INTRODUCTION

Oronasal fistulae are well-known complications following surgery of the cleft palate. The incidence of postoperative fistula varies considerably in different series ranging from 0-50% ***(Millard, 1976)***. Symptoms related the palatal fistulae can quite significant because of embarrassing nasal regurgitation and interference with proper speech articulation ***(Shelton, 1984)***.

Recurrence rates following palatal fistula closure have been reported as high as 16% to 65% in various series respectively ***(Schultz, 1991)***. Secondary repair of recurrent oronasal fistula is one of the most challenging and difficult problems in the field of plastic surgery ***(Ashtiani, 2005)***.

Numerous techniques described for the closure of these communications testify to unsatisfactory results obtained. Most of these techniques have unpredictable results and may give equal degree of success and failure. The main reasons for the high failure rate have been ascribed to immobility of the scar, poor vascularity of the scarred tissue, tension on the flaps leading to impairment of the blood supply and inadequate trimming of scarred tissues ***(Tipton, 1970)***.

One of the important general principles in palatal fistula closure is not only to cover the oral side of the fistula. A double layer closure of the palatal fistula offers the best chance of

successful repair especially in large fistula. For nasal layer closure 'Hinge Flaps' Converse et al. works successfully in the mid palatal fistula but, it does not suite majority of the anteriorly placed fistula as the mucosa of the anterior palate easily disrupts, is difficult to mobilize and difficult to suture ***(Kirschner, 2006)***.

A labiobuccal flap raised away from the scarred tissue ideal for closure of the nasal layer in the anterior palatal fistula, provided the length of the flap matches the length of the defect and the presence of pass freely to the defect without undue tension ***(Pribaz, 1992)***.

A large oronasal fistula requires distant tissue for providing oral surface coverage because the local tissue is either too meager or unsatisfactory. The temporoparietal fascia pedicle flap has been used successfully for palatal defect the donor site is usually well hidden, but the dissection is tedious ***(Furnas, 1987)***.

Forehead and nasolabial flaps have been described for one stage reconstruction, but obtaining enough tissue from this area in a young patient is difficult especially when the fistula is wide. Moreover, the resultant donor area defect can be difficult to close and probably would be very noticeable ***(Govila, 1990)***.

The tongue is a richly vascularized organ that can provide a broad based pedicle flap ***(Pigoti, 1984)***, such a flap is associated with a relative lack of complications with high

success rate in children and adults alike. Residual tongue deformity is practically non-existent and the function remains unaffected. There is no increase in the morbidity, the only disadvantage of this procedure is that it needs two stages for completion.

AIM OF THE WORK

The aim of this study is to discuss the updated surgical methods of closure of palatal fistula.

Embryology

The embryological development of the palate may be subdivided into two phases:

- Primary palate: (i.e. primary at the time of formation).
- Secondary palate.

The primary palate (prolabium, premaxilla and upper 4 incisors anterior to the incisive foramen).

About the day 24 one can identify the fronto-nasal process, which is bounded on each side by the pair of the first arch-derived maxillary processes. By the day 28 the nasal placodes is evident as local ectodermal thickenings on either side of the inferior aspect of the fronto-nasal process.

The neural crest-derived mesenchyme soon condenses and proliferates at the borders of the placodes, the lateral and medial rims expanding outward to form a pit that becomes deeper as the lateral and medial processes grow. The maxillary processes at this time grow rapidly and soon approach each other and medial processes. The human primary palate forms at the bottom of the nasal pit with the meeting of the medial surface of the maxillary process and the lateral surface of the medial process.

This is soon followed by fusion of the lateral processes, more superiorly and anteriorly, with the medial process. All this

occurs between the 40th and 48th days of embryonic life. At the same time, the medial processes merge with each other to form the intermaxillary segment. This segment gives rise to the philtrum and the pre-maxilla, an area of the palate bounded by two lines from the incisive foramen to the alveolar bone between the lateral incisor and canine on each side ***(Moore, 2002)***.

The secondary palate (hard, soft palate, uvula and maxillary teeth posterior to the incisive foramen).

The human palate is derived from two projections from the paired maxillary processes of the first branchial arches, termed palatal shelves. Initially these shelves are in a vertical position on each side of the developing tongue, but as the mandible grows, the tongue moves downward and the shelves become more horizontal and grow toward each other. This happens in wave-like fashion in a postero-anterior direction under the control of an "intrinsic shelf force (Fig. 1) ***Walker & Frazer, (1956) and Burdi & Silivery, (1969)*** have shown that in males the shelves assume the horizontal position by the 7th week while in females; they are not horizontal until the middle of the 8th week. This implies that in the female, the secondary palate is open longer than in males, hence longer susceptibility to any teratogenic influences explaining the greater incidence of 2ry palatal clefts in females.

Once the palatal shelves are in the horizontal position they grow towards the midline and meet first at the incisive foramen then posteriorly as far as the uvula (fig.2). Consolidation of the contact occurs as mesoderm merges, one side with the other and the intervening epithelial cells die ***(Burdi, 1998)***.

Theories of the cleft palate:

As with the development of the 1ry palate, secondary palate formation is complex and there are numerous potential possibilities for development failure ***(Trasler and Fraser, 1997)***:

A structural abnormality with in the shelves preventing their movement to the horizontal position .

The shelves could be normal but too narrow and thus unable to meet each other in the midline.

A reduction in shelf force would bring about a delay in their movement to the horizontal.

Anything that interferes with the displacement of the tongue from between the shelves at the proper time.

Disproportionate growth of the head could make it wider than normal so that the shelves would fail to reach each other.

The epithelium of the shelves could fail to fuse for various cellular reasons. The pathogenesis of the human palatal clefting is certain to be heterogenous.

Malformation of the cleft lip and palate is not caused by a single factor. ***Fraser (1997)*** believed that the majority of cases are caused by multi factors, and that embryological and environmental factors are very important. These factors must act early during pregnancy, since palatal development is completed by 9-12 weeks of fetal life.

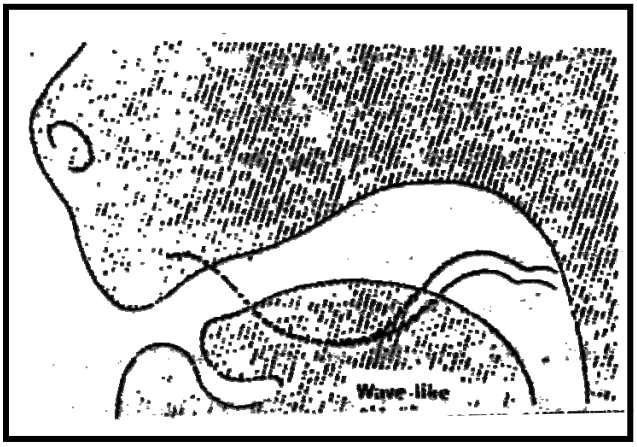

Figure 1: Horizontal movement of the palatal shelves in a wave like fashion ***(Smith, 2000)***.

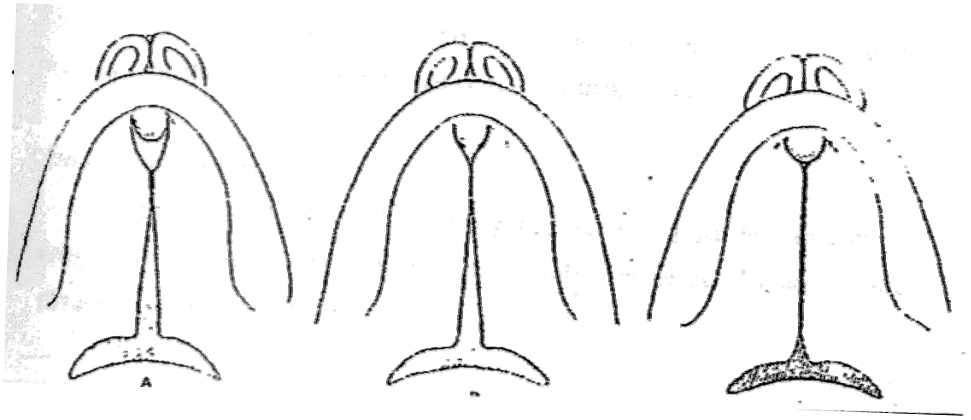

Figure 2: Anterior and posterior contact of the palatal shelves.

- ***These factors are:***

Certain drugs such as Valium have proved to cause clefts when given during early pregnancy ***(Safra and Oakley, 1976)***. Dilantin cytotoxic drug, methyldopa and anti-epileptic drugs are another examples ***(Friis, 1999)***.

Dietary regimen deficient in folic acid is another factor while increased doses of vit. A also produce clefts. If was also found that such activity was increased by cortisone and methyluracil. Smoking was reported by ***Khoury et al. (1998)*** to have an effect, the off springs of smoking mother were between 1.6 and 2 times more likely to have a cleft than those of non-smoking mothers.

Some diseases such as Rubella and epilepsy were proved to increase the incidence of clefts in the off springs if occurred during the first 3 months of pregnancy.

The genetic factors appear to be important, Frasae, divided the genetic factors underlying clefts into two major categories:

Mutant genes responsible for some clefts that are associated with some rare syndromes that may be autosomal dominant or recessive, An example of autosomal dominant type is ectodermal dysphasia.

Chromosomal aberrations occasionally involved in clefts that are associated with other more major congenital anomalies. Examples of this, is triosomy 13, where cleft lip and/or palate occur in 60-70% of triosomy 13.

Frequency: The incidence of cleft lip/palate (CL/P) by race is 2.1/1000 in Asians, 1/1000 in whites, and 0.41/1000 in blacks. Isolated cleft palate shows a relatively constant ratio of 0.45-0.5/1000 births. The foremost type of clefting is a bifid uvula, occurring in 2% of the population. The second most frequent type is a left unilateral complete cleft of the palate and prepalatal structures. Midline clefts of the soft palate and parts of the hard palate are also common. Complete clefts of the secondary palate are twice as common in females as in males while the reverse is true of velar clefts. About 7-13% of patients with isolated cleft lip and 11-14% of patients with CL/P have other anomalies at births.

(Moore, 2002).

Inheritance Rate:

Once child with a cleft is born into a family, the chance of clefting increases significantly in the subsequent siblings. These chances are higher when the first infant has cleft lip + cleft palate and lower when he has cleft palate alone. Also the incidence of clefts in children of the affected person is greatly increased compared to normal population (Table 1) ***(Lynch, 2001)***.

Table 1: Genetic risks in cleft lip and palate

Relationship to index case	Cleft lip/palate	Cleft palate
Siblings (overall risk)	4%	1.8%
Siblings (no other affected)	2.2%	-
Siblings (2 affected siblings)	10%	8%
Siblings and affected p arents	10%	-
Children	4.3%	6.2%
Second degree relatives	0.6%	-
Third degree relatives	0.3%	-
General population	0.1%	0.04%

(Prague, 2000, 14:235)

Anatomy

Warwick, Williams, and Romanes, stated that the complete palate is characteristic of mammals, and its development is associated with the ability to suck. It separates the mouth from the nasal cavity and extends backwards into the cavity of the pharynx, forming a partial division between the oral and nasal parts of the pharynx.

The Normal Palate consists of two parts:

A static part: (bony shelf) forming the anterior two thirds and termed the hard palate.

A dynamic part: forming the posterior 1/3 with a fibro-muscular basis termed the soft palate ***(Trier, 2001)***.

The Hard Palate ***(Fig. 3)***:

The bony palate is made up of the premaxilla, the palatine processes of the paired maxilla, and the paired horizontal palatine bones. The main mass of the hard palate is made by the palatal processes of the maxilla; posteriorly, the horizontal plates of the palatine bone complete the bony shelf, the posterior border of the hard palate is free, sharp and concave showing a projection in the middle line called the posterior nasal spine. The greater palatine foramen lies between the palatine bone and maxilla, while the lesser palatine foramen perforates the palatine bone itself ***(Sicher and Du Brul, 2003)***.

Three partitions meet the superior surface of the palate: vis, the septum in the midline, and the medial walls of the maxillary sinuses laterally ***(McMinn et al., 2001)***.

The Mucous Membrane:

The palate is covered on its oral surface with oral mucoperiosteum of varying thickness, the mucosal surface is covered with a stratified squamous epithelium ***(McMinn et al., 1999)***.

The mucous membrane of the anterior part of the hard palate is strongly united with the periosteum and the two cannot by stripped from each other, but together can be stripped from the bone, the attachment to bone is secured by multiple fibrous tissues pegs (Sharpey's fibres) that leave a finely pitted bone surface on the dried skull; posteriorly, the mucous membrane and periosteum are separated by a mass of mucous gland tissues.

No longer sharpey's flbres are needed and the bone surface is smoothly polished.

