palate forms the anterior two-thirds of the palate and separates the nasal cavity as well as the paired maxillary sinuses from the oral cavity. Anteriorly and centrally, the hard palate consists of the premaxilla, which gives rise to incisor teeth; the premaxilla extends posteriorly to the incisive foramen. The major portion of the hard palate is made up of the palatine processes of the paired maxilla. The horizontal processes of the palatine bones form the posterior edge of the hard palate, whose posterior sharp and concave border shows a midline posterior nasal spine. The greater palatine foramen exists between the palatine and the maxillary plates and the lesser palatine foramen perforates the palatine bone itself (Fig.1).

Three partitions meet the superior surface of the palate: The septum in the midline, and the two medial walls of the maxillary sinuses laterally.

Mucous membrane:

The hard palate is covered on its oral surface with oral mucoperiosteal of varying thickness. ***Markus et al., (1993)*** divided this fibromucosal layer into 3 zones. Awarness of these zones will minimize the adverse effects of surgery. These zones are the fibromucosa of the palatal shelves, lining the palatal vault in the midline; The maxillary fibromucosa, lying between the midline palatal mucosa and the gingiva, and the gingival fibromucosa lying lateraly between the maxillary mucosa and the teeth. The palatal fibromucosa is very thin but thickenes laterally where the palate starts to curve downwards. At this point it merges into the adjacent maxillary zone which is thicker and contains a large amount of connective tissue. The maxillary fibromucosa is well-vascularized and contains the neurovascular bundle. The gingival fibromucosa lies laterally between the maxillary fibromucosa and the teeth. This varies in width being narrower in the region of the incisor and canine teeth and wider more posteriorly.

The mucous membrane of the anterior part of the hard palate is strongly adherent to the periosteum so that both layers cannot be stripped apart. However, these two layers can be stripped from the bone together. Fibrous tissue pegs (Sharpey's fibres) secure the attachement of the periosteum to the bone. Posteriorly, the mucous membrane and the periosteum are separated by a mass of mucous gland tissues. ***(Last ., 1984).***

The mucous surface is covered by stratified squamous epithelium but the thin upper (nasal) surface mucoperiosteum is

(Smith, 1983).

The soft palate :

The soft palate is a flexible muscular flap which is attached anteriorly to the posterior edge of the hard palate and extends posteroinferiorly into the pharyngeal cavity. Its free posterior border has a median conical projection of variable length, 5 to 25mm, the uvula, from the base of which a fold of mucous membrane containing muscle fibres sweeps down to the lateral wall of the oropharynx; this is the palatopharyngeal fold. The folds on both sides together form the palatophoryngeal arch. More anteriorly, a smaller fold, also containing muscles fibres, passes from the soft palate to the side of the tongue on each side; this is the palatoglossal fold. The two folds form palatoglossal arch which marks the junction of the buccal cavity and the oropharynx *(Beasely,1987).*

The basis of the soft palate is the triangular palatine aponeurosis which is formed by the expanded tendons of the tensor palati muscles that join in a median raphe (medial border). It is attached to the posterior edge of the hard palate and the crest of palatine bone (anterior border) while the posterolateral border blends with the side wall of the pharynx. The aponeurosis splits, near the midline, to enclose the uvular muscle; all the other muscles of the soft palate are attached to it. **(Last 1984).**

Mucous membrane :

The mucous membrane covers the entire soft palate. Pseudostratified ciliated columnar epithelium with goblet cells cover the nasopharyngeal aspect of the palate. Non-keratinising stratified squamous epithelium with few taste buds covers the oropharyngeal aspect of the soft palate as well as a transition area at the posterior part of superior aspect that comes in contact with Passavant's ridge. The lamina propria is very vascular and contains many elastic fibres. The thickness of the palate and uvula is occupied by a large mass of mucous and serous glands below the palatine aponeurosis. Scattered

(Beasely 1987).

The mucosa of the soft palate is yellow red in colour while that of the hard palate is pink. The nasopharyngeal mucosa is deep pink due to its thin mucosal covering and increased vascularity below the epithelium. ***(Smith, 1983).***