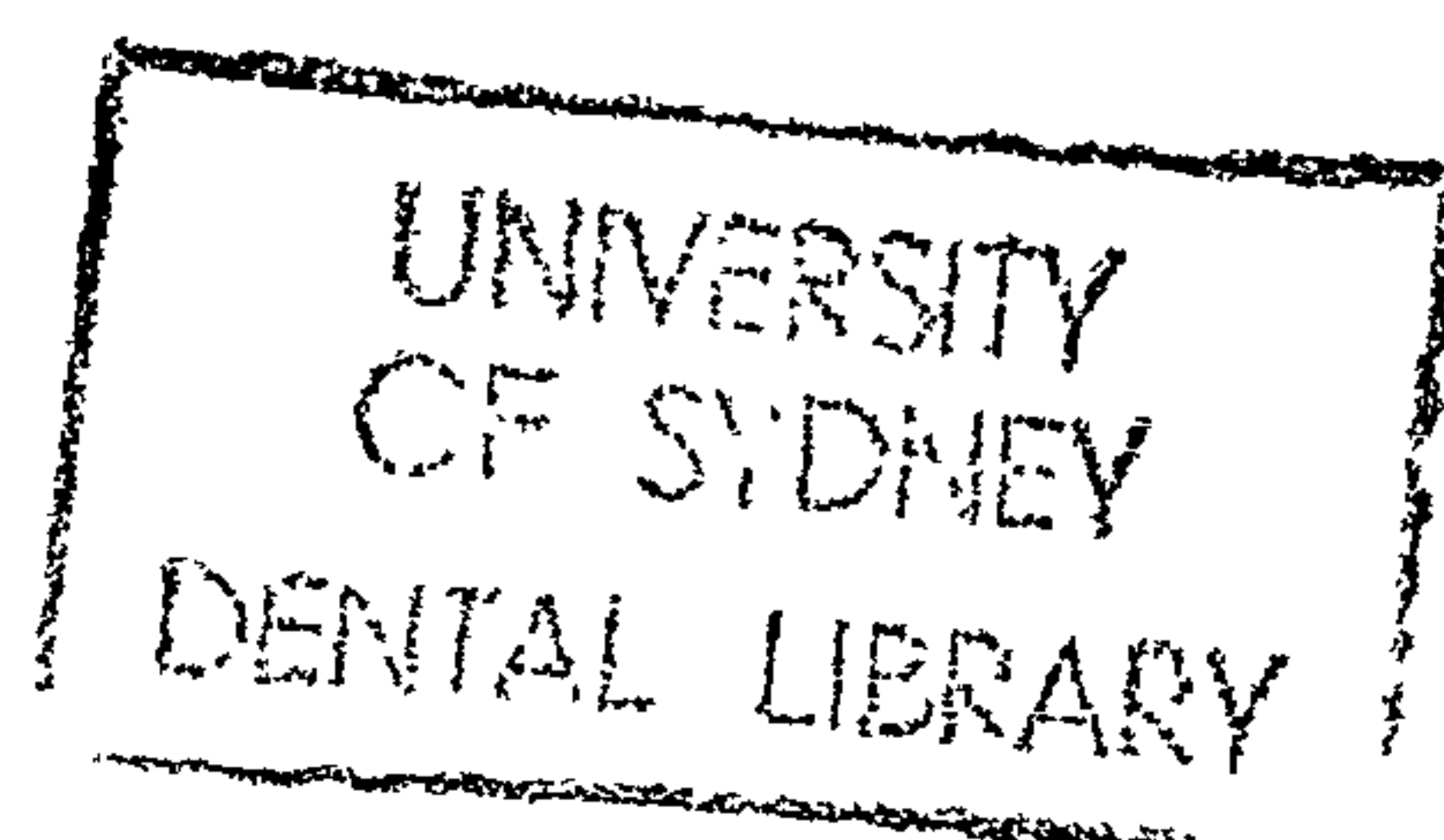


ORTHODONTIC ASSESSMENT OF PRIMARY SURGERY
IN UNILATERAL CLEFT LIP AND PALATE CHILDREN

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This thesis is submitted in partial fulfilment
of the requirements for the degree of Master of
Dental Science.

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1979.



ACKNOWLEDGEMENTS

The completion of this thesis was the result of valuable assistance from a number of people.

I am sincerely indebted to my supervisor, Associate Professor K. Godfrey (Department of Preventive Dentistry, University of Sydney), for his guidance and assistance during the course of this study.

My grateful appreciation is extended to Dr. D. Dey (Plastic Surgeon, Royal Alexandra Hospital for Children) for his encouragement, expert advice, permission to examine patients and records and the opportunity to observe his surgical procedures, the results of which form the basis of this thesis.

I also extend my thanks to the staff at the Royal Alexandra Hospital for Children, for allowing me access and use of patient records. I feel very privileged to have been able to observe and participate in the "Cleft Palate Team".

I would like to thank the Audio-Visual Department at the United Dental Hospital for their help in providing photographs.

My special thanks are also given to Miss Margaret Harop and Stephen Harop who have spent many hours assembling and typing the thesis.

Most importantly, I deeply thank my parents who provided me with the support, incentive and opportunity to further my education.

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SECTION 1

INTRODUCTION

A cleft of the lip and palate is a biologically imposed insult on the form of the skeletal maxillary jaw and on the function of the soft tissues in the facial, oral and pharyngeal regions.

Each child is born with the inherent potential for a specific amount and direction of facial growth, as well as the variables in the pattern of growth which may or may not be conducive to the development of a malocclusion. Superimposed on this there exist the additive and special problems associated with the presence of the cleft deformity and possible effects of surgery.

Current surgical techniques based on the understanding of facial growth have made the stereotype concave face of the cleft patient part of the past. This is partly a result of the co-ordinated efforts in many branches of medicine and improved methods for late reconstructions but mainly, it is a product of refining the procedure of primary repair.

There is considerable controversy concerning the proper treatment and management of children with clefts. In order to select the "best methods of treatment" it is essential for all active rehabilitation teams to examine their results periodically.

Recording the incidence and severity of the malocclusion can help to define the relevant problems and emphasize areas for improvement in reconstructive techniques. The data recorded could also be used to establish a base line which we can use to compare the efficacy of diverse treatment procedures.

The object of the present study was to evaluate and compare the occlusal results in a group of complete unilateral cleft lip and palate children where a variation of technique of primary surgery was introduced for anterior palate closure,

SECTION 2

REVIEW OF LITERATURE

Cleft Palate Mechanism

Experimental research has shown that formation of the face and associated intra-oral structures consists of a complete series of delicately balanced events involving extensive cell migrations and tissue interactions. These events are sensitive to a host of genetic and/or environmental influences. (Steffek, 1977.)

Most major malformations of the head and face are associated with the first branchial arch.

The medial, lateral nasal and maxillary processes are derived from the facial mesenchyme of neural-crest cell origin and these in turn are major contributors to the primary palate. (Johnston, 1966.)

At seven weeks the epithelium invaginates to form the nasal pits. Fusion of the epithelium of the medial and lateral nasal process subsequently occurs below the nasal pit and mesenchymal penetration follows. It is here at the region of the isthmus where clefts of primary palate (cleft lip) occur as represented in Fig. 1.

During the eighth and tenth weeks of gestation of the secondary palate formation, ridge-like shelves arise from the medial aspects of the maxillary process on either side of the tongue to become the palatal processes. Then two events occur: a rotation or translocation takes place and this is followed by a fusion of the paired palatal processes as represented in Fig. 2.

The classical theory of His described by Arey (1942) and Patten (1940) states that clefts are the result of failure of fusion or incomplete fusion of the palatal process and face buds. This theory does not explain simple clefts and clefts where epithelial bridges have remained post partum.

The "modern" hypothesis is described by Veau (1938), Tondury (1961), Politzer (1952) and Stark (1954) who felt that the defects are due to failure of the mesodermal tissue invasion into the epithelial wall. This theory explains not only the incomplete and bridged clefts but also the lack of other associated facial anomalies in the immediate area and the apparent regressive tendencies of some seemingly well-formed lips and palates.

North (1963) states that one should regard the process of mesodermal penetration as having been delayed but not wholly missing. There has only to be a slight delay for the process of invasion and fusion to be overtaken by that of splitting; and this provides a possible illustration of a method by which a cleft may be produced. Their theory does provide a possible explanation for the various degrees of incomplete cleft and the exact type of cleft.

The mechanism underlying the cleft lip was described by Brown (1971) as due to a reduced proliferation in migrating cranial neural crest cells resulting in an insufficient facial mesenchyme for successful fusion.

Local factors such as increased cell death interfere with fusion of the isthmus region and cleft lip occurs. (Lejour, 1970.)

Genetic predisposition can operate to cause the malformation in mice. (Trasler, 1968.) Exactly what occurs during the anatomical definition of primary palate is not yet entirely clear.

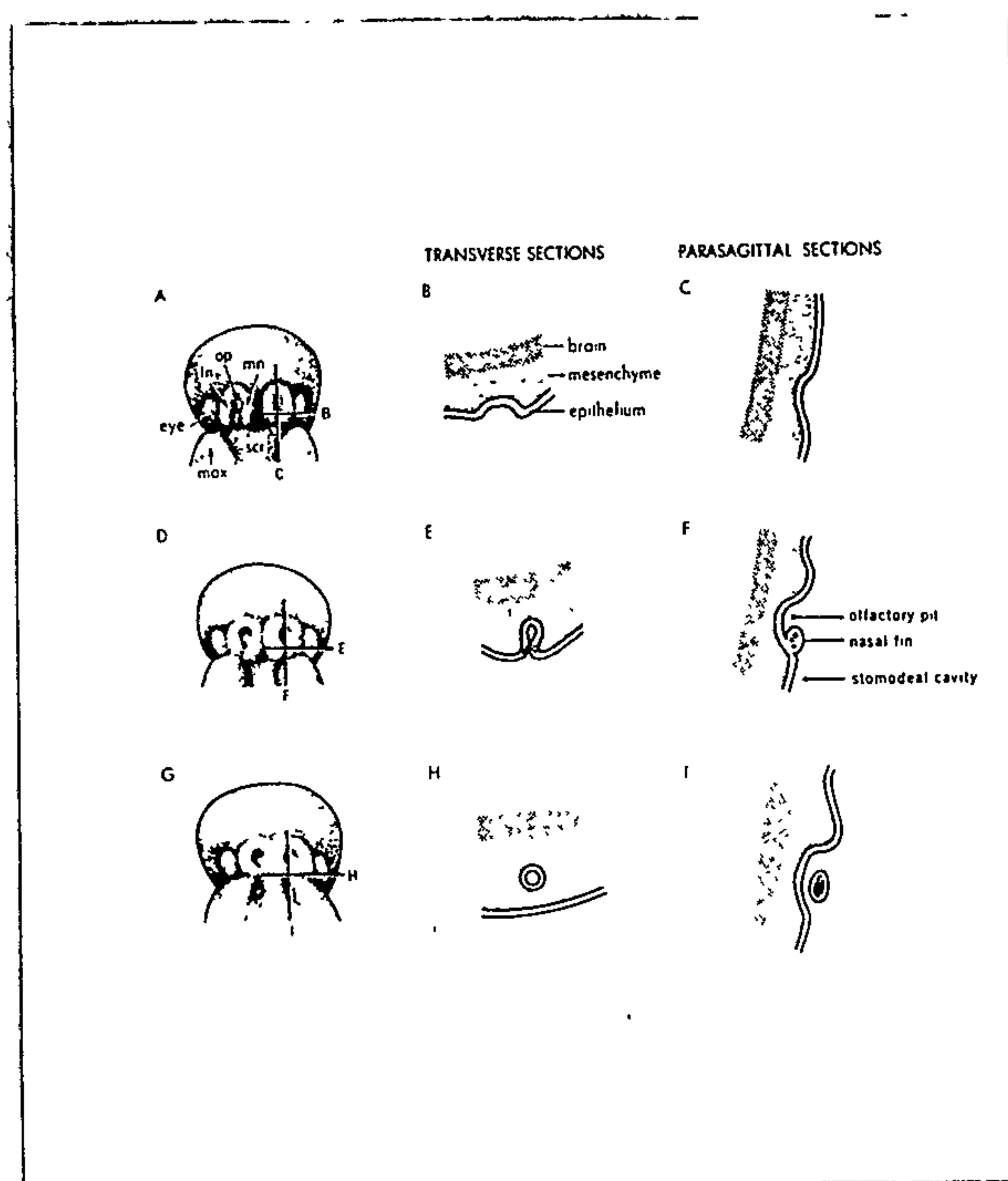


Fig. 1. Schematic diagrams illustrating primary palate development. A, D, G depict primary palate viewed from in front and below with mandibular arch removed. Structures indicated in A include: median nasal process (mn), lateral nasal process (ln), olfactory placode (op), maxillary process (max), and stomodeal cavity roof (scr). Section planes are indicated by broken lines. Transverse sections are illustrated in B, E, H, and parasagittal sections in C, F, I.

Folding process occurring in inferior portion of olfactory placode (B, E) brings epithelia of medial and lateral nasal processes into apposition (D,E) to form fused epithelial nasal fin or seam (F). Epithelial cell death (dots in E, F) resulted in dissolution of portion of epithelial seam. This portion is replaced by mesenchyme (H,I) so that mesenchymes of medial and lateral nasal processes become continuous below olfactory pit. Caviation process in epithelium separating olfactory pit and stomodeal cavity (F,I) completes formation of initial nasal passage. (Johnston, Hassell and Brown, 1975.)

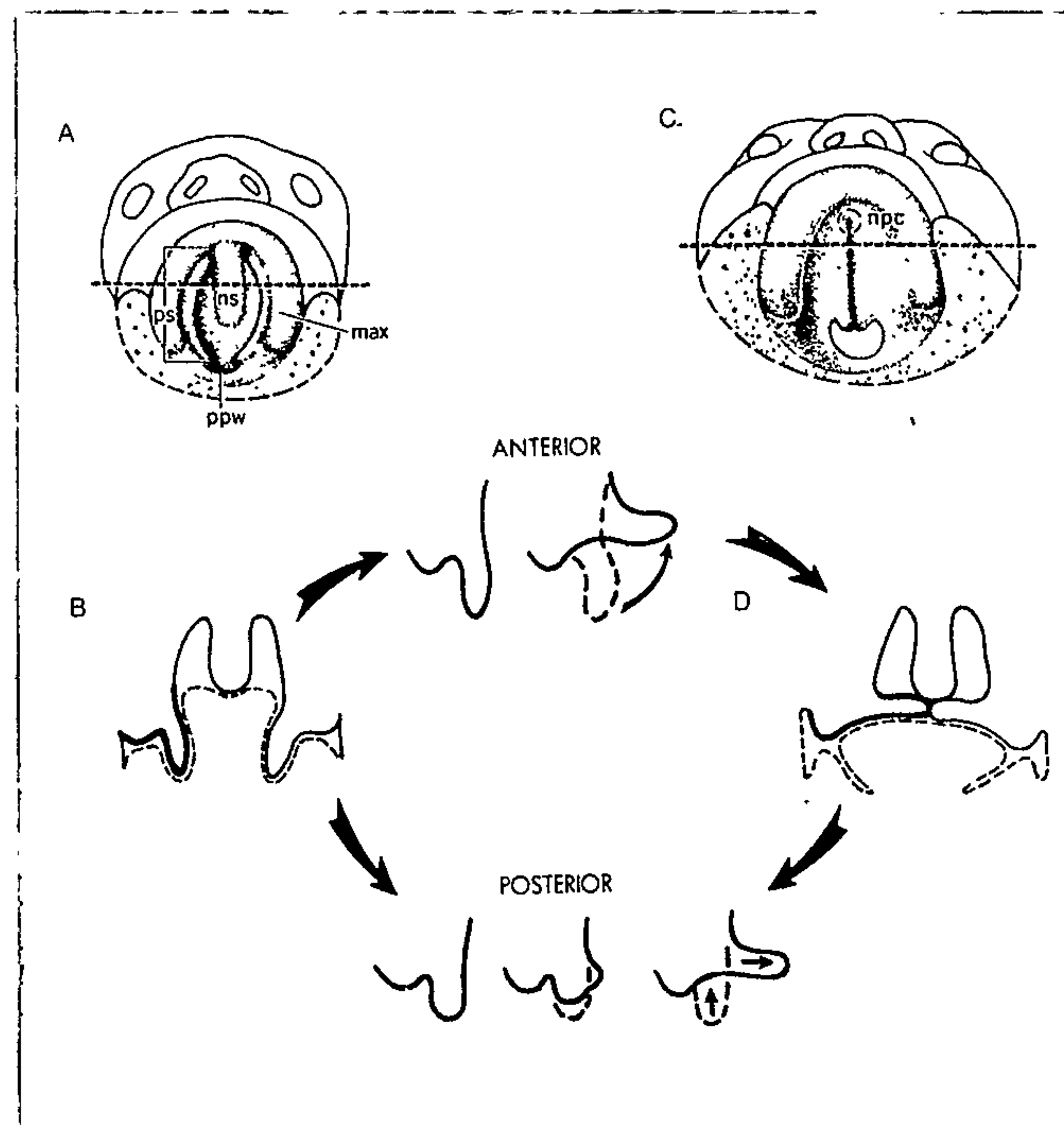


Fig. 2. Diagrammatic presentation of secondary palate formation. Mandible and tongue are removed in A and C and represented by broken lines in frontal sections (B,D). Anteroposterior extent of palatal shelf (ps) is indicated in A, as well as nasal septum (ns), basal maxilla (max), and posterior pharyngeal wall (ppw). Future position of nasopalatine canal is outlined by broken circle (npc) in C. Prior to rotation shelves grow vertically beside tongue (B). After rotation, shelves contact each other and inferior surface of nasal septum (C,D). Work conducted primarily on rat embryos indicates that elevation process varies considerably at different anteroposterior levels (see diagrams between B and D of elevating shelves indicated by heavy outlines). (Johnston, Hassell and Brown, 1975.)