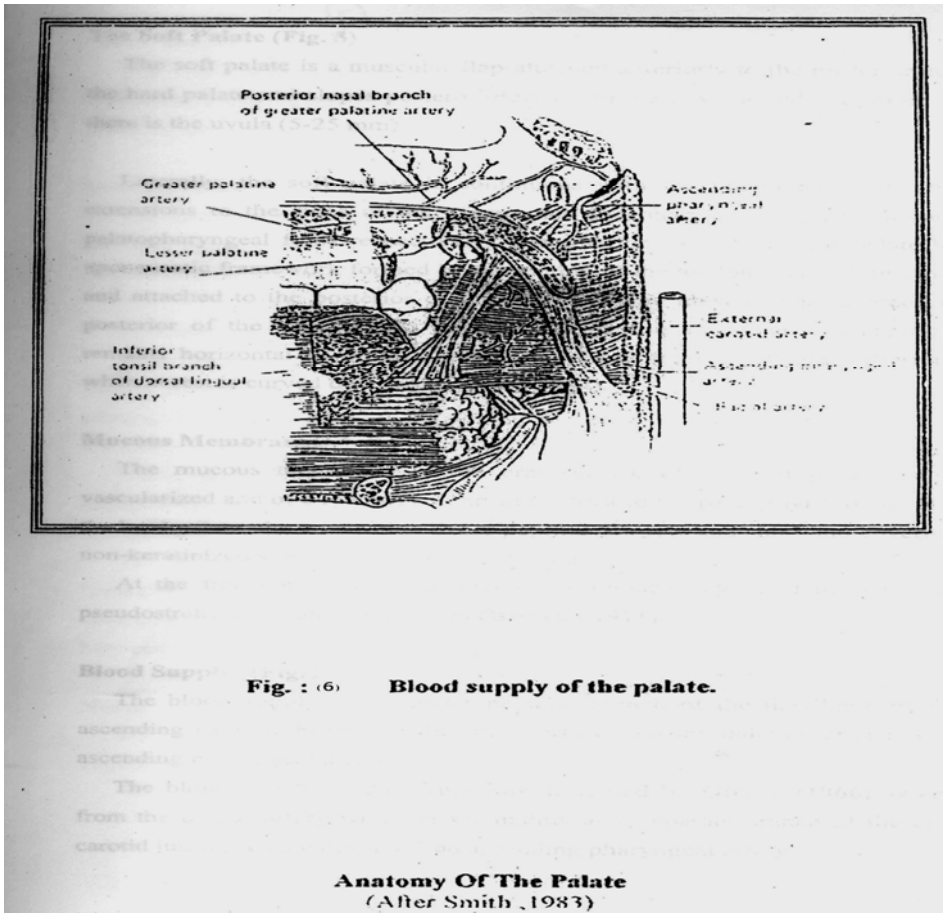

Figure 3: Anatomy of the palate ***(Thompson et al., 2005)***

- ***Blood Supply:***

The blood supply of the hard palate is provided by the greater palatine artery which arises from the 3rd part of maxillary artery. It emerges from the greater palatine foramen and passes around the palate to enter the incisive foramen and passes up to the nose. Veins accompany the artery back to the pterygoid plexus, other veins pass back to the supra-tonsillar region joining the pharyngeal plexus. Lymphatic return is along

side these latter veins to the retropharyngeal and deep cervical lymph nodes ***(Sinnatamby, 1999)***.

- ***Nerve Supply:***

The nerve supply is by the anterior palatine nerve (a branch of the maxillary nerve via the pterygo-palatine ganglion) as far forward as the incisive foramen, the anterior part of the palate, behind the incisor teeth (area of the premaxilla) is supplied by the two nasopalatine nerves from the same source ***(Sicher and Du Brul, 2002)***.

The Soft Palate ***(Fig. 3)***:

The soft palate is a muscular flap-attached anteriorly to the posterior edge of the hard palate and slopes postero-inferiorly to the few, curved margin on which there is the uvula (5-25 mm).

Laterally, the soft palate is continuous with the pharyngeal wall and has extensions to the sides of the tongue and the pharynx, the palatoglossus and palatopharyngeal folds respectively. The anterior part of the soft palate has an aponeurotic framework formed by the tendon of the tendons veli palatini muscles and attached to the posterior of the bony palatine muscles and attached to the posterior of the bony palate, the posterior part is muscular, the anterior part remains horizontal in position, while the more freely movable posterior part, when at rest is curved downwards ***(Sinnatamby, 1999)***.