Mechanisms underlying the cleft palate defect seem to fall under five categories:-

- (i) Inhibition of shelf growth. (Poswillo, 1975 and Steffek, King and Derr, 1967.)
- (ii) Failure or inhibition of palatal shelf rotation. (Fraser, 1961.)
- (iii) Failure of epithelial fusion of the palatal shelves. (Avery, 1965; Loevy, 1962; Hamilton, Boyd and Mossman, 1962; Latham, 1966; Subtelny, 1955 and Vij and Anagasuntheram, 1971.)
- (iv) Post-closure reopening. (Kitamura, 1966; Krauss, 1970 and Lejour, 1970.)
- (v) Failure of mesodermal penetration. (Stark, 1954.)

Much debate is currently in progress about what factor (or factors) provide the forces for palatal shelf transposition and the question of the mechanism involved remains unsolved.

A more significant finding occurred in regard to palatal shelf surface phenomena during contact and fusion phases of palatal closure in terms of adhesive surface coatings. Epithelial cells exhibited a "specific coating" immediately before contact of the shelves. Mato, Smiley and Dixon (1972), Pourtois (1970) in mice, and DePoala, Drummond and Miller (1975) in rabbits, showed increased glycoprotein synthesis in the palate was needed in shelf-to-shelf binding.

A histochemical and electron-microscopic study of human fetuses has been undertaken to establish whether portions of palatal epithelium have been programmed to break down at a specific time as shown by

Mato, et al (1972), Smiley and Koch (1972), Trasler and Fraser (1963) and Tsai and Verrusio (1977) or, as others have shown, namely, that contact between the shelves triggers the epithelial disruption. (Goss and Avery, 1975.) For fusion to occur two epithelial-covered mesenchymal tissues are needed. Goss, Bodner and Avery (1970) noted that the degree of fusion is approximately inversely proportional to the stages of differentiation of the mesenchymal tissue in the area of fusion. Pourtois (1967) and Trasler and Fraser (1963) postulated that in A-J mice the cleft palate occurred with cleft lip as the result of either a systemic alteration of the capability to fuse or as a mechanical consequence of cleft lip. The palatal shelves had the capability to fuse (as shown in vitro) but failed to do so because of the increased width of the face or underdevelopment of shelves associated with cleft lip. Pourtois subsequently noted that there was a time limit such that fusion will not occur if the epithelium came into contact either too late or too early.

Another area of recent research is concerned with the spatial arrangement of structures in the oral cavity at the time of palatal closure. Fraser (1969) suggested that the stage at which the palatal shelves move, may be influenced by:-

- (i) an intrinsic shelf force,
- (ii) the mechanical effect of changes in the cranial base,
- (iii) the width of head,
- (iv) the width of shelf,
- (v) the length of head,
- (vi) the forward movement of the jaw and hence the tongue,
- (vii) the size and mobility of tongue,
- (viii) the epithelial competence to fuse,
- (ix) the pharyngeal musculature, and
- (x) the neck extension.

Mutant genes or teratogens could interfere with palatal closure by acting at any one of these points.. In Man, a cleft palate may result from minor changes individually indistinguishable at several points in the system, all interacting to delay shelf movement beyond the threshold.. Fraser introduced the term "Multifactorial Threshold System" to describe the above.

Teratogens have been used to demonstrate key events and structure involved in palatogenesis and also to show alterations in structure contiguous with the palate. For example, what is the role of the embryonic chondrocranium including the cranial base and nasal capsular cartilage in the formation of the palate?

Harris (1967) and Verrusio (1970) noted in mice that the events prior to and during palate closure coincided with a straightening of a "kinked" presphenoid segment. Long, Larsson and Lohmander (1973) hypothesized that teratogens which produce clefts in the A-J mouse also inhibited cranial base straightening that is essential for upward movement and contact of the palatal shelves.

Diewert (1976) showed a positive correlation between alterations in palatal blood vessels and palatal clefts induced in rat fetuses with such teratogens as cortisone, Vitamin A and 6-amino-nicotinamide. Other teratogens such as lithium tranquilizers, cortisone, lathyrogenic compounds, viruses and antibodies have been shown to cause clefts by various workers.

Advances experimentally have indeed been made in associating altered events during critical periods of palatogenesis with clefts. Yet, primary mechanisms and primary loci of deviant growth still remain unsolved even with contemporary technology. (Burdi, 1977.)

Epidemiology

The majority of spontaneously occurring malformations result from an interplay between many genetic and environmental factors rather than a single factor. (Kalter, 1954 and Fraser, Walker and Trasler, 1957.)

Only the effect of genetic factors have so far been shown in humans. The mode of inheritance is still not clear for cleft lip and palate malformation. There is evidence of recessive character but dominance occurs.

Several analytical studies have suggested associations between certain factors and orofacial clefts but none of these have been firmly confirmed. Some of the factors are:-

- (i) Maternal consumption of therapeutic drugs which produce nausea. (Richards, 1972 and Drillien, Ingram and Wilkinson, 1966.)
- (ii) Miscellaneous drugs, including antiemetics. (Richards, 1969.)
- (iii) Maternal bleeding. (Drillien, et al, 1966 and Fraser, 1970.)
- (iv) Toxaemia. (Fraser, 1970.)
- (v) Increased maternal antagonism to insulin. (Vallance-Owen, Braithwaite, Wilson, Edwards and Maurice, 1967.)
- (vi) Toxoplasmosis antibodies. (Erdelyi, 1957.)
- (vii) Antiepileptic drugs. (Pashayan, Pruzansky and Pruzansky, 1971.)
- (viii) Diet deficiency. (Fögh-Anderson, 1955.)

A further study of the possible relationship of nutrition and biochemistry to clefts (especially during pregnancies of women who have already had affected children) could well be rewarding, as it

might be possible, by suitable treatment, to prevent clefts in known high-risk cases. (Hibbard and Smithells, 1965 and Langman, 1961.)

To date it seems fairly well established that cleft lip (with or without associated cleft palate) and isolated cleft palate are embryologically and genetically two different and independent disorders. (Fögh-Anderson, 1955, and Spriestersbach, Dickson, Fraser, Horowitz, McWilliams, Paradise and Randall, 1973.)

Saxen and Lahti (1974), working on a Finnish sample, have shown that environmental factors seem to be more closely associated with the production of cleft lip and palate cases, while in the cleft palate case alone, the genetic factors may be more involved.

Therapeutic drugs and exposure to the influenza viruses during pregnancy were the more-recent environmental factors considered in reaching this conclusion.

In a recent epidemiologic and experimental study on the etiologic variables in cleft lip and palate (Saxen, 1975), the concept of the classical multifactorial model in the production of mammalian oral clefts was generally supported. This study was based on a study of 599 cases of oral cleft.

Incidence of Clefts

Spry and Nugent (1975) found between the period of 1949 to 1968 the incidence of cleft cases in South Australia was 1.41 per 1000 (1 : 711) live births. This figure corresponds with that of 1.42 per 1000 in Northumberland and Durham, England (Knox and Braithwaite, 1963), 1.51 per 1000 in the South West region of England (Campbell Wilson, 1972), 1.66 per 1000 in Tasmania (Rank and Thomson, 1960) and 1.21 per 1000 reported by Chi and Godfrey (1970) in their N.S.W. hospital study for the years 1964 - 1966.

However, Brogan (1973) found that for the period 1971 to 1972 there had been a marked reduction in the number of babies with cleft lip and palate deformities in Western Australia. He understood that a similar decline may have been observed in other units in Australia and Europe. The following table shows the figures for Western Australia over the last four years converted to a rate per 100000 births.

Table 1 : Incidence of cleft lip and palate deformities in Western Australia per 100000 births.

Year	Cleft Lip	Cleft Palate
1969	150	85
1970	108	61
1971	95	86
1972	56	35

However, Fara (1972) found the incidence of clefts in Czechoslovakia to be on the increase. In 1970 it reached almost 2 per 1000 of all newborn children.

Current statistics reveal that approximately 1.43 per 1000 births in the United States has a cleft lip or cleft palate. (Steffek, 1977.)

Chi and Godfrey (1970), Zilberman (1973) and Spry and Nugent (1975) showed that left-sided unilateral defects were almost twice as common as right-sided unilaterals. Further, Spry and Nugent found that overall cleft lip and palate was twice as common as cleft palate. There were almost twice as many males as females with cleft lip and palate, while the majority of cases with cleft palate were females. They also observed that unilateral cleft lip and palate was nearly three times as common as the more severe bilateral cleft lip and palate.

Chi and Godfrey found that there is a higher relationship between the isolated cleft palate and other body malformations than exists for cleft lip and palate.

Growth in Cleft Lip and Palate Patients

One of the most perplexing, yet fundamental, problems encountered in the habilitation of patients with congenital cleft palate seems to be the effect of primary palate surgery on subsequent growth and development of the craniofacial complex. Graber (1949) has drawn attention to traumatic and/or repeated surgery which has caused many critical re-evaluations of surgical procedures being used and their effects on growth.

Muir (1974) notes that in the past it has been difficult to know how much of the changes in facial growth after birth and after operative treatment were due to the initial deformity and how much due to the effects of treatment. In general, it may be said that the operative repair creates forces which tend to improve the alignment of the underlying skeleton but that the deformity, due to lack of bone, will persist. In so far as the upper dental arch is concerned, a properly executed operation creates forces which tend to improve the shape of the arch anteriorly. However, in the region of the premolars a repair of the cleft creates unfavourable forces which tend to cause contraction of the arch.

On reviewing some of the research carried out on cleft palate patients, contradictory results have arisen and this necessitates stringent attention to all relevant variables. Several aspects must therefore be considered in any evaluation of maxillary growth. Pinkerton, Olin and Meredith (1966) felt that these inconsistencies are due to the following:-

- (i) poor sample selection may account for many of the discrepancies,
- (ii) studies report data on a group of subjects who vary widely in age,
- (iii) groups mixed as to type of cleft,
- (iv) majority of reports give no history of treatment, and
- (v) lack of an adequate control population.

Also consideration is required as to the method of analysis used by the researcher and the possible source of errors, whether it be using:-

- (i) Cephalometric methods.
- (ii) Frontal plane tomography. (Harvold, 1954 and Coupe and Subtelny, 1960.)
- (iii) Three-dimensional analysis of casts utilizing stereophotogrammetry. (Berkowitz, Krischer and Pruzansky, 1974.)
- (iv) Models which show details of maxillo-facial relationship in three dimensions. (Wada and Miyazaki, 1975.)

However, all these methods are extremely valuable clinical tools both in describing the facial patterns and in documenting the natural history of children with cleft defects.

Dickson, Grant, Sicher, Dubrui and Paltan (1974) have stated,

".....before one can attempt to summarize aberrations in growth and development a precise understanding of the normal processes is important."

It is also noted that,

"....data concerning the identification, description and study of maxillary growth aberrations will automatically be limited to an extent directly related to the degree of agreement and/or disagreement about the normal processes."

The aspects of normal maxillary growth about which investigators seem generally agreed, are:-

- (i) Scott (1953)
Cartilaginous nasal septum seems to be involved in maxillary growth and development in the first six years of life.

- (ii) Enlow and Bang (1965) .
Appositional growth in the tuberosity region seems to be an important aspect of antero-posterior development of the maxilla.
- (iii) Moss' "functional matrix" theory.
The integrity of intact functioning intra-oral, circumoral and circum-pharyngeal soft and bony tissue is necessary in order to have concomitant normal maxillary development.
- (iv) Singh and Savara (1966) and Lebert (1962) .
The development of the maxillary alveolar process and increases in maxillary vertical dimensions are directly related to the presence and normal eruption of the teeth.
- (v) Sillman (1964), Scott (1959) and Krogman and Sassouni, (1957).
Maxillary growth is extremely prolific from birth to age two or three and again circumpubercally, being about 90% complete by age seven, with mostly remodelling, resorption and deposition thereafter.

The above aspects represent important features integral to the understanding of the literature concerning the effect of palatal surgery on maxillary growth.

Cleft Pattern Formation

Latham (1969) observed that deformity first appeared in the 6th week and increased rapidly in severity until about 12th week, thereafter progressing more slowly. He observed in unilateral conditions of clefts the associated deformity was characterized by:-

- (i) Maxillary displacement.
- (ii) Premaxillary distortion.
- (iii) Malformation of the nose.

Wada and Miyazaki (1975) emphasized that downward growth inhibition of the maxilla occurred under conditions of premaxillary protrusion, lateral segmental displacement and segmental rotation towards the cleft in the anterior alveolar region. Latham (1969) explained this pattern formation by firstly illustrating the two normal maxillary growth mechanisms. The first mechanism commences with almost simultaneous chondrification of the nasal septum and appearance of the maxillary and premaxillary ossification centres. The latter centre becomes connected to the nasal septum by the anterior septopremaxillary ligament and are thereby pulled forward so that the developing upper jaw shares in the general forward growth of the cartilage making up the embryonic facial skeleton. The second mechanism becomes active at about middle fetal life and may be explained in terms of the intrinsic ability of the maxilla for growth upwards and backwards on their free orbital and posterior surfaces.

According to Latham these mechanisms for the unilateral cleft lip and palate cases are,

".....that the septopremaxillary ligament dictates the nature of the septal deviation and final deformity was characterized by an upward tilting premaxillary region which appeared to result from interference by the bent nasal septum with subsequent downward displacement of the maxilla by an intrinsic mechanism".

Thus the nasal septum plays a role in premaxillary growth and this deviation must be the cause of reduced maxillary height. This prenatal growth mechanism still continues up to the age of six months even with the lateral pulling force of the orbicularis oris muscle and of other oral functions prominent after birth.

Wada and Miyazaki (1975) followed the child through after lip and palate repair to the age of 4 years. They observed that antero-posterior growth inhibition of the maxilla occurred just after lip repair and that this difference from the normal was not compensated for by the age of 4 years. From the standpoint of the growth mechanism, this feature must be due to the three-dimensional bending of the premaxilla by the moulding action of the lip. This was also observed by Wood (1972).

Wada and Miyazaki's study supports the theories of Scott (1953) regarding the importance of the nasal septum in maxillary growth and development in the first six years of life.

Role of Nasal Septum

Latham (1968, 1969) and Robertson (1973) tried to emphasize that in later stages of the prenatal period the maxilla shows an intrinsic capacity for bone formation on the orbital and posterior free surfaces, moving in a relatively normal manner downward and forwards with neither the nasal septum nor growth in the sutures playing a significant role.

Bergland (1973) suggested that lateral segments disconnected from the nasal septum are underdeveloped because they are deprived of a growth thrust exerted by the nasal septum. There is an unbalanced growth force driving the non-cleft maxilla laterally and inferiorly initiating compensatory growth at the zygomatico-maxillary suture. (Latham and Burston, 1964.)

The above can be used to explain inferio-laterally inclined maxillae and the asymmetry mentioned by Subtelny (1955) and Coupe and Subtelny (1960) being commonly associated with complete unilateral cleft cases.

Bergland (1973) using a tomographic x-ray method to examine a relatively rare type of bilateral complete cleft in the primary palate combined with an apparently normal secondary palate, revealed no junction between the nasal septum and the secondary palate, yet the maxilla developed within normal limits. The overdevelopment of the nasal septum so frequently seen at birth is not real overgrowth but rather premature release of a relatively normal inherent growth potential. In contrast, the lateral maxillary segments follow an apparently normal growth rate. The clinical problems originate from an unsynchronised growth of various facial structures and this has to be taken into consideration in planning treatment for these cases.

There still exists a lot of controversy as to the role of the nasal septal cartilage in midfacial development. Experiments by Stenström and Thilander (1970), Kvinnsland and Briestein (1973) and Searls (1976) have given contradictory results. We are still left with the unresolved question of whether the growth sites identified in Searls' study on rats create an active thrusting force or are mere compensatory responses to midfacial movement of the surrounding structures.

Tissue Deficiency

There is considerable evidence implicating mesenchymal deficiency as the underlying problem in most cases. Studies of embryos subsequent to the clefting process indicate that the development of the bony maxillary complex is affected, showing both overall retardation and deficiency. (Stark, 1954.) Hence a cleft is usually more than a separation of normal bone - it is also deficiencies of alveolar and maxillary bone in clefts of the anterior maxilla. (Stark and Ehrmann, 1958.)

Ross (1970) questioned whether the growth problems are related to a primary deficiency of tissue or to a lack of growth potential.

Avery and Devine (1959), Stark (1954) and Kraus and Ahern (1966) have stated that while there is evidence of a mild or moderate embryonic deficiency of tissue in the maxillary complex, the deficiency is eliminated by birth in the majority of cases so that compensatory changes can occur.

The question of the quantity of bone in the hard palate has produced some controversy. Some authors consider the palatal shelves to be normal but separated, while the following claim that the shelf width is less:- Coupe and Subtelny (1960), Huddart, MacCauley and David (1969), Atherton (1967) and Peyton (1934.)

Peyton (1931) after measuring casts of 91 normal and 57 cleft lip and palates concluded that:

".....there was no evidence of significant difference between both groups at one year of age; apparent difference in tissue deficiency was noted at 3 years."

Dorrance and Bransfield (1943) stated that soft palate deficiency is directly related to that of hard palate deficiency. This correlation

has not yet been definitely established but may indicate why some clefts are more difficult to close as compared to others.

Grabner (1949) stated that growth of maxilla was deficient in all dimensions, that is, laterally, vertically and antero-posteriorly.

Coupe and Subtelny (1960) using frontal laminagraphic x-ray technique on 120 cleft children between the age of 1-3 years concluded that there was a tendency for palatal tissue deficiency and the extent of this deficiency was related to cleft type. Deficiency and displacement of osseous tissue was greatest in bilateral clefts and in unilateral clefts. However, some individual cleft palate cases did not show any actual deficiency in palatal tissue. Therefore, we should evaluate each case on an individual basis.

Tsuji (1966) observed in adult cleft models a growth inhibition in the anterior alveolar region especially in the premaxillary region. He questioned the cause of this inhibition. While it is possible that the cause of this growth inhibition is related to a cleft lip and palate, it is unknown whether this is due to a deficiency of tissue, to a segmental displacement or to a lack of arrest of growth potential.

Huddart, MacCauley and Davis (1969) observed that:

".....in unilateral cases due to the repositioning of the lesser segment a greater amount of tissue posterior to the posterior palatal plane. This implies that there is greater formation of tissue anteriorly than posteriorly in cleft lip and palate, but this tissue is displaced away from the front of the mouth, thereby increasing the width of the alveolar cleft. This makes more tissue available posteriorly for the creation of a long functional soft palate thereby perhaps aiding speech in contrast to cases of cleft palate only, where segmental retroposition cannot occur."

Huddart (1979) observed that the width of palatal tissue at birth was deficient by approximately 22%. There was also a deficiency in the area of palatal mucosa which ranged from the equivalent of 2.5% to 32.2% of the area of normal palate. The mean deficiency was 16.8%. However, with growth it was noted that tissue deficiency dropped to 9.5% at 4 months. Also he noted that the alveolar arch length of the intact side was significantly less than normal at birth, but the length on the cleft side was approximately equal to that found in the normal.

Atherton (1967) believed the lateral segment of the cleft maxilla may be underdeveloped in the antero-posterior length because it was disconnected from the nasal septum. Berland (1967) found this not to be true.

Robertson (1973) observed in cleft cases that there were deficiency and non-deficiency types in relation to the alveolar ridge area. He observed that risk of collapse is greatest in those cases where there is marked deficiency of tissue, which occurs in 1 out of 4 complete clefts.

Chierici, Harvold and Vagervik (1973) stated that we should differentiate between primary defect and the adaptational responses of normal structures to an abnormal environment. At birth, defects may be difficult to distinguish. Those contributions to facial morphology which are due to inadequate cell potential should be distinguished from those which are simply normal responses to certain distortion and dysfunctions in related systems. If this difference can be made it may be possible to focus attention on the primary defect and to prevent development of secondary deformity.

Current evidence points to a mild hypoplasia of the maxillary complex in most cases which tends to lessen somewhat during later development. Significant bony deficiencies and associated deformity tends to be generally limited to the immediate region of the cleft.

Segmental Displacement

All the studies share in common the question whether the growth inhibition in relation to cleft lip and palate are due to either:-

- (i) deficiency of tissue,
- (ii) segmental displacement,
- (iii) lack or arrest of growth potential, or
- (iv) a combination of all the above.

An accurate description of maxillary displacement and/or growth deficiencies in unoperated cleft palate patients allows us to observe not only the expected normal growth pattern but also the growth patterns that may later be influenced by surgical intervention. It may also offer some insight as to which type of growth inhibition might be inherited or biomechanically compensatory in origin and which may be iatrogenic.

Bishara, Krause, Olin, Weston, Van Hess and Felling (1976) considered that with respect to maxillofacial growth and development in cleft lip and palate patients it may be helpful to pursue the following questions:-

- (i) Does the unoperated cleft individual have the same maxillofacial growth potential as the non-cleft individual?
- (ii) Do all unoperated cleft types have the same growth potential?
- (iii) What effects do cleft management have upon growth?

Innis (1962), in examining unrepaired clefts of the North Borneo natives, found:

".....a degree of medial collapse of the lateral alveolar segment associated with diminished downward vertical growth of the alveolus, resulting in crossbites lateral and/or anterior open bite."

He felt that there was diminished growth potential in the cleft area and believed that this deformity will occur even if closure was attempted.

Ortiz-Monasterio, Olmedo, Trigos, Yudovich, Velazques and Fuente-del-campo (1974) showed that unoperated complete cleft adults had good facial growth although the palatal segments were displaced laterally. Mestre, Dejesus and Subtelny (1960) noted good potential for normal growth in their unoperated Puerto-Rican adults.

Bishara (1973) observed in unoperated clefts that although the palates were geometrically displaced, they had developed within normal limits. Peat (1974) indicated that neonatal cleft palate, when it involves the lip, can be displaced either laterally or medially and only rarely may be deficient in mass. Wada and Miyazaki (1975) observed also that cleft neonatal palate size was within normal limits in all three dimensions.

Berkowitz, Kirscher and Pruzansky (1974), measuring casts three-dimensionally, demonstrated that cleft palate was geometrically distorted in all frames of space and in complete clefts the lateral segments were laterally displaced. Troutman (1974), without explaining the mechanism, concluded that some cases of complete clefts had collapsed arches.

Van Limborgh (1964) studied cleft skulls at embryo, foetal, new-born and adult stages. He concluded that the growth process is slower

than in normal individuals but the growth potentials are essentially the same except in the region of the cleft. He didn't know whether this was a temporary or permanent retardation in growth and whether this would be compensated for by a prolonged period of growth or by an increased rate of growth at a later age. He noted that skeletal structures ultimately reached their normal dimension. The deviations of the various bony structures observed are not primary but secondary in nature. He observed a pattern in which these deformities became more and more constant in various types of clefts and the factors governing them must be characterized by their increasing importance during life. One such factor might be the absence of certain areas or centres of growth. However, the relative importance of muscular and masticatory forces is questioned.

Examining repaired clefts, Harvold (1954) noted that asymmetry and deformity of the facial skeleton is localized mainly in the alveolar and palatal processes and in the midsection, i.e., the nasal septum and premaxilla. This abnormal tissue which occurs early in foetal life has established a particular pattern of growth and development but later alterations in environment will further modify this pattern. Harvold stated that the major portion of the deformities seen in cleft lip and palate cases cannot be due to reduced growth potentials - it is not clear how he arrived at this conclusion. The narrow arches may be attributed to a change in maxillary bone position and a concomitant inhibition of vertical alveolar growth.

Ross (1970) agrees with the observation that severe distortion of the maxillary complex noted at birth is due to muscular imbalance (in accord with Pruzansky, 1955; Slaughter and Brodie, 1949; and Subtelny, 1961) and nasal septum growth (in accord with Latham and Burston, 1964) and not to intrinsic inadequacy.

Huddart, MacCauley and Davis (1969) and Subtelny (1955) have demonstrated that the posterior maxilla and adjacent structure are wider than normal in children with complete clefts. There is good evidence from Moss (1965), Ross and Coupe (1965) and many others that this excess width involves other areas of the face including the orbits, the zygomatic bone, mandible and nasal cavity. While these may be partially intrinsically derived developmental factors, there are secondary factors which contribute and which may be completely responsible.

Atherton (1967) analyzed maxillary development of 17 skulls with complete unoperated cleft lip and palate from birth to old age. Noted in the infant specimens were marked laterally inclined maxillary bones and pterygoid plates which tend to decrease to normal in older specimens. The bones on the cleft sides were smaller in all three dimensions. In some cases, he noted normal or greater than normal vertical growth on the cleft side (except for the canine region). This was contrasted to less vertical development on the non-cleft side, an occurrence perhaps related to the deviant direction of the septovomer structures.

Pruzansky (1953) and Subtelny (1964) noted differences in width and extent of the cleft in terms of the anatomic structures involved. In some instances width of cleft may be related to a displacement of tissue rather than an absence of tissue.

Robertson (1973) states that the initial deformity is the result of displacement of jaw segments and in which absence of tissue plays a secondary role.

Pruzansky (1953, 1973) observed that not all cases of congenital cleft lip and palate were alike. In his analysis of deformity he considered:-

- (i) the adequacy of parts,
- (ii) the distortion of parts,
- (iii) the geometric relationships of parts, and
- (iv) the relationship to contiguous anatomic structures.

Genetic and General Make-up of the Child

Congenital malformations are conditions, the origins of which we usually do not know and can determine only rarely. With subsequent development the conditions may undergo qualitative and quantitative alterations. We still do not know whether changes in cleft palate patients are due to the cleft or to genetic predisposition of the cleft. (Ross, 1975) However, genetic and developmental differences between all individuals (whether normal or abnormal) are present and affect the expression of the cleft.

Subtelny (1974) points out that cleft lip and palate is a biologically imposed "insult" on the form of the skeletal maxillary jaw and on the function of the soft tissue in the facial, oral and pharyngeal regions. It is a cleft in the maxillary jaw and some of the contiguous musculature and not solely a cleft in the dental arch. As such, a child is born with potential problems related to the cleft itself. Superimposed on this, he is born with the inherent potential for a specific amount and direction of facial growth as well as variables in the pattern of growth which may or may not be conducive to the development of a malocclusion. We have learnt that just as all clefts are not alike, all growth patterns in cleft lip and palate individuals are not alike. The cleft children seem to have a genetic tendency for some differences in growth potentials in the mandibular as well as the maxillary jaw. The growth in these patients seems to be restricted to an antero-posterior direction. However, deficiency of growth may also occur simultaneously in the vertical direction - these patients show a tendency to an open bite as growth progresses into the later stages of the facial growth span. This can be one of the limitations in treatment imposed by the pattern of facial growth in some of these individuals. Therefore, we must consider the genetically and/or functionally imposed controls.

Pruzansky (1955) stated that:

".....children with clefts are endowed with inherent potentialities for growth and development that reflect their genetic heritage and the metabolic climate in which they thrive".

Subtelny (1962) noted that some cleft patients have a potential for attaining a favourable facial appearance whereas some, right from the time of birth, do not have this potential. This factor was confirmed by Silla (1969) when he noted that basically there were two types of maxilla:-

- (i) a very wide maxilla compared to the mandible with an occlusion characterized by a deep bite; or,
- (ii) accentuated maxillary underdevelopment from the beginning.

Berndorfer (1963) considers the method of surgical operation, technical procedure and the time of operation plays an important part, but we cannot be sure of a satisfactory result unless we know the special and individual properties and constitution of the deformed child. Every child has its own particular biological characteristics. There have been some studies indicating that surgery may have an affect on growth of the mid-third of the face but Spriestersback, Dickson, Fraser, Horowitz, McWilliams, Paradise and Randall (1973) emphasise that other variables must be considered in addition to surgery:-

- (i) Many cleft children present with a maxilla that is deficient in size at birth and surgery may, in that case, serve to make an unfavourable situation worse.
- (ii) Size of maxilla may be adequate prior to surgery but it is possible that these patients may have

an inherent tendency towards less growth in the maxillary complex or perhaps a slower growth as they mature. However, Silla (1969) takes the opposite view that successive surgical operations do not seem to exercise a negative influence upon maxillary development in favourably-sized maxilla;

Aduss (1971) emphasised that children with clefts are like any other youngsters in that they may or may not have an inherent imbalance in facial skeletal patterns and may or may not present aberrations in tooth position or arch length problems. Thus, superimposed on the variables found in all children there exist the problems associated with the presence of the cleft deformity. He concludes by stating that current conservative surgical techniques which do not resort to presurgical maxillary orthopedics and primary bone grafting do not interfere with facial growth and development. Therefore, the assumption can be made that children with clefts will proceed to develop as well as children without clefts. However, anterior crossbites and/or some degree of maxillary hypoplasia may occur. Aduss considered that the prevalence of anterior crossbite was not significantly different between the cleft and non-cleft group.

Bishara, Krause, Olin, Weston, Van Hess and Felling (1976) point out that most investigations agree that differences in maxillofacial growth and development exist in individuals with clefts of lip and/or palate who have undergone surgery in childhood when compared with non-cleft children. The causes of these differences, however, are still controversial. Bishara, et al (1973) have attempted to present some factors to explain these differences:

- (i) Inherited trait, i.e. genetic influence on size and form.
- (ii) Acquired trait - biomechanically obligatory adaptive changes in size and form.
- (iii) Induced traits - changes in growth, i.e. in size and form imposed through surgery and orthopedic or orthodontic management.
- (iv) Combination of above and with others not yet identified.

Huddart (1967) also emphasized that the cleft condition may be superimposed on a potential malocclusion which is genetically determined. Those children who would have had an Angle Class II malocclusion if no cleft had occurred are at an advantage. A potential Class III case seems to be converted into a Class I case by the cleft. Whilst a Class I or Class III case seems to be converted into a mild or severe Class III malocclusion, as the case may be. From his observations, he states that where a child would have had broad well-formed arches if no cleft had been present, the development of a cleft and its subsequent treatment tends to give rise to a relatively well-formed arch with minimum collapse of segments.

Maxillary Growth

Despite the abnormal matrix established in the embryo and the distortion of the maxilla which occurs due to abnormal muscle forces in the fetus, growth proceeds in a relatively normal manner thereafter. Ross (1975) considered that the inherent growth potential of the maxillary complex of cleft subjects is excellent. This has been confirmed in mature unoperated cleft lip and palate patients. However, many investigators have confirmed the fact of a retrusive tendency of the midface and an inadequate antero-posterior dimension of maxilla in repaired cleft lip and palate patients. (Chierici, Harvold and Dawson, 1970; Dahl, 1970; Foster, 1962, 1970; Graber, 1949, 1954; Horowitz, et al, 1976; Levine, 1963; Ross and Johnston, 1967 and Shibasaki and Ross, 1969.)

Hayashi, et al (1976) studied the SNA angle which measures the forward growth of the midface relative to anterior cranial base. Change of this angle with increase in age was small in the control group. They showed a tendency for SNA angle to decrease with age in cleft groups and explained this as being due to the tight lip musculature and scar tissue on the alveolus. Shibasaki and Ross (1969) also confirmed the above statement. However, Krogman, et al (1975) noted the SNA angle to be greater in the complete cleft lip and palate group than in both cleft palate only and the normal group. In his normal group the angle is relatively constant though it does decrease slightly. In the cleft face, point A is more retruded than in the normal which means that point A is a register of some inhibition or deviation in the forward growth of the midface. However, since the SNA angle is larger in complete cleft lip and palate group than in cleft palate cases only, and since key ridge to ANS distance contributes slightly less to maxillary length than does pterygomaxillary fissure to key ridge distance, it is logical to point to anterior segment (premaxillary) positioning rather than to incremental failure.

Others have shown that the maxilla is located in a more backward position relative to the cranial base in cleft palate cases in comparison to controls. (Blaine, 1969; Dahl, 1970; Foster, 1970; Hama, 1964; Sekiguchi, 1971 and Shibasaki, 1973.) Other workers have observed concurrent retrusion and hypoplasia of the maxilla.

However, in one study (Aduss, 1971) it was found maxillary length and position in repaired cleft lip and palate children did not differ from the controls. He explained this as being the result of the current conservative surgical procedures which can produce satisfactory results without interfering with growth.

Atherton (1967) reported a shorter length of the maxilla on the cleft side and a significant distal position of the non-cleft side in the unoperated complete unilateral cleft skulls.

Foster (1959) from his 200 cleft patient sample noted a 34% incidence of maxillary retrognathism compared to 9% obtained by Leech (1958) and 11.1% obtained by Walther (1960) for the non-cleft population. Foster questioned how much of this was due to surgical repair and how much to genetic variations in size of jaws and agenesis associated with cleft formation. This genetic predisposition underlying the cleft was also questioned by Chierici, et al (1970).

Hayashi, et al (1976) from a cross-sectional study on complete unilateral cleft lip and palate children from 4 to 18 years of age, considered appositional growth at the maxillary tuberosity and sutural growth to have an important role in increasing maxillary depth. Björk (1966) postulates that sutural growth continues until 17 years of age, while Latham and Burston (1966) consider it effective only up to 2 years of age. Scott (1959) believes it ends after seven years of age. Asai (1973) measuring from point A to the pterygomaxillary fissure found that maxillary depth increased up to 17 years of age. Hayashi, et al (1976) found that the increment in growth of the maxilla from one age to another was smaller in the cleft group than in the controls, especially after 8

years of age. This may suggest either lower maxillary growth potential in the cleft group or the effect of the maxillary ankylosis as suggested by Ross (1970).

In many techniques the hamulus is fractured and some dissection of tissues in this area is carried out to release tension on the soft palate rudiments and to alter the direction of pull of the tensor palati muscle. The mucoperiosteum covering the palate is raised and is displaced medially and frequently posteriorly. As a result of these procedures, a continuum of scar tissue joins the maxilla, the palatine bone and the pterygoid plates of the sphenoid, perhaps inhibiting separation of these bones and having a direct influence over maxillary growth.

The palatal plane in cleft lip and palate patients is located in a relatively superior position in the nasopharyngeal area, which appears to be a consequence of short upper posterior face height. (Blain, 1969; Brader, 1957; Dahl, 1970; Hama, 1964; Horowitz, et al 1976 and Levine, 1963.) The upper face height in clefts has been reported to be less when measured from nasion to the nasal floor. (Hayashi, et al, 1976). Graber (1954) observed that the nasal floor descent is in a parallel fashion in the normal sample. In his cleft patients it was observed to be erratic with a tendency for the posterior end to drop more than the anterior. Hayashi, et al (1976) explained that this decrease in upper face height may be related to the deceleration of the downward development of the anterior part of the nasal floor. On the other hand, the greater lower face height is probably due to the upward position of the nasal floor and the backward rotation of the mandible.

There is general agreement that a cleft population usually demonstrates an increase in their total facial height and the increase

is often found in the lower third of the face. (Farkas and Lindsay, 1972, 1973; Mowbray, 1977 and Ross and Johnston, 1972.)

We must remember that a cleft child, compared to a normal child, presents with alteration of the resorptive and appositional process of nasal floor, a deviated nasal septum (Aduss and Pruzansky, 1963 and Latham, 1969) and reduced sutural growth which may be considered as causes of underdevelopment of the maxilla in a downward direction.

Various workers have stated that the factors we should consider that may individually or in combination affect the growth potential of the face, particularly the maxillae, are:-

- (i) the nefarious effect of surgery in terms of the age at which surgery is performed and type of surgery performed,
- (ii) the mere presence of a cleft may affect the growth of the region either by a lack of "proper parts" to support the midface in its normal growth or through a lack of optimal stimuli which are produced by normal function,
- (iii) the severity of the palatal cleft both in antero-posterior direction and in width, and
- (iv) pre- and post-operative care including orthodontic and prosthodontic treatment.

Mandibular Form and Posture

It has been hypothesized that adaptational changes in mandibular shape are related to altered postural position of the mandible under conditions of maxillary deformity, without a compensating increase in alveolar height. (Chierici, Harvold and Vagervik, 1973; Harvold, 1959; Ross, 1970; Ross and Coupe, 1965; Ross and Johnston, 1967 and Shibasaki and Ross, 1969.)

Shibasaki (1973) considered the characteristics of the mandible as a result of morphological adaptations in oral functions in response to small oral and pharyngeal cavities.

Ross (1970) has stated factors that may contribute to alteration in mandibular posture as being frequent respiratory infections, nasal septum deviations, contracted maxillary arch and low palatal vault. Further, Hayashi, Sakuda, Takimoto and Miyazaki (1976) regarded intra-uterine factors, difficulties in feeding and surgical procedures as contributors to changes seen in the mandible.

It has been claimed that the length of mandible is shorter than normal, (Levine, 1963; Hama, 1964; Dahl, 1970 and Deuschle and Kalter, 1962.) However, Ross and Coupe (1965) and Rosenstein (1975) consider it being unaffected. Rosenstein noted a higher frequency of steeper mandibular plane angles.

Horowitz, Graf-Pinthus, Bettex, Vinkka and Gerstman (1976) consider that the size of the gonial angle warrants attention as a key morphological trait. Horowitz, et al and Scott (1959) believe that muscle function determined the ultimate form of the mandible at the gonial angle and that reduced muscle activity would account for the flattening in this area and therefore the obtuse gonial angle. When this angle is obtuse a retrognathic appearance

is produced because the chin is displaced in a downward and backward direction. (Hama, 1964; Dahl, 1970; Krogman, Mazaheri, Harding, Ishiguro, Bakiana, Meier, Canter and Ross, 1975 and Vora and Joshi, 1977.) However, this is not always true as Rakosi (1970) has shown non-cleft prognathic cases as having high gonial angles. Hayashi, et al (1976) considered that the inclined mandibular plane in clefts to be partly the result of the reduced surface bone apposition in the gonial area.

Aduss (1971) from his study emphasised that two factors needed further analysis: the anterior cranial fossa and mandible to determine whether the increased gonial angle compensated for a decreased mandibular ramal height and/or body length, and whether the cleft-group population has an elevated anterior cranial fossa.

Enlow (1971) considered the major site of posterior and upward growth of mandible to be in the ramus. Many investigators have noted a short ramus in cleft groups. (Dahl, 1970; Levine, 1963; Nakamura, Savara and Thomas, 1972; Sekiguchi, 1971; Shibasaki, 1973 and Vora and Joshi, 1977.)

Many investigators have observed that the mandible was positioned significantly more posterior than normal when related to the cranial base. Bishara (1973), Hama (1964), Hayashi, et al (1976), Krogman, et al (1975), Levine (1963) and Ross and Coupe (1965) observed in their studies that the SNB angle was smaller in cleft groups when compared to controls. It is interesting to note that in their 255 cleft sample they found a 93% incidence of anterior crossbite. Susami (1967) studied growth of the dento-facial complex of non-cleft Japanese children presenting with anterior crossbite from deciduous to adult age. He found the mandible to be larger than in the controls in body length. This resulted in a marked increase of the SNB angle in the mixed dentition stage. Thus, growth of non-cleft subjects with anterior crossbite was quite different from that of the cleft group.

However, many investigators have shown that with an increase in age, the skeletal chin tended to become more prognathic. (Osborne, 1966; Levine, 1960 and Coccaro and Pruzansky, 1965.) Quinn, Pickrell and Massengill (1971) are of the opinion that the mandibular prognathism in cleft patients is often due more to the lack of soft-tissue balance than to the bone or skeletal profile relationship. Therefore, it is obvious that the form of mandible is different in cleft individuals. This confirms the hypothesis of Chierici, et al (1973) that it is a phenomena that is secondary to the cleft deformity.

Naso-Pharyngeal Area

In recent studies there has emerged the important role of posterior craniofacial development in the cleft lip and palate syndrome. We should consider what consequences posterior growth deficiencies may have because of a large number of cleft lip and palate children who have problems with deglutition in infancy and in attaining adequate speech skills later. There is not much information available concerning abnormal development of the nasopharyngeal area. (Horowitz, et al, 1976.)

Limited data relating to the nasopharynx development in cleft lip and palate children show that height increases with age, but that both vertical and depth dimension are smaller than normal until seven years of age. (Coccaro, Pruzansky and Subtelny, 1967.)

Horowitz, et al (1976) found an association between the smaller nasomaxillary-pharyngeal complex area and the reduced upper posterior face height dimension in cleft children. Brader (1957) noted a comparably smaller pharyngeal area in his mixed cleft sample. However, Subtelny (1955) noted that the nasopharynx width is larger in cleft than unaffected individuals.

Ricketts (1954) believed that abnormal pharyngeal depth may be one of the bases of velopharyngeal inadequacy in individuals with a cleft and that these children may exhibit cranial base abnormalities.

Schweiger (1966) examining 67 cleft palate individuals between 12 and 16 years of age observed that the cranial base angle, bony depth of nasopharynx and position of maxilla did not appear to be related to the amount of velopharyngeal opening. The variables showing a significant relationship with velopharyngeal opening were nasopharyngeal depth, total palatal length, and palatal movement.

Pruzansky (1973) observed that anomalies of the skull base and upper cervical column are significant determinants of the prognosis for palato-pharyngeal valving. However, there are many investigators who found no differences in cranial base angle or nasopharyngeal depth among subjects with unoperated cleft palates, operated cleft palates and normals. (Brader, 1957; Engman, Spriestersbach and Moll, 1965; Mestre, Dejesus and Subtelny, 1960 and Ortiz-Monasterio, Serrano, Valderrama and Cruz, 1959.)

The causal mechanism of deficient nasomaxillary-pharyngeal growth in cleft lip and palate has yet to be demonstrated. The adverse effects of a muscular ring that is incomplete because of a cleft in the soft palate have been pointed out by Subtelny (1955). Surgical repair restores much of the physiological capacity of this muscle ring but morphological evidence shows diminished posterior cranio-facial growth in cleft lip and palate patients as presented by Horowitz, et al (1976) and early studies. It is strongly suggested that the functional level attained remains below that of non-cleft individuals and that there is little catching up of growth in this area. Surgical repair may well contribute to this growth lag.

Keith and Campion (1922) and only recently Moss and Salentijn (1969) proposed that the respiratory function has a primary influence on the growth of the nasal, oral and pharyngeal functional spaces. Cleft lip and palate patients have disturbed oro-nasal air-flow and this impairment of respiratory flow could result in diminished growth in the naso-pharyngeal area.

Narrowing of the nasal airway occurs in a significant number of individuals with cleft lip and palate due to:-

- (i) Hyperplasia of the conchae.
- (ii) Deviation of the septum.
- (iii) Nasal aperture stenosis. (Drettner, 1960.)
- (iv) Increase in adenoid tissue. (Brader, 1957.)

This narrowing is presumed to be the cause of the higher nasal pathway resistance found in these patients. (Warren, Duany and Fischer, 1969.)

Therefore, structures such as the nasal septum, maxilla, pharynx, etc. may show deviant morphology because they are primarily affected by the clefting, although the possibility remains that at least some of these structures are basically normal and that they develop abnormally because of a proximity to adjacent abnormal structures.

Cranial Base and Associated Structures

There have been studies that have suggested that the clefting process does not produce an isolated anatomical defect but may be a syndrome phenomenon affecting other structures. (Dahl, 1970 and Farkas and Lindsay, 1972.)

Krogman, et al (1975) in their study stated:

".....there is evidence that palatal clefting has growth and/or development repercussions in the associated cranial base and facial structures, probably a reflection of embryonic growth-timing, i.e., palatal clefting may be associated with a concomitant growth dysplasia of basic-facial structures in their formative stages at the time when palatal clefting occurred."

Coccaro and Pruzansky (1965) are in agreement when they state:

".....cleft lip and palate must be considered as an anomaly complex involving organs contiguous to the cleft."

Snodgrass (1954) noted evidence of overall irregularity and failure in growth and development in individuals with clefts. However, Ross (1965) reported a smaller cranial base in cleft children, but felt that it was due to the smaller total size of these children rather than a "reflection of an abnormality in the cranial base."

Horowitz, et al (1976) studying 39 complete unilateral cleft children and comparing them to normals at the mean age of eight years noted that the cranial base, mandibular ramus, palate nasopharyngeal, maxillary complex and lower face accounted for 92% of the variance with the latter two providing the best discrimination between clefts and non-cleft individuals.

Blaine (1969) analyzed the cranial base and mid-facial region of 443 operated and unoperated cleft children. He stated that as a result of aberrations in the mid-palatine region growth forces of the vomer may in part be expended by exerting force on the vertical plate of the ethmoid. This upward component of force might raise the anterior cranial base and result in the slight opening of the cranial base angles and force the nasal floor downward. There was a tendency toward obtuseness in all angles studied in relation to the posterior cranial base. This was also observed by Aduss (1971), Dahl (1970), Hama (1964), Hayashi, et al (1976) and Shibasaki (1973). Aduss and Pruzansky (1963), Ross (1965) and Sekiguchi (1971) found no differences in cranial base angles between the cleft group when they compared them to controls.

A frequently cited study by Moss (1956) describes a marked kyphosis of the sphenoid bone associated with clefts. Moss also described a downward and backward rotation of the neural skull, whereas Ross and Coupe (1965) reported a downward and backward rotation of the facial skull around a fulcrum located in the region of the nasion. Later Moss (1957) found premature closure of the frontal suture was three times more frequent in cleft than in non-cleft skulls.

Björk (1961) stated that when the elongation and the lowering of the medial fossa are proportional in magnitude, the shape of the cranial base is unchanged from childhood to adulthood. However, when the posterior lengthening of the base is greater than the lowering, the base flattens out with growth. Hayashi, et al (1976), examining cranial base growth in unilateral clefts subjects from 4 to 18 years, observed very little change with increase in age, in either the cleft or control subjects. However, the angle for the cleft group was larger than for the controls. This increase in cranial base angle may be related to the greater angulation of the medial pterygoid plates in all cleft types as reported by Subtelny (1955). This is assumed to have probably occurred early in the embryonic stage. As already mentioned it may

be related to the dysostosis sphenoidalis as reported by Moss (1956).

Examining the cranial base length, Levine (1963), Sekiguchi (1971) and Shibasaki (1973) noted no differences in length and growth rate at any age between cleft and non-cleft groups. However, Dahl (1970) stated that the anterior cranial base length (N-S) was shorter and Röss (1965) reported the total cranial base length was shorter. Aduss (1971) found that only girls with clefts revealed shorter cranial base length.

Animal Studies as they Relate to Orofacial Growth Development.

(a) Lip Repair

The influence of soft tissue forces on skeletal growth has been questioned. Ritsila, Alhopuro, Laine and Ranta (1973) reported on an investigation using two weeks old rabbits in which the normal median cleft of upper lip was closed by paring the edges and suturing. At six months "slight shortening" of the maxilla and "marked shortening of the body of the mandible and alterations of several mandibular angles", were observed.

Bardach and Eisbach (1977) reported findings from an experiment which entailed monitoring of postsurgical lip pressure and subsequent midfacial growth. Working on rabbits they concluded that pressure exerted on the alveolus by the repaired lip was considerably greater than normal in the early postsurgical weeks and probably contributed an additive retrusive influence to that already provided by the surgical creation of the clefts.

Later, Eisbach, Bardach and Klausner (1978) set out to test their previous hypothesis:

".....that the pressure of the repaired cleft lip on the maxillary segments is an inhibitor of facial growth resulting in secondary maxillofacial deformities."

The results of direct cephalometry of the skulls of 33 rabbits were presented. They noted significant inhibition in antero-posterior facial growth in the control group in which a cleft lip, alveolus and palate were surgically created and left unrepaired. In comparison to the lip

repaired groups, this inhibition was increased, indicating that primary unilateral cleft lip repair is an inhibitor of facial growth. However, the full nature and extent of the influence of primary cleft lip repair are not yet known and cannot be ignored. Therefore, all the adverse effects of the repaired cleft are not due to palatal surgery.

Many investigators have noted a binding effect on the maxilla by tight repair of cleft lip. Burston (1959), Johanson and Ohlsson (1961), McNeil (1956) and Stellmach (1959) have devised methods of repositioning the maxillary segments in infancy and stabilizing them.

(b) Cleft Palate Repair

An aim from animal experiments is to elucidate control mechanisms that influence growth in the maxillofacial complex. A more important goal is the identification and control of surgical variables used with cleft palate repairs that might interfere with postsurgical growth of the face.

Herfert (1958), from his experiments, inferred that the major growth-damaging variables in cleft palate surgery were interruption of palate neurovasculature. However, Kremenak and Searls (1971) and Sarnat (1953) disagreed and demonstrated that there were no growth effects of early bilateral interruption of the greater and lesser palatine neurovasculature. This served as a basis to change from a von Langenbeck palatal repair to a modified V-Y design which involves neurovascular interruption in the primary procedure to facilitate soft palate elongation.

Inhibition of maxillary growth was produced in animals when surgical denudation of palatal bone adjacent to deciduous teeth occurred. (Kremenak, et al, 1971 and Lynch and Peil, 1966.)

Olin, Morris, Geil, Pratt and Kremenak (1974) believed that post-surgical contraction of palatal mucoperiosteal excision wounds are similar to that reported for skin wounds. It was noted that major interruption of increases in arch width coincided with the period of soft tissue contraction. The more recent efforts by the group focussed on the contraction phase of early healing of surgical wounds. They hypothesized that this contraction is the primary event that initiates the ensuing growth inhibiting process and that development of techniques to prevent wound-healing contraction should enable us to prevent this undesirable growth sequelae. They were able to prove this point in several studies in which they periodically severed:-

- (i) attachments between wound margins,
- (ii) central granulation tissue, and
- (iii) gingival attachments of teeth adjacent to wounds.

These procedures all resulted in reduction of both contraction and jaw growth aberrations. They also showed that transplantation of autogenous free grafts of other oral mucosa into hard mucoperiosteal excision wounds resulted in a reduction of the contraction and this was followed by normal or "supranormal" increases in maxillary arch width.

Interesting experiments were performed by Latham, Smiley and Gregg (1973) and Calabrese, Winslow and Latham (1974) testing the hypothesis that new tissue could be induced to form in the

growing face by application of approximately controlled physical stress. They demonstrated this in one study where they artificially produced clefts in dogs and gradually closed them using pin-screw appliances. After two weeks they noted that histologically new bone formed and resorbed as necessary to maintain skeletal continuity. In another study they produced an artificial suture medial to the molar alveolar process and showed new bone formed in these osteotomy sites as the cleft was closed by a pin screw appliance.

The main emphasis in research today is to analyse surgical wound healing and its contractile phenomena. Research still continues to study normal and abnormal growth and function in the craniofacial complex. It is hoped to develop ways to avoid or compensate for growth-inhibiting surgical variables.

Immediate Effect of Repair on Growth

(a) Lip Repair

The surgical reconstruction of the lip and floor of the nose establishes the facial-pharyngeal ring of musculature consisting of orbicularis oris, the buccinator and the superior constrictor muscle of the pharynx. The sphincteric nature of peri-oral muscles can be seen to have a strong moulding influence on the underlying bony structures. It eliminates the abnormal muscle pull which appears to be the major cause of the rotation and expansion of the maxillary segments.

Pruzansky and Aduss (1964) observed that lip repair was able to bring the free ends of the alveolar segments into approximation or overlap in approximately 80% of cases.

There are a number of variables which may alter these effects:-

- (i) Tightness of the repaired lip.
- (ii) Repair of the alveolar cleft. (Muir, 1966.)
- (iii) Intrinsic deficiency of the maxilla.
- (iv) Abnormal initial form or relationship of the segments.
- (v) Persistence of a tongue thrust into the cleft region.

Despite the anterior narrowing the posterior maxillary width remains greater than normal.

Mazaheri, Harding, Cooper, Meier and Jones (1971) from observations in unilateral cleft lip and palate children ranging from 6 months of age to 5 years, concluded that reduction of the cleft after lip surgery is attributed to:-

- (i) segmental repositioning,
- (ii) downward growth,
- (iii) changes in angulation of palatal shelves,
and
- (iv) mesial growth of palatal shelves of the
lesser segment.

Hagerty and Hill (1963), examining complete clefts without and with lip repair only, observed that there was some lag in antero-posterior maxillary growth in both cases. This may be due to congenital defect of the compressive effect of tight lip surgery. If the lip repair was done at three months, they noted that there was significant lingual version of the incisor teeth, posterior displacement of the upper lip and exaggerated fullness of the lower lip. This did not occur if repair was performed at two years. This delay of approximately two years was advantageous for the following reasons:-

- (i) The infant being larger simplifies lip surgery.
- (ii) The increased maturity of the dentition.
- (iii) The extent of growth of maxilla at the time of lip surgery plays a role in the eventual facial contour.

They concluded, and many other investigators agree, that secondary deformities of the maxilla are directly related to lip repair rather than to palatal repair. (Subtelny, 1957 and Swanson, 1958.)

Wada and Mazaheri (1975, 1976) evaluated segmental displacement and changes of the alveolar arch "tri-dimensionally" using maxillofacial models on 87 complete unilateral cleft

lip and palate patients and 62 normals. They examined each child at 6 months (prior to lip surgery), 2 years (prior to palate surgery), 3 years and 4 years. Prior to lip repair, clefts and normal subjects showed similar patterns of maxillary growth. It became clear that the antero-posterior growth inhibition of the maxilla occurred just after lip repair and this difference from the normal was not compensated for by the age of four years.

Robertson and Fish (1975) observed an increase in arch width in the two year period following palatal surgery. However, during the same period the intercanine width remained static and the absence of significant growth in this area can be attributed to early repair of the anterior palate and the continued effects of lip pressure. Therefore, we should concentrate on the improvement of lip and anterior palate surgery.

Hagerty and Hill (1963) consider it most important in maintaining the cleft width in relationship to maxillary and mandibular arch alignment and activating the growth potential of the smaller segment.

(b) Palate Repair

The failure of the hard and soft palate to unite in the midline produces a profound disturbance in the interplay and balance between the involved muscle groups and the bony processes which provide their origins and insertions. Slaughter and Pruzansky (1954) outlined the several muscle groups that should be considered:-

- (i) the tongue acting to displace the palatal process,
- (ii) the lateral and medial pterygoid muscles which during mandibular function exert an unopposed lateral pull on the pterygoid plates, and
- (iii) the levators and tensors of velum being antagonistically unopposed in cleft patients contribute to the divergence of the pterygoid plates.

Subtelny (1955) emphasized the fact that we are dealing not only with a break in the structures but with displaced and distorted structures as well. Closing the hard and soft palate defect, immediately restores the palatopharyngeal ring of muscle and the function of speech and swallowing are assisted. However, significant morphological changes occur in the maxilla and pharyngeal area as a result of this cleft repair. The basic problem is the inadequate amount of tissues in the region of the cleft. The surgeon must either borrow from an adjacent area, repair under tension or perform a two-stage repair procedure. (Ross and Johnston, 1972.)

Herfert (1958) noted good maxillary growth and alignment in a number of his twelve year old patients who had lip repair but no repair of the palate and perhaps most significantly, no anterior palate repair. He favoured a two-stage repair where the soft palate was repaired at 18 months and hard palate closed between 6 - 8 years of age. This approach was previously adopted by Schweckendiek (1951) and later by Slaughter and Pruzansky (1954). Hotz (1973) also adopted the above procedure and explained that growth of the palatal shelves enabled

hard palate closure using a small median mucoperiosteal flap. This avoids denudation of bone along the gingival border which is largely responsible for maxillary growth inhibition. Since this method may have an adverse effect on soft palate function, the usual procedure is to mobilize palatal mucoperiosteal flaps using a releasing incision close to the alveolar process. This leaves areas of denuded bone close to the alveolus where residual scar tissue forms and is responsible for the inhibition and distortion of growth.

Dey (1974) emphasized that if incisions transgress the alveolar mucoperiosteum on the ridge before the teeth erupt, localized dental and alveolar deformity will always result. This was supported by Ross (1975) when he stated that by avoiding surgery adjacent to the alveolus the dento-alveolar accommodation mechanism will be freely expressed.

Dunn (1952) considered lack of development of the upper jaw as not being intrinsic, but probably due to the usual technique of elevating the periosteum and mucous membrane from the palatal bone, thereby introducing a scar tissue covering of palatal bone which constricts it and interferes with periosteal blood supply. He considered periosteal blood supply very important in the growth of bone of this type which grows by lamination rather than from the centres of ossification. Therefore, he advocates vomer flaps and simple closure of the soft palate. On the other hand, Sarnat (1958) found that neither the surgical trauma of raising a flap nor deprivation of blood supply is the cause of maxillary and facial lack of growth, a finding agreed with by the work of Foster (1962) in humans with complete alveolar clefts.

Robertson and Fish (1975) observed that, in the two year period following palatal surgery, a further increase in posterior arch width occurred indicating that permanent inhibition of palatal growth does not result from surgical procedure on the hard palate.

Latham and Burston (1966) had shown that lateral activity in the mid-palatine suture is greatly diminished by 18 months and has ceased by 2 years and, therefore, between 18-24 months lateral growth of hard palate takes place as a result of alveolar appositional growth only. Consequently, an operation on the hard palate at this time cannot be expected to inhibit growth due to scar tissue formation. Dahl (in press) explained medial displacement of the lateral segment on the affected side following surgery as being mainly a mechanical movement. Long-term effects on maxillary growth are added to these immediate changes. Such effects are fibrosis or bony ankylosis produced by the palatoplasty and the abnormal condition in the mid-palatine suture. He was able to demonstrate in his longitudinal roentgenocephalometric studies by the implant method that spontaneous growth corresponding to the mid-palatine suture of the maxilla is retarded in operated patients.

It is a widely held view among British plastic surgeons that the timing of repair of the secondary palate should be dictated by the need for acquiring normal speech rather than by fear of interfering with subsequent growth and development. Jolley (1954) found significantly better speech results in patients for whom the palate had been repaired before the age of three years.

Surgery can aid in directing the natural growth process into proper channels through establishment of muscle balance across the defect. (Slaughter and Pruzansky, 1954.) However, the surgery also introduces morphological changes and new growth factors which almost invariably have a deleterious effect on facial development. (Ross and Johnston, 1972.)

Phenomenon of Catch-Up Growth

Mazaheri, Harding, Cooper, Meier and Jones (1971) examining casts of 30 complete cleft patients between 6 months and 5 years of age, noted a significant retardation in the antero-posterior and lateral dimensions immediately after surgery. This lag diminished in most subjects after four years of age. They concluded that growth was favourable as long as it was not retarded by surgical intervention and scar tissue. This was also confirmed by Mapes, Mazaheri, Harding, Meier and Canter (1974) in their serial study and Robertson and Fish (1975). It is interesting to note that Berkowitz, Kirscher and Pruzansky (1974) demonstrated by means of a geometric study that in complete bilateral cleft lip and palate patients the palatal surface area doubled from birth to 1½ years of age.

Coccaro and Pruzansky (1965) using cephalometrics, measured skeletal profiles of complete unilateral clefts from 3 months to 7 years of age and compared them to normal individuals. They noted that the deleterious effects of surgery on growth of middle face do not apply to children in their study. They contradicted the opinions of Graber (1949), Krogman (1954), Slaughter and Brodie (1949) and Snodgrass (1954). Today the surgical procedure has changed and is not inhibitive to the growth process. Coccaro and Pruzansky noted that convexity of skeletal profile was greater for the clefts than for the normals, but there was a reduction in convexity of the soft tissue profile. They explained this as a flattening of the nose with the soft tissue of the upper lip overlying point A being reduced in thickness in the cleft lip sample, when compared to normal.

Krogman, et al (1975) hypothesized that:

".....operative intervention in cleft palate cases which minimally involves bone growth potential will guide and facilitate maxillo-facial growth in the individual so that post-operative

growth in a catch-up manner will provide for the achievement of an acceptable normal craniofacial dental growth pattern".

On the basis of two serial samples of cleft palate only and unilateral cleft lip and palate they presented data which could substantiate their hypothesis.

A recent study of Aduss (1971) reported good longitudinal facial growth and concluded that plastic surgery actually encouraged rather than inhibited growth. Therefore, the cleft palate children grew much like children without clefts. This may be true for some cleft samples. However, it is also true that there are still many cases showing the concave profile. Pruzansky (1973) believes that:

".....maybe within certain defined limits the success or failure of the surgical procedure depended more on the initial state than on the variables inherent within the manoeuvre."

Hotz (1973) believes the frequent pseudoprognathism has to be attributed to premature and inadequate surgical methods.

Occlusion in Cleft Patients

(a) Arch Collapse

Aduss and Pruzansky (1967) stated that there are two fundamental questions central to their clinical efforts:-

- (i) Is collapse of the arch an inevitable sequel to repair of cleft lip and palate? If not, what proportion of cases do collapse? What is the prevalence of malocclusion, particularly cross-bite in children with cleft lip and palate where the reconstruction did not include pre- and post-surgical orthopedics and bone grafting?
- (ii) If some arches collapse and others do not within the surgical experience of a school of surgeons using an essentially similar procedure for all of their patients; what are the factors which favour or prevent such collapse?

It is possible that variants in morphology peculiar to the individual patient are the determining factors for ultimate arch form.

After examining many clefts, Aduss and Pruzansky came to the conclusion that the amount of post-operative collapse may be related to:-

- (i) Size and shape of the alveolar process adjacent to the cleft.
- (ii) Size and shape of the inferior turbinate on the cleft side.

- (iii) Size and inclination of the nasal septum.
- (iv) Size and shape of the palatal shelves.

Battle and Whitfield (1970) noted that a collapse of the upper arch may occur very early and is probably due to a combination of factors such as presence of defect in the bony plate, pressure of buccinator muscles and early forward growth of the nasal septum. The fact that collapse does not occur in all cases may be attributed to a variety of causes, such as:-

- (i) the width of the cleft
- (ii) the rate of growth,
- (iii) the action of the tongue within the mouth, and
- (iv) the built-in development part of the maxilla itself.

Muir and Bodeham (1966) considered that the deformity which results from the maxillary collapse is determined by four factors:-

- (i) the size of the bone defect, which varies from case to case,
- (ii) the original position of the lesser segment in relation to the greater segment,
- (iii) the original amount of rotation of the premaxillary position of the greater segment, and
- (iv) the type of primary repair with particular reference to the amount of raw surface left between the alveolar ends.

Even when there was originally marked rotation of the premaxilla, repair by a method designed to minimize scarring in the gap will delay collapse long enough for the premaxilla to rotate back into line before the position becomes fixed so that the shape of the arch is good. (Burian, 1955 and Muir, 1966.)

Dey (1974) also confirmed that anterior palatal closure at the time of primary lip repair may well contribute to a collapse of the dental arch and the production of crossbite. This movement occurs as a result of tilting of the maxillary segments or perhaps a movement which takes place at the sutures along the line of the base of the skull. Muir and Bodeham (1966) confirmed this displacement as a rotary movement hinging on the pterygoid plates. Therefore, the deformity is maximal anteriorly where the canine tooth is inside the bite, and minimal posteriorly where the molar teeth appear in good position.

Bernstein (1972) demonstrated in complete unilateral clefts a small increase in posterior crossbite malocclusion, and a notably high

incidence of collapse of the posterior dental arch in the group that had the alveolar cleft repaired at the time of the cleft lip repair at 11 weeks of age. There was a 24.7% incidence of medial rotation of palatal shelf in patients with the lip repair only and 35.5% in patients subjected to lip and anterior palate repair. Bernstein and Harding and Mazaheri (1972) considered that maxillary growth retardation after early surgical closure of the alveolar cleft might be due to a growth interference of the vomer-premaxillary suture.

Foster (1962) explained two types of medial collapse of upper alveolar arch:-

- (i) a "pre-operative" type of collapse - this occurs before any operation has been performed, and
- (ii) a "buckling" type of collapse following palate repair.

Many investigators attribute collapse to surgical repair of the palate. (Dunn, 1952; Henry, 1955 and Hyslop and Wynn, 1952.) Pruzansky and Aduss (1964) have shown that in complete unilateral clefts following cheiloplasty and prior to palatal repair collapse of the alveolar arch occurred in close to 40% of the cases and suggested this to be the result of the splinting effect of the reconstituted orbicularis oris musculature. Hagerty (1957), Swanson (1958) and many others also confirmed this collapse as being due to lip repair only.

Maisels (1973) observed that 30% of all cases may not need any early orthopedic treatment. However, of the remaining cases there are those whose response to treatment is unfavourable and by 6 months of age the alignment is still defective or the growth of the lateral segments grossly inadequate. Reasons for this occurring are still completely unrecognised or only poorly understood. In order to avoid the excessive pull resulting from single layer closure of the anterior palate and subsequent arch collapse and to facilitate post-

operative orthopedic treatment, the anterior palate is left unrepaired in these cases.

In conclusion, his message was that to prevent arch collapse and gain the best results from treatment the orthodontist should be able, from frequent observation of the growth pattern and response to treatment, to firstly detect the susceptible cases and then to decide, as the case may require, upon the nature and timing of the surgical repair procedures.

(b) Crossbite Development

Hagerty (1957) tried to explain how a crossbite is produced. The alveolar ridge is situated at the inferior extension of the maxilla and is subjected to a medial force of the lip which is greater than the lateral force of the tongue. One palatal segment is in continuity with the nasal septum. This serves as a flying buttress which stabilizes the alveolar segments on the side opposite the cleft. The alveolar ridge on the cleft side is unable to withstand the compressive force of the lip and drifts medially as represented diagrammatically by the broken line in Fig. 3. Hagerty observed that 2/3 of all unilateral complete cleft lip and palate patients and 3/4 of all bilateral cleft lip and palate patients had a crossbite deformity. He reasoned that the expanding force of the tongue is no match for even the most loosely repaired lip and that collapse of the dental arch is to be expected. He emphasized that to prevent collapse of the dental arch it is necessary that the arch be supported in compression. He resorted to an expandable bar which is placed at the time of lip surgery (three months of age) and left in place until satisfactory ossification of the maxilla has taken place.

Crossbite development as explained by Chierici, Harvold and Dawson (1969) appears to be closely related to the degree of displacement of segments. This substantiates Harvold's (1954) explanation of the mechanism of post-surgical collapse of a cleft maxilla by inward rotation of the smaller segment around a fulcrum situated in the tuberosity region. This usually takes place before all the teeth are fully erupted and a corresponding reduction of alveolar growth in height will be noted. When the arch is compressed the canine and premolars erupt lingually and the normal vertical growth of the alveolar process is obstructed by the tongue. The conclusion which may be drawn from these studies is that the major part of deformities seen in cleft lip

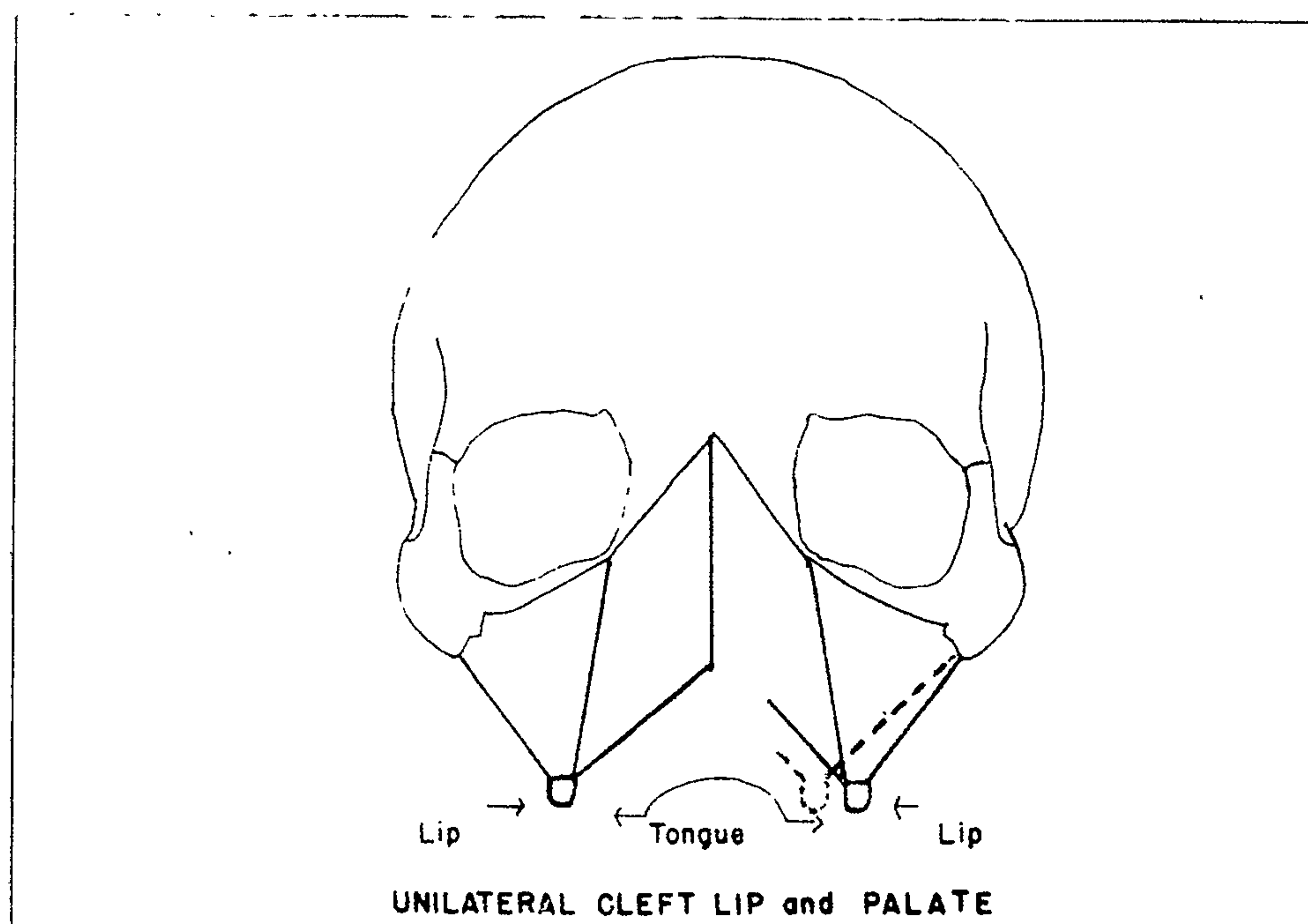


Fig. 3. Shows diagrammatically how crossbite is produced. The alveolar ridge on the cleft side drifts medially under the compressive force of the lip. This is represented by the broken lines. (Hagerty, 1957.)

and palate cases cannot be due to reduced growth potentials. The narrow dental arch may be attributed to a change in maxillary bone position and concomitant inhibition of vertical alveolar growth. Subtelny and Brodie (1954) directed attention to this aspect of the problem. Their conclusions, like Harvold's, were based on cephalometric x-ray techniques. They noted that the moulding influence of the repaired lip sometimes was of sufficient strength to over-rotate the segments medially. The alveolar process of the smaller segment may become contained within the premaxillary alveolar element of the larger segment. The inhibition of alveolar growth and development resulting from arch collapse has also been cited by Johnston (1958), Posen (1957) and Swanson (1958.)

In order to consider the vertical component of crossbite in the coronal plane or the asymmetry in the facial skeleton, Aduss and Pruzansky (1967), Coupe and Subtelny (1960), Harvold (1954) and Nakamura, Savara and Thomas (1972) as well as Wiepner and Hollmann (1975) took cephalometric radiographs in the frontal plane. They indicated that the bizygomatic distance of cleft patients fell within normal range while the nasal cavity, bony naso-pharynx width and bizygomatico-maxillary distance were increased in operated and non-operated clefts.

Wiepner and Hollmann's study also showed appreciable narrowing of the dental and alveolar arches medial to the molars. Harvold (1954) considers this narrowing to be attributed to a change in maxillary bone position and a concomitant inhibition of vertical alveolar growth. Severe scar contraction in complete clefts may cause asymmetry in the facial skeleton outside the alveolar process. Harvold found that the lower part of zygomatic bone at the key ridge may be moved 1.0 to 1.5 mm medially. This was confirmed by Subtelny and Coupe (1960) when they observed slight medial displacement of the cleft side

in unilateral complete clefts in their measurements from the lateral limit of the nasal cavity and zygomatico-maxillary suture to the vertical midline. However, these movements were not significant.

Derichsweiler (1964) observed that malocclusion is present from the very beginning. The crossbite seen in cleft patients can be seen in the new born prior to the operation and is not due to the consequence of the operation. The causative factors, such as the insufficiency of the transverse support of the alveolar arch and missing midline palatine suture which introduces a contradictory growth pattern, exist prenatally. Some causative factors appear at the least after birth such as pressure from the cheek muscle, and retarded growth of maxilla with crossbite appearing later in life. He emphasized that we must assume that the basis for a normal occlusion and relationship is not present from the beginning. Therefore, the malformation cannot usually be eliminated by either early treatment or osteoplasty.

Swanson, MacCallum and Richardson (1956) examined 111 cleft cases at a mean age of 8.2 years prior to palate surgery. They concluded that the constriction of the maxillary arch follows the repair of the cleft lip. They observed that the maxillary first molars erupted often in correct bucco-lingual relationship although a posterior crossbite occurred in the deciduous dentition. This can be interpreted in many ways:-

- (i) It may mean that early surgery does not inhibit the lateral growth in this important area of active bone proliferation prior to the eruption of the first molars.

- (ii) Lip surgery causes rotation of the buccal segments (maxillae) so that the distal ends at which appositional growth will take place flare outward resulting in greater maxillary arch width across the first molars.
- (iii) Since the nasopharynx is relatively wider than normal prior to repair, sufficient width is maintained despite surgery.

Ranta, Oikari and Haataja (1974) in their material observed that crossbite of the first permanent molars was always preceded by severe crossbite of the deciduous molars.

Swanson, et al (1956) considered the fact that rapid expansion of the maxillary arch does not lead to breakdown of surgical repair and the expansion has been shown to be a result of the repositioning of bony segments. Therefore the fibrotic contracture of palatal scar tissue is relatively unimportant. They also observed that the scars of the upper lip repair and perverted function of the entire orbicularis oris complex led to the tipping of lower incisors. The increase in mandibular plane angle and a corresponding decrease in the incisor-mandibular plane angle can be considered a desirable compensatory mechanism.

Ross (1975) emphasised that surgery is somehow responsible for poor arch development. Inhibition of growth, however mild, added to intrinsic maxillary hypoplasia which may be present, places severe demands on the dentoalveolar adjustment mechanism and interferes with the development of good occlusion.

Burston (1959) noted that a malocclusion at this early age will be likely to produce a profound effect on the developing alveolus and lead eventually to a gross collapse of the maxillary arch, so often seen in later years.

Pruzansky and Aduss (1964) noted in patients with only conventional treatment that the original width of the alveolar cleft was not related to final arch form following lip repair. Previously it was thought that the wider the alveolar cleft the greater the amount of undermining required to repair the lip. Therefore, the greater the tension across the midline and hence a greater incidence of maxillary collapse would follow. Pruzansky and Aduss did not support this idea.

Ranta, Oikari and Haataja (1974) observed that the original width of the cleft did not influence the upward rotation of the alveolar ridge but it did influence the narrowing of the cleft, being more pronounced when the cleft was originally wider.

Pruzansky and Aduss (1964), studying the deciduous occlusion, noted that 1/3 of their 33 cases demonstrated no crossbite. In 8 cases the crossbite was limited to the canine only. Total unilateral crossbite combined with anterior and buccal crossbite was found in only 13 cases or 30.2% of the entire sample. They concluded that the prevention of maxillary collapse before the surgery of the cleft palate, in the majority of cases, is not necessary.

Friede and Johanson (1974) and Kling (1964) evaluated cases in which treatment entailed orthopedic correction and bone grafting. Most of their patients had a more-or-less pronounced rotation of the maxillary jaw segments with anterior (88.5%) and later (57.7%) crossbite present. Kling concluded from her own data that her results did not seem to differ very much from those achieved earlier with less advanced methods of treatment. Compared with Aduss and Pruzansky (1967), her results were less impressive than those achieved by the more "conventional method".

However, Nylén, Körlof, Arnander, Leanderson, Barr and Nordin (1974) obtained 31% for frequency of anterior crossbite and 28% for buccal crossbite in their mixed dentition primary bone grafted group. They proceeded to compare these figures with those obtained by Bergland (1967) and Pruzansky and Aduss (1967) who examined non-grafted deciduous dentition cases. The figures presented by Bergland, (anterior crossbite 29%, buccal crossbite 35%) and Pruzansky and Aduss (anterior crossbite 26%, buccal crossbite 40%). These figures were claimed to be lower for the anterior segment and higher for the buccal segment when compared to those of Nylén, et al.

Therefore, Nylén, et al concluded that their results were promising and felt justified in continuing their bone grafting procedures.

Hellquist and Skoog (1976) showed in their periosteoplasty group lower incidence of crossbite when compared to Huddart and Bodenham (1972) who used presurgical orthopedic treatment. In their control groups the incidence of no crossbite was similar, i.e., being 10% in the Hellquist and Skoog group and 11.8% in the Huddart and Bodenham group.

In contrast to this, a surprisingly high frequency of no crossbite (25% to 40%) was reported from other studies of the deciduous occlusion in complete unilateral cleft lip and palate patients subjected to only surgery. (Bergland, 1967; Pruzansky and Aduss, 1967; Mazaheri, Harding, Cooper, Meier and Jones, 1971 and Nordén, Linder-Aronson and Stenberg, 1973.)

Fögh-Andersen and Dahl (1964) analysed 119 cleft patients and noted that 87% presented with a typical asymmetrical lateral crossbite of the lesser segment in the deciduous dentition. In the permanent dentition the crossbite apparently increases

in severity with time, i.e., asymmetrical bilateral crossbite. They explained this to be probably the result of lingual eruption of premolars on the non-cleft side, in many cases owing to crowding in the arch as well as the effects on the alveolus due to palatal surgery. Dahl also noted that 89% of unilateral complete clefts presented with anterior crossbite as compared to 40% in cleft palate only. He explained this to be the result of scar tissue, which keeps the upper incisors at a steeper angle than normal. This anterior crossbite may result in a sagittal forced bite and if several incisors are in crossbite there is a risk of inhibiting growth in the upper incisor region. He advocates incisor correction in early mixed dentition.

It should also be noted that vomer flaps were employed in their reconstruction and Aduss and Pruzansky (1967) and Bergland (1973) suspect these flaps to have a restrictive effect on the growth of the midface.

Troutman (1974) observed that the incidence of buccal crossbite was greater in his sample than samples reported elsewhere - 47.8% as opposed to 29% - 39.6%. He questioned whether the effect of presurgical passive and active maxillary treatment alone could improve the buccal crossbite incidence. In 17 of the 22 patients chosen for the study, lateral maxillary segments were in satisfactory relationship prior to surgery and the passive type of appliance was utilized. Five patients were considered to have a degree of collapse of the cleft maxillary segment and were treated with an active jack-screw type of orthopedic appliance.

In patients treated with passive appliances there was a 31.1% increase in the percentage of the patients with no crossbites and a 30.2% decrease in the percentage of patients with complete buccal crossbites when compared to controls who had received no type of early treatment. Patients treated with the maxillary expansion appliance prior to lip surgery all showed some type of crossbite in the deciduous dentition. Overall, in the treated group there were 20.5% more patients without any type of crossbite and 29.7% fewer patients with complete buccal crossbites than in the group of patients who received no early treatment.

In conclusion, Troutman states that good arch alignment in cleft patients should be maintained prior to and immediately following lip surgery and, if possible, following palatal surgery. If the cleft lateral segment is collapsed prior to lip surgery, expansion of these segments should be delayed at least until the complete deciduous dentition has erupted.

Nordén, et al (1973) investigated the deciduous dentition in children with complete clefts of lip and palate when treatment had been confined to surgery of the soft tissues. When the results were compared with similar materials of Bergland (1967) and Pruzansky and Aduss (1967) very good agreement was found in the frequency of crossbite alone. The frequency of about 44% in Pruzansky and Aduss' study is almost the same as in the Nordén, et al material (43.7%) while Bergland's figure is lower (about 35%). The combination of crossbite and frontal inversion is more frequent in the Nordén, et al material (about 31%) than in the studies of Bergland (29%) and Pruzansky and Aduss (26%), but the differences are not statistically significant.

Lower incidence of crossbite and frontal inversion were noted by Nordén, et al (1973) than in subjects where pre-surgical orthodontic treatment of the jaw and early bone grafting were undertaken. (Kling, 1964.) However, the differences between the subjects are not statistically significant. Robertson (1971) reported that in his series, the insertion of a bone graft did not prevent the occurrence of crossbite, in fact he found it to be more prevalent.

Hotz and Gnoinski (1976) compared two different surgical procedures where in one group the lip was closed at three months and the palate between 2-2½ years of age and in the other group the lip was closed at six months, soft palate at 18 months and hard palate between 6-8 years of age.

Hotz, et al found great differences in crossbite between the two groups, the latter group definitely being lower for anterior crossbite in unilateral clefts and deciduous molar crossbite in bilateral clefts. These differences are particularly noteworthy since in the second group no orthodontic treatment was applied after soft palate closure at 18 months of age, whereas in the first group, occlusion of deciduous teeth has been corrected in many cases. A high incidence of canine crossbite was also noted and considered to be due to the Burian flaps (consisting of a small mucoperiosteal flap and large vestibular mucosal flap) which was used for alveolar repair at the time of lip closure. This variation in surgical procedure has since been abandoned.

Bergland (1967) looked at the relationship between surgical procedures and the degree of post-operative deformity of the maxilla. He used a Le Mesurier repair of lip and a modification of Veau's Vomer flap repair on the hard palate at 7 months of age and Von Langenbeck technique for soft palate at 3 years, 2 months of age. Examining the deciduous dentition at a mean age of 5 years, 5 months, he observed that 60% of the cases

presented with no crossbite or slight crossbite of canine only, and the remainder with anterior or lateral crossbite. Among the latter group there was hardly a single case of really deformed maxilla. Most had good arch alignment but the upper jaw seemed too small to fit the lower jaw. The crossbite group may indicate that cleft palate malocclusion following modern surgery is a question more of maxillary size than of maxillary shape. This is consistent with the clinical manifestations in the permanent dentition where the achievement of maxillary overjet is still the most difficult and frequent problem. He also observed that good occlusal conditions in the deciduous dentition does not eliminate later orthodontic treatment but only simplifies it.

Bishara, Krause, Olin, Weston, Van Hess and Felling (1976) examined normal, unoperated complete unilateral cleft lip and palate and unilateral cleft lip and alveolus cases. In unilateral cleft lip and palate the maxillary relation and size was not different from the normal. However, the mandible was retruded and its plane steeper, with the incisors lingually inclined. In unilateral cleft lip and alveolus there was a tendency for protrusion and an increased maxillary depth. The mandible was normal with incisors labially inclined. Bishara (1973) noted in isolated cleft palate cases that both the maxilla and the mandible were retruded and that the mandibular plane was relatively steep. Chierici, Harvold and Vagervik (1973) considered that the facial skeletal relations in cleft-palate-only cases might in part be "mechanically compensatory" to the defect itself, resulting in the above characteristic. Clinical judgements of dental models indicated the unoperated complete clefts expressed the same tendency for a slight medial collapse of the maxillary segment on cleft side and a lateral rotation of the premaxillary segment on the non cleft side.

Therefore, sometimes what is considered to be the "untoward effects of surgical management" such as crossbite may be also present perhaps to a lesser degree in the unoperated individuals.

(c) Prevalence of Crossbite in Deciduous and Mixed Dentition in Operated Patients

Bergland (1973) and Bergland and Sidhu (1974) tried to record longitudinally the changes in frequency of various types of crossbite from deciduous to mixed dentition in unilateral cleft lip and palate. They noted that contractile forces following lip repair ceased before the end of the first year of life. The maxillary segments stopped moving medially and even the palate repair at two years did not seem to influence the segment position. However, in most cases the stability in the deciduous dentition occlusion comes to a sudden end with eruption of upper incisors. Bergland and Sidhu (1974) noted that the changes in anterior crossbite are nearly doubled from 40% to 78%, mainly at the expense of the "no-crossbite" group. They explained this as being due to an increasing retrusive position of the maxillary complex occurring in cleft subjects and a considerably steeper than normal path of eruption of the upper incisors. An inhibitive influence on the forward growth of the premaxillary part of the maxilla by the vomer flap operation cannot be disregarded either. The increased frequency of buccal crossbite, as the deciduous dentition proceeds to the mixed dentition stage, is relatively slight.

Bergland and Sidhu suggested that this increase was due to the retrusive positioning of the maxilla contributing to a relative forward shift of the mandible and thereby resulting in an impairment of the borderline buccal crossbite.

They concluded that the apparent deterioration of the maxillary deformity occurring in the early mixed dentition stage seems to be confined to the incisors and their adjacent tissue, i.e. the alveolar process.

This confirms the clinical experience that the premaxillary area is highly susceptible to changes in teeth relationship and there is considerably higher degree of stability in the lateral regions of the maxillary arch in unilateral complete clefts.

These observers concluded that early mixed dentition is a crucial stage in the development of the cleft malocclusion.

Ranta, Oikari and Haataja (1974) analysed 221 cases of unilateral cleft lip and palate. All the operations took place at the age of 1.5 - 2.0 years using the Cronin-Brauer push-back technique. About half of the children under 7 years were found to have some degree of crossbite or edge-to-edge bite and about 75% of the 7 - 9 year olds. Their figures for the prevalence of posterior crossbite in the deciduous dentition was about 25% and for the anterior crossbite in the deciduous and mixed dentition was 14%. An examination of the prevalence of anterior and posterior crossbite in deciduous dentition reveals a very small difference between children under 5 and 5 - 7 year olds. It seems probable that when crossbite develops this occurs at a very early stage. The period of particularly fast growth in the maxilla and mandible is limited to the first four years. During this period the scar tissue may decrease the rate of growth of the maxilla so much in comparison with that of the mandible that anterior and/or posterior crossbite has time to form. Their slightly higher prevalence of posterior crossbite was explained to be due to the strong influence of the scar tissue on the lateral growth of the upper jaw, or may stem from the better compensatory change in the anterior than in the posterior region of the lower jaw.

In agreement with Bergland and Sidhu (1974) study, they observed that the frequency of crossbite was slightly higher for the permanent than for the deciduous incisors. This may be due to the effect of the scar tissue in the upper jaw which slows down anterior growth. However, the crowding which fairly often occurs may in some cases cause the incisors to erupt more palatally than if there had been adequate space in the dental arch.

Their theories agree with those of Jolley (1954), Lynch and Peil (1966) and Kremenak, et al (1967), that scar tissue creates a contracting force on the maxilla and alveolar process. This supports the theory of Ross and Johnston (1976) that the effect of the initial scar tissue contraction is not usually great but it has a long-term effect on the growth of the upper jaw. Therefore, the timing and type of operation has an effect on the development of occlusion.

Evaluation of the Arch Form and Occlusion

Traditionally there are three aims in the treatment of congenital clefts of lip and palate:-

- (i) the production of normal speech.
- (ii) the provision of a normal appearance.
- (iii) the procuring of normal dental function.
(Dey, 1974.)

The result of cleft palate treatment can be evaluated from many different aspects, e.g. speech and appearance, and perhaps the easiest to assess is the occlusion with several classifications developed for this purpose. The success or failure of treatment can be related to the frequency with which crossbites appear in the sample studied.

The descriptive classification of occlusion by Pruzansky and Aduss (1964) involved dividing the occlusion into six categories:-

- (i) no crossbite present
- (ii) canine crossbite only
- (iii) buccal crossbite only
- (iv) anterior and buccal crossbite
- (v) anterior and canine crossbite
- (vi) incisor crossbite only.

Matthews, Broomhead, Grossman and Goldin (1970) in contrast divided the occlusion into:-

- (i) Class A - where all the segments of the maxilla are in normal occlusion with the mandible.
- (ii) Class B (1) - the tooth bordering the cleft on the lesser segment is in lingual occlusion.
- (iii) Class B (2) - normal occlusion of the greater segment but lingual occlusion of the lesser segment.
- (iv) Class B (3) - the maxillary arch is perfect but is too small.
- (v) Class C - an overall Class III occlusion of all segments of the maxilla and in addition, collapse of some part of the small maxillary arch.

Hotz and Gnoinski (1976) used a simple score which involved counting the number of crossbite teeth per patient. The crossbites were recorded separately for deciduous molars, canine and anterior teeth.

While these classifications describe the type of deformity present, they do not consider its extent. To overcome this, a numerical classification of crossbite malocclusion has been developed by Huddart and Bodenham (1972). Each maxillary tooth was assessed on a points basis, depending on its position relative to its antagonist in the lower jaw. A rating from +1 to -3 was used according to whether there is overjet (+1), normal overlapping contact (0), cusp-to-cusp contact (-1), crossbite contact (-2), or crossbite out of contact (-3). (Fig. 4.) When the classification is used for unilateral cleft palate cases with only deciduous teeth present in the mouth, it is divided into three segments:-

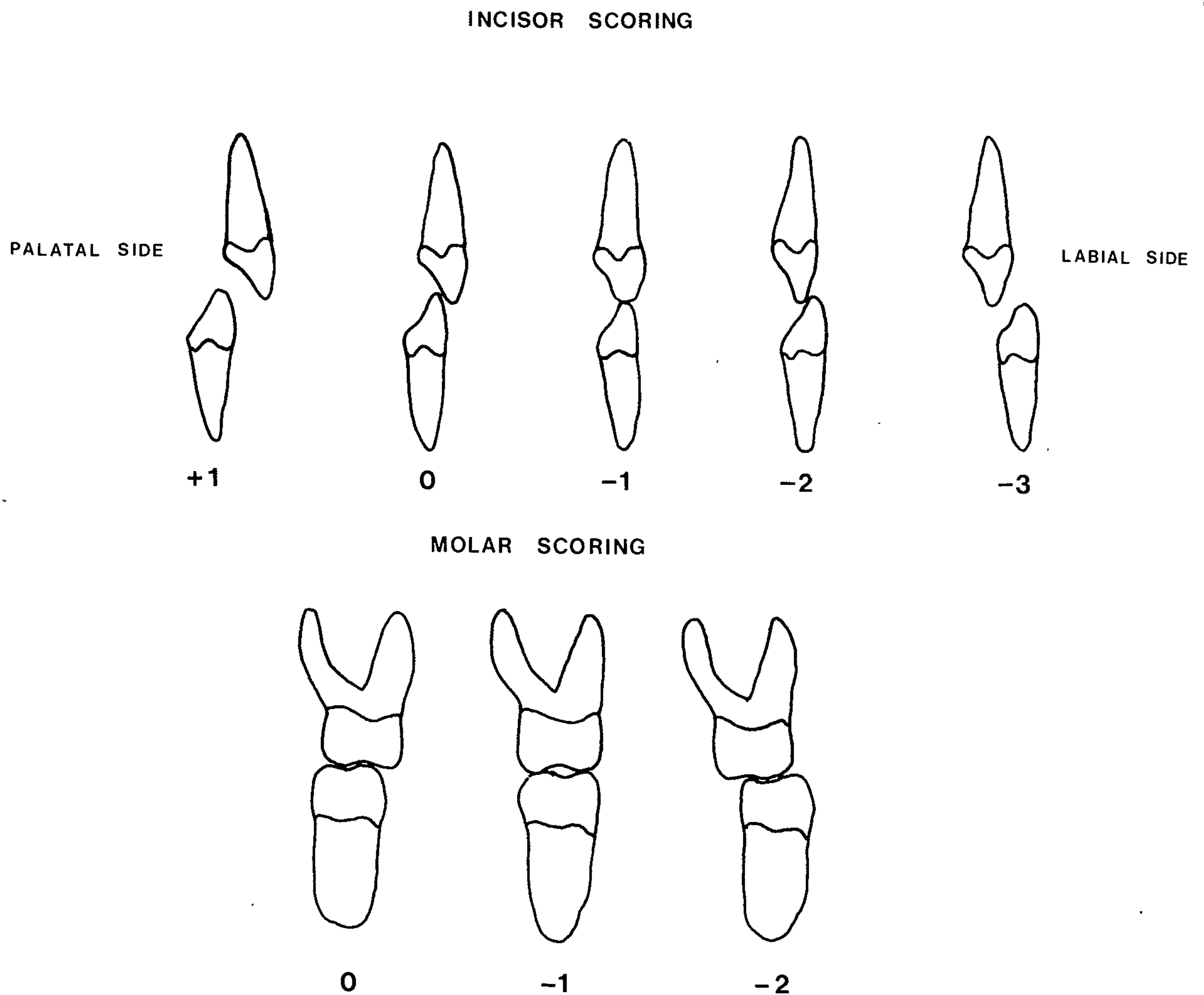


Fig. 4. Numerical classification - occlusal scoring.

Occlusal scoring of crossbite malocclusion as applied to the deciduous maxillary arch. The upper diagram shows the degree of severity in the antero-posterior relationship of the anterior teeth and the lower diagram shows the bucco-palatal relationship of the molars.

(Huddart and Bodenham, 1972.)

- (i) The incisor segment - concerned with two deciduous central incisors.
- (ii) The buccal segment, non-cleft side - concerned with deciduous canine and both deciduous molars.
- (iii) The buccal segment, cleft side - concerned with the same teeth as for (ii). (Fig. 5.)

A score for each segment is produced by adding the scores for the individual teeth within the segment, and a total score for the occlusion is obtained by adding the segmental scores. (This method of assessment was used in this investigation.)

Additional comparisons can be made to the following studies which used the Huddart and Bodenham (1972) numerical classification of occlusion (Table 2, page 5.5):-

- (i) Huddart and Bodenham (1972) group of 34 cases where the lip and palate are repaired separately.
- (ii) Huddart (1967b) compared 8 groups depending whether lip and palate was repaired separately or simultaneously and whether presurgical orthopedics was used.
- (iii) Robertson (1973) evaluated presurgical orthopedic cases and compared them to only surgically treated cases.
- (iv) Friede and Johanson (1977) examined bilateral and unilateral complete clefts treated with a three-stage surgical procedure including a single layer vomer flap. They compared three groups of repair,

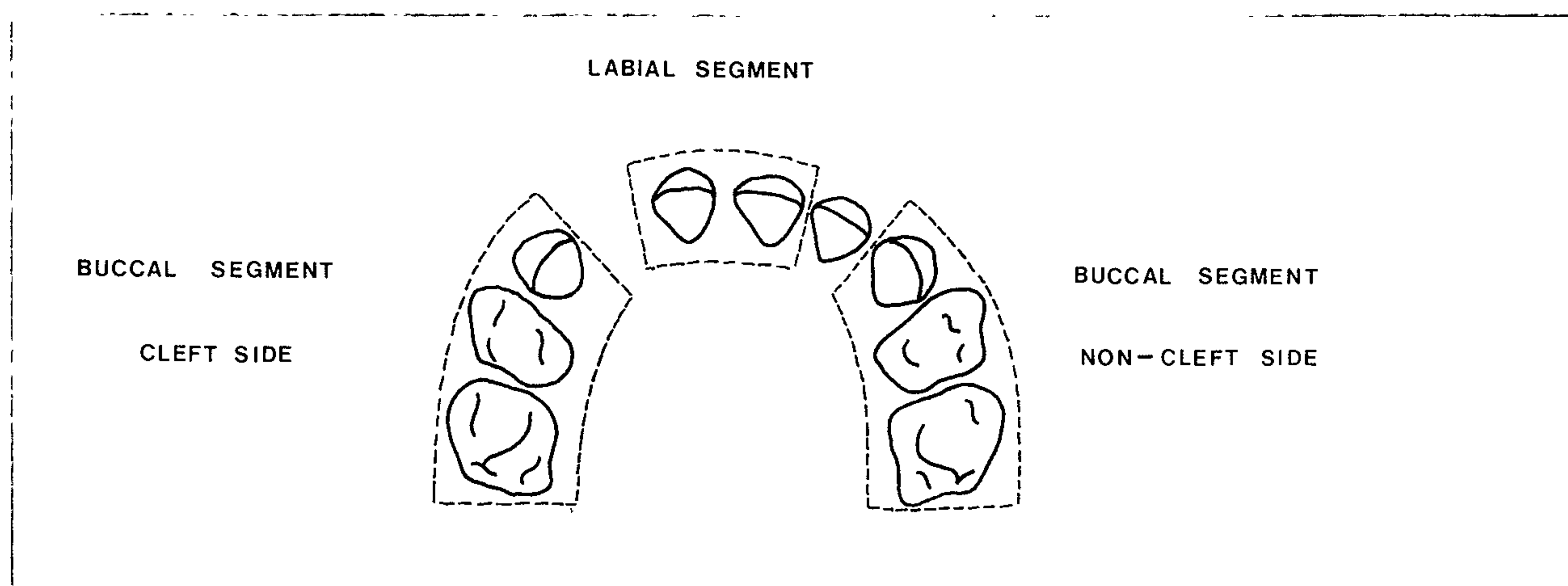


Fig. 5. Numerical classification - subdivisions of the maxillary arch.

Subdivisions of the deciduous maxillary arch for the assessment of crossbite malocclusion in unilateral cleft lip and palate cases. Each buccal segment consists of the canine and the two deciduous molars. Whilst the labial segment is made up of the deciduous centrals. The deciduous lateral incisors are not included in the constitution of the segments as their presence and position is quite variable in cleft cases.

(Huddart and Bodenham, 1972.)

viz., those whose repair involved vomer flap, vomer flap and primary bone grafting, neither vomer flap nor bone grafting.

- (v) Hellquist and Skoog (1976) investigated the influence of periosteoplasty upon the growth of the maxilla and the prevalence of malocclusion in the deciduous dentition. The patients were divided into groups and compared. Those whose reconstruction was combined with periosteoplasty were subgrouped according to whether periosteoplasty was performed once or twice and compared to a control group not subjected to periosteoplasty.

Comparisons of these studies to the present investigation are considered in the discussion of the results.

Aims of Treatment in Cleft Patients

An important aim of a long-term integrated treatment plan for cleft cases should be to reduce the overall treatment time without compromising the final results. We now have sufficient knowledge of occlusal development in cleft subjects to distinguish between periods of stability and stages where impairment has to be anticipated. Therefore, our basic principle is to determine developmental stages at which therapy could be employed most effectively. (Bergland, 1973.)

Troutman (1974) emphasizes the fact that the reason for success or failure in the treatment of patients with cleft lip and palate are multifactorial. Only when we understand all of the facts concerning the mechanisms which cause clefting, genetic growth factors and the environmental factors which cause changes in developing persons, will we be able to prescribe treatment on an individual basis and expect consistent success.

Robertson and Fish (1975) emphasized that the final results of treatment depend not only on the treatment procedures employed but also on the type and degree of deformity at birth, the inherent post-natal growth pattern of the cranio-facial complex of the individual patient and the effects of treatment both surgical and orthodontic, on this growth pattern.

Associated Techniques in Rehabilitation

(a) Bone Grafting

Probably the most significant suggestion made in the 1960's was that early bone grafting might well improve the results achieved, by preventing the occurrence of a crossbite in the deciduous canine area on the cleft side, allowing the teeth to develop and migrate through the grafted bone and providing support for the base of the alar cartilage. (Norden, et al, 1973.)

Some writers recommended the insertion of bone at the time of the lip and anterior palate repair ("primary" grafting), while others preferred, in a first stage, to prepare a bed for the graft which was then inserted at a subsequent operation.

Pickrell, Quinn and Massengill (1968) in a four-year follow-up of 25 bone grafted cases observed that the graft in the maxilla did not increase in size concomitant with facial growth development, and its orthopedic effect decreased as its incorporation progressed. This was also confirmed by Rehrmann, Koberg and Koch (1970) and Rehrmann (1971). They had to actually insert a retainer to prevent collapse of maxillary segments.

Robertson and Jolley (1972) and Robertson (1973) observed that children with bone grafts had poorer occlusion and facial development. They concluded that bone grafting caused retardation in development of the maxillary arch. There was a higher incidence of anterior and buccal crossbite (Kling, 1964; Derichsweiler, 1964; Pickrell, et al, 1968; Robertson and Jolley, 1968, 1972; Rehrmann, et al, 1970 and Friede and Johanson, 1974.) Kling (1964) observed a remarkable increase

of lateral crossbite (88%) and mandibular pseudo-prognathism in 58% of the cases. It seems that early bone grafting affects the anterior position of the maxilla.

Matthews, Broomhead, Grossman and Goldin (1970) stated that:-

".....if primary bone grafting is ultimately abandoned it will be because the long term results do not justify it, not because of technical hazard."

Over 70% of their 5-6 year old patients had Class III malocclusion. They noted that the arch had been maintained and from x-ray the bone graft had persisted and there was no evidence of maxillary collapse. However, the results were dentally, and sometimes cosmetically, unsatisfactory.

Ross and Johnston (1972) pointed out that if the graft is placed in an area where maxillary growth does not occur after the first or second year of life there should be no direct interference with long-term maxillary development. Wherein lie the many poor results? Is it only a question of the difference in operative procedure (Pfeifer, 1972) or an insufficient bony transformation of the graft (Schrudde and Tranner, 1972) or a case where the grafted bone does actually hold the anterior ends of the segments together like a claw and thus stifle their intrinsic growth potential? (Koberg, 1970.)

Schmid, Widmaier, Reichert and Stein (1974) from 1962 followed up 87 children who were treated by osteoplasty of the alveolus and hard palate. In their work great emphasis was placed on atraumatic handling of the involved tissue. Method of closure involved mobilizing two flaps at the vomer and the lateral edge of the gap, thus obtaining two layers of mucous membrane to

line the oral and nasal cavity at 8 months. At 4 years of age the velum was closed using Widmaier's (1961) method whereby the palatine arteries remained intact. They claimed that the reason for their good results was undisturbed blood supply to the upper jaw (which differs considerably from other methods) and the prior orthodontic treatment when indicated.

There are many advocates of secondary bone grafting after development of the adult dentition, especially as an aid to orthodontic and prosthetic construction. (Obwegeser, 1971 and Matthews, 1974.)

Chalmers (1965) showed that grafted bone has virtually no capacity for growth unless subjected to great stress.

Chierici (1977) hypothesized:-

".....that tension facilitates the extension of bone-inducing stimuli and influences the differentiation of tissue in intra-alveolar transplants."

Working on rhesus monkeys he observed that biophysical forces contributed to bone formation. As the new bone formed from the edge of the cleft and replaced the resorbing transplant, a new suture developed between the extended cleft margin. This suture could permit growth adjustment.

Vargervik (1978) applied the above theory to 15 maxillary clefts and found similar results.

Therefore, it seems apparent that if a suture can be retained or developed in the cleft area with scarring kept to a minimum, bone across the cleft could be established at any age without significant inhibition of growth.

(b) Periosteoplasty

Periosteoplasty introduced by Skoog (1969) also known as "boneless bone graft", is a soft tissue procedure with the establishment of periosteal continuity between the maxillary segments. Doubled layered periosteal lining across the maxillary cleft is created by the reconstruction of the nasal floor by two mucoperiosteal flaps from the deep aspect and, in addition, a periosteal flap from the antero-lateral aspect of the maxilla.

Hellquist and Skoog (1976) in their 5-year follow up study of complete unilateral clefts treated by periosteoplasty examined the effect of periosteoplasty on maxillary growth and deciduous occlusion. In the repair of small alveolar clefts, periosteoplasty was combined with the implantation of Surgicel. In large alveolar clefts periosteoplasty was performed in two stages. The analysis of occlusion revealed an increase of buccal crossbite after second periosteoplasty while the buccal crossbite after one periosteoplasty was smaller than in the controls. This demonstrates the importance of the size of the original defect and the possibility that repeated periosteoplasty may cause a locking of cleft segment and interfere with transverse growth of the maxilla. (Prydsö, Holm, Dahl and Fögh-Andersen, 1974.)

Skoog (1967) and Hellquist (1971) observed that periosteoplasty seems to reduce the tendency for maxillary collapse and aids good segmental positioning at the stage of palate repair. However, following this surgical intervention, the difference between the periosteoplasty series and controls disappeared over

a period of 2-3 years. Apparently, the new bone within the alveolar cleft did not withstand scar contraction of the palate repair. This mechanism is still being studied.

Hellquist and Skoog (1976) concluded from their study that complete unilateral clefts treated by periosteoplasty demonstrated no retardation or impairment of maxillary growth. Results of this technique look promising although another ten years would be required to prevent convincing proof of these preliminary conclusions.

(c) Palato-Vomerine-Plasty

Holm, Dahl and Fögh-Anderson (1974) showed that by placing in contact the muco-periosteal flap from the hard palate and the vomer, the formation of morphologically normal bone without suture connection with the nasal septum results. They concluded that this may inhibit possible compensatory transverse growth in the other maxillary sutures and cause locking of the lateral segment to the nasal septum on the cleft side. Dahl (1970) confirmed this by showing that the maxillary base width was significantly reduced with a 91% incidence of crossbite on the affected side compared to 47% frequency among unoperated patients. Therefore, Holm, et al (1974) suggest that osseous closure should be avoided until sutural growth of the upper face has terminated.

Contrary to the finding of Holm, et al (1974) and Strenström and Thilander (1974), using vomer flaps covered with full-thickness grafts, observed almost no constriction of the maxilla. There was cleft-bridging and some regeneration in 8 of their cases with crossbite present in only 3.

Friede and Johanson (1977) found that the bone formation in some areas of the cleft does not seem to be of significant importance for facial growth. However, in the occlusal analysis only the bilateral clefts with some bony bridge showed more frequent occurrence of Class III relations than those without bone formation. They also observed greater growth retardation with their vomer flap group compared with the non-vomer flap group. Long-term clinical experience has led some authors to suspect that closure of the hard palate by use of a vomer flap can arrest midfacial growth. (Pruzansky and Aduss, 1967; Steinhardt, 1973; Bergland and Sidhu, 1974; Blocksma, Leuz and Mellerstig, 1975 and Peat, 1979.) Experimental studies have indicated that the growth retardation might be caused by surgical trauma to the septovomer region. (Sarnat and Wexler, 1968.) Whether the denuded vomer is disrupted from blood supply or mucosal scar tissue is forming, the retarding factor is still a matter of speculation. (Latham, Deaton and Calabrese, 1975.)

(d) Orthopedic Treatment

Efforts to prevent facial skeletal deformities through the use of a dental guide plane were first supported by McNeil (1956) who stated that:-

".....their function is to mould the deformed arch into correct anatomical alignment."

Later Burston (1959), being an adept advocate of the McNeil technique, stated that:-

".....once this collapse has been established, the teeth, particularly the canines on the affected side(s) will erupt into incorrect occlusion. This is of great importance because, as the infant grows older, appositional bone growth of the maxilla becomes increasingly important as contrasted with sutural growth. Malocclusion of the teeth at this early age will be likely to produce a profound effect on the developing alveolus and lead eventually to the gross collapse of the maxillary arch so often seen in later years."

Skoog (1967) believes that presurgical orthopedic treatment must be confined to cases with collapse of the maxilla.

However, early maxillary orthopedic treatment and the use of passive appliances to prevent postsurgical collapse continue to be a technique used by many throughout the United States and especially Europe. However, a controversy concerning the effects and advisability of performing neonatal arch manipulation currently exists. Although, its advocates claim that it tends to normalize feeding, tongue posture and swallowing and to guide physiological growth of the palatal segments, no conclusive evidence has been reported to support these claims.

Hotz and Gnoinski (1976) and Hotz, Gnoinski, Nussbaumer and Kistler (1978) advocated pre- and post-surgical orthopedic treatment and emphasized that such treatment would not be efficient in the long run unless it was strictly co-ordinated with adequately timed conservative gentle surgery. Their

objectives for presurgical orthopedic treatment were to monitor function, guide growth and the position of maxillary segments and delay surgery in order to allow intrinsic developmental potentialities to become manifested. As a rule, three plates were necessary until the eruption of the first deciduous molars and when it was considered four-fifths of maxillary development had occurred. They observed that maxillary collapse occurs mainly during the first three to six months after palatoplasty when the pull of the contracting scar is the strongest. They intercepted with a conventional retainer containing a pivot screw which was turned 0.25 mm per month to keep up with the growth of the arches. This was based on the fact that previously collapsed segments showed a greater tendency to relapse after expansion and that primarily retained ones, relapsed very little. Although good occlusal results were seen in the deciduous and early mixed dentition, arch instability was a problem, and they admitted to some arch collapse when retention was removed.

Huddart (1973) noted that there were three factors contributing to the width of the palatal cleft. These are tissue deficiency, lateral segmental displacement and an increased slope of the palatal shelves. It is possible to rationalize the use of presurgical treatment on the grounds that by altering some or all of the factors affecting the size of the cleft it is possible to reduce the severity of the original condition. In consequence, the reduction in width of the cleft in relation to the width of the tissue present makes the cases much more favourable for repair by surgical means. However, it appears that the changes which the orthopedic plate produces are essentially transient and have no permanent effect on the factors influencing arch form occlusion at 5 years of age. (Huddart and Bodenham, 1972; Huddart, 1974 and Peat, 1974.)

Peat measured the crossbite frequency in complete unilateral cleft lip and palate patients who had presurgical oral orthopedic treatment. His figures (at 1979) are shown in Table 3.

Table 3. Frequency of crossbite in presurgical orthopedic treated complete unilateral cleft lip and palate patients.

Dentition	Incisor Segment	Buccal Segment	Total Cases
Primary	30%	95%	74
Mixed	68%	94%	77
Permanent	35%	98%	57

Comparing with other workers, represented in Table 4, Peat concluded that presurgical oral orthopedics does not improve dental occlusion and that there was a progressive increase in incisor and buccal crossbite during growth.

Table 4. Frequency of crossbite in unilateral complete cleft lip and palate patients.

	Incisor Segment	Buccal Segment	Total Cases
Pruzansky 1964	25%	70%	59
Kling 1964	57%	88%	26
Bergland 1967	29%	64%	31
Nordén, et al 1973	31%	75%	16

Robertson (1973), Krischer and Shiere (1974) and Rosenstein (1975) all reported a significantly better occlusion in their orthopedic group when compared to non-treated cases. Robertson concluded that the difference in the long-term results may be due to the quality of surgery. He also admitted that some of the children in his 5-year group still had poor occlusion. His reasons were that their presurgical management was incorrect or that, superimposed on the cleft deformity, there may be variations in skeletal pattern, crowding or absence of teeth that affect the results achieved.

It is important to stress that in all of the long-term evaluation studies with early arch manipulation the presence of dental crossbite of the canines and/or buccal teeth is still present, although to a lesser degree.

Troutman (1974) concluded from his study that if the maxillary lateral segments in complete clefts are in good arch alignment prior to lip surgery, an attempt should be made to maintain these segments in an acceptable position prior to and immediately following lip surgery at least until, and if possible following palatal surgery. If the cleft lateral segment collapsed prior to lip surgery then, unless there is a need to expand and mould these segments to facilitate surgery, expansion of the lateral segments should be delayed at least until the complete deciduous dentition has erupted at which stage it can be more easily corrected and maintained with less trauma to the patient.

Ross (1975) concluded that the value of such a procedure is probably extremely limited since long term results appear to offer no improvement in arch shape beyond that which is obtained by lip closure alone. It only provides a guiding pressure and does not prevent the interference to maxillary growth and dento-alveolar adjustment which will occur following palate repair.

Pruzansky (1973) states that there are two schools of thought concerning orthopedic appliances:-

- (i) that cheiloplasty should be supported by pre- and post-surgical maxillary orthopedics with and without bone grafting, and
- (ii) that the effectiveness and benefits of such procedures are limited and the cost incurred is inordinately high for the value gained.

From the long-term studies, it appears that presurgical treatment facilitates lip and palate repair and does not appear to have any long-term adverse effects on occlusion despite a mildly inhibiting effect on tissue growth. (Huddart, 1979.)

In conclusion, the general concensus among investigators is that orthopedic pre-surgical treatment does not appear to have a long-term favourable effect, though, some consider that benefits can be gained and should accordingly be carried out when conveniently possible.

PRINCIPLES OF CLEFT SURGERY

The aim of modern surgeon is to carry out physiological reconstruction of the region affected by cleft, Fara (1972) suggested that the following conditions must be fulfilled:-

- (i) The time schedule for the individual operational steps must be divided taking into consideration the growth periods and in view of the fact that surgery slows down development for a short period.
- (ii) It must be decided, in each individual case, which formation will grow appropriately and improve its shape merely by being incorporated into the functional unit and which formation is actually deficient and has to be augmented.
- (iii) Tissue insufficiency must be supplemented either from the immediate vicinity (augmentation by a flap from the lateral side) or by transfer from remote parts of the body (bone grafting into the defect in the maxilla) or by transposition (retroposition of the short palate).
- (iv) Atypically placed or deformed tissues must be given a natural shape (elevation of the nasal cartilages) or moved in the correct direction (detaching the proximal cleft muscle - insertions of the lip and palate and folding down into a horizontal course.)

- (v) Better development of the individual formations must be stimulated by renewing the functional units in the vicinity (the effect of the reconstructed labial muscle upon development of maxilla) by reinforcing their base (the effect of the bone implanted under the base of the wing for development of the nose) or by adding tissue with vascular and nervous supply.
- (vi) The operation must be carried out physiologically; no tissue must be damaged; the periosteum in the vicinity of the dental buds must not be elevated; denuded areas must not be left to subsequent scarring and the extent of mobilisation must be decided in order to prevent tension in the suture line (the effect of lip pressure upon the lateral maxillary segment).

UNILATERAL CLEFT LIP SURGERY

To produce satisfactory repair of the single cleft lip, Steffensen (1953) listed certain criteria:-

- (i) Accurate skin, muscle and mucous membrane union.
- (ii) Symmetrical nostril floors and vertical nostrils.
- (iii) Symmetrical vermilion border and Cupid's bow reproduction.
- (iv) Slight eversion of the lip.
- (v) A minimal scarring which by its contraction will not affect the above criteria.

The surgeon who undertakes repair of cleft lip must consider not only the aesthetic and functional results for the lip alone, but also the effect on the dental arches and the dentition. (Hagerty, 1957.) When the lip is closed, an orthodontic device is brought into play which can produce overcorrection with medial displacement of the alveolar ridge or ridges and result in collapse of the dental arch. This is especially true when cleft lip repair is carried out before the midfacial bones are well ossified and able to resist pressure.

The repaired lip should be full and loose and thus avoid compression of the dental arch. It is interesting to note that in the development of surgical procedures to attain a normally functioning and aesthetically pleasing lip, a rather loose full lip has been obtained. (Hagerty, 1957.)

The cleft lip deformity has been separately considered to be a displacement of a normal complement of parts (Blair and Brown, 1930), a triangular tissue deficiency low on the lip border on the columellar side (Brown and McDowell, 1950) and a linear deficiency displacement in the line of the cleft (Clifford and Pool, 1959). Cosman and Crikelair (1965) observed that there is an absolute deficiency of tissue in the unilateral complete cleft lip of a roughly trapezoidal shape as seen from the front and of a tapering tetrahedron as seen laterally. The magnitude of the defect is greatest beneath the nostril floor rather than at the vermilion border of the lip.

Numerous procedures of lip repair have been described. Some kind of flap is required to increase the vertical height of the lip.

In 1830, Dieffenback introduced lateral undermining incisions to allow sliding of the flaps together with less tension.

In 1844, Mirault was the first to establish the principle of using tissues from a site of abundance and shifting it to a site of paucity by means of a triangular flap from the lateral segment across the lower margin of the cleft.

Brown and Blair (1930) modified Mirault's triangular flap repair by refining it and making the technique more exact. These authors are credited with attempting to make a Cupid's bow and normal pouting appearance of the lip. They also introduced the methods for the correction of the alar deformity which is still an accepted approach today.

Hagedorn (1892) introduced a square or rectangular flap from the lateral lip segment. This technique was later popularized by Le Mesurier (1949.) In this procedure a quadrangular flap is developed from the lateral side of the cleft and rotated into the medial side of the cleft using a modified Z-plasty.

The Cupid's bow was surgically reconstructed and not preserved. Some difficulty was encountered in controlling the length of the lip. To overcome this problem Maes, Richey and Shaw (1971) described a method of reversing the basic landmarks of the Le Mesurier procedure. With this procedure they have utilized the pre-existing Cupid's bow for the repair.

There have been others who have modified and clarified the Le Mesurier repair. (Steffenson, 1949 and Brauer, 1953.) These modifications seem to give consistently good results and undoubtedly this will always remain a popular repair.

Tennison (1952) transferred a triangular flap from the lateral to the medial side. Instead of one half of the bow being made from the lateral side and one from the medial, both came from the medial element. This eliminates the problems of the Le Mesurier method. The method of surgery used on the subjects in this present study is based on the Tennison method of lip repair. This is represented in Fig. 6.

Randall's (1959) procedure resembles that of Tennison by letting down the medial edge and wedging above it a triangle from the outer side. At the time Millard's (1957) rotation-advancement operation was considered the most outstanding contribution to improved cleft lip repair. This method is represented in Fig. 7. Millard refers to the medial element as the strong element of the lip. In his first step he brings down the medial side of the lip by using a long oblique incision and the defect remaining is higher in the lip. Closure is by advancement from the lateral side after undermining. This procedure tends to produce a more natural alar base and floor of the nose than do the other operations.

Davies (1971) criticised Millard's technique by stating that approximating the two convex curves appears to leave the majority of the bulk in the centre of the lip thereby producing a tight lower third of lip. The repair tends to fall somewhere on the scale between a

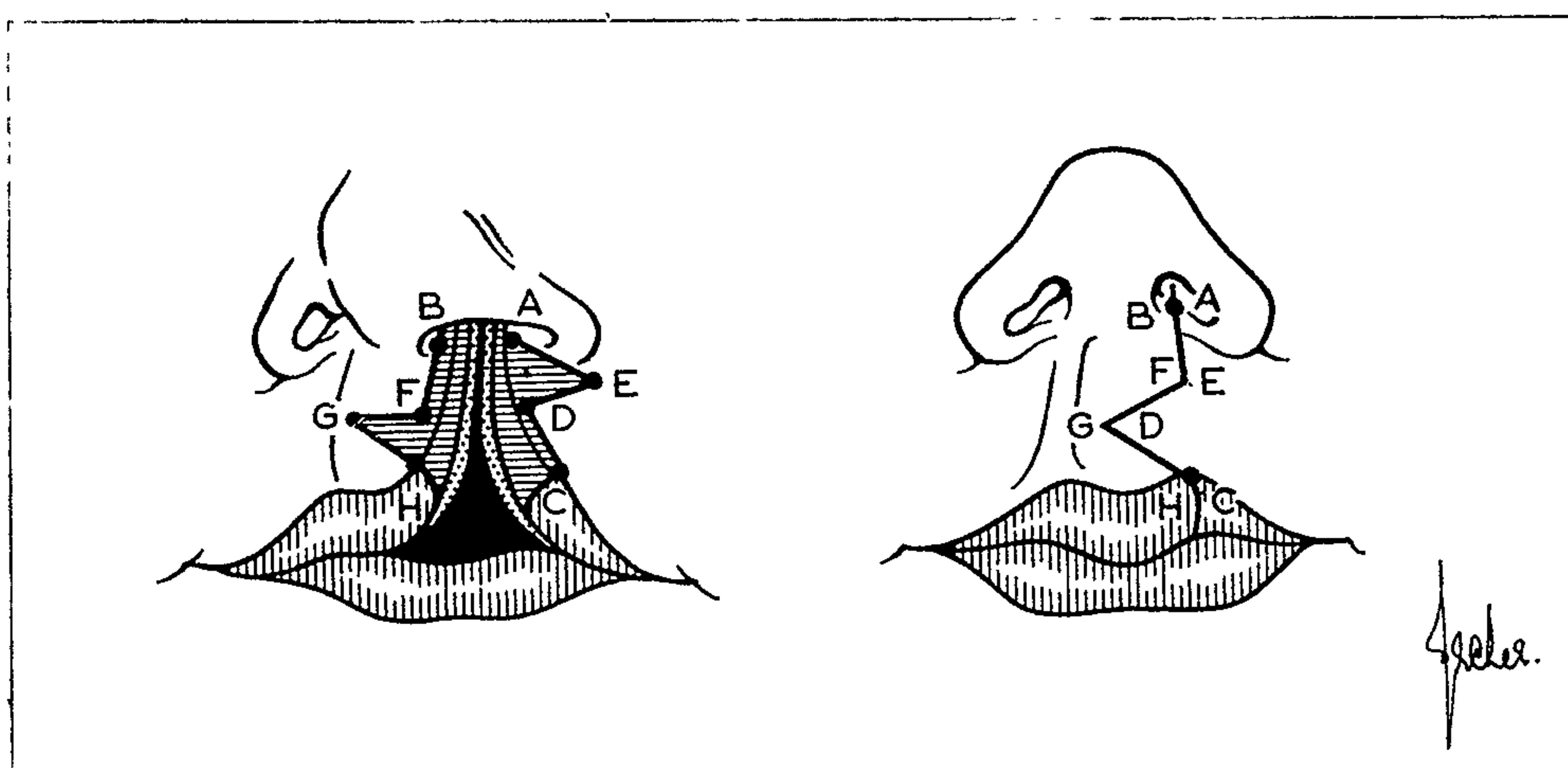


Fig. 6. The Tennison Operation.

- A,B. Points at the nostril margin approximation of which will constitute a satisfactory floor.
- H. Peak of the Cupid's bow on the cleft side.
- F. So that BF is half the height of the lip on the uncleft side (usually 4 to 5mm). F is usually 1 or 2 mm from H.
- G. So that GH equals FG equals BF. (BG should be 2 mm longer than BF).
- C. On the red margin where vermillion is of normal thickness.
- D. On the red margin so that CD equals BF.
- E. So that AE equals DE equals CD. (CE should be 2 mm longer than CD).

(Holdsworth, 1970.)

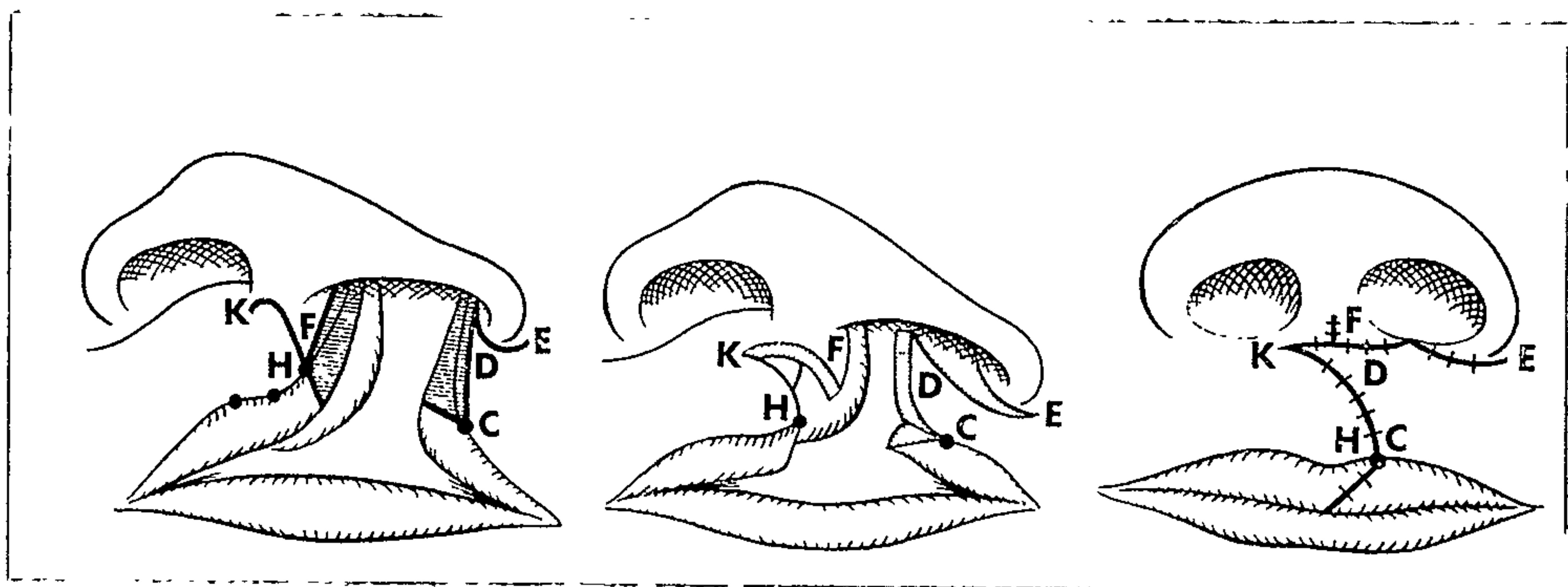


Fig. 7. The Millard Operation.

- F.H. Peak of the Cupid's bow on the cleft side.
 - H,K. Extends upwards along the red margin and curves under the columella to permit the opened lip to drop down so that the red margin becomes symmetrical.
 - D. On the lateral side of the cleft in the nostril floor. (In a partial cleft this point could be in a Simonart's band.)
 - D,E. Extends beneath the alar base, long enough to permit D to meet K.
 - D,C. Along the red margin, equals H,K.
- (Holdsworth, 1970.)

straight line repair and a Z-plasty and this suffers from the advantages and disadvantages of both. Kaplan (1978) and Randall (1971) noted a shortening of the vertical height on the repaired side in Millard's repair which is due to the contraction of the long straight line.

To overcome tension and constant tendency to fore shortening, Bernstein (1970) modified the Millard operation by introducing a small triangular flap at the bottom of the philtrum which provides extra lip height and also contributes to its maintenance after healing.

Kaplan (1978) explained the occasional shortness of vertical height in the Millard repair and excessive length in the triangular flap repair as due to differential lip growth after repair. He also theorized that muscle forces and skin tension are most important factors controlling lip growth but genetic tendencies have some contributing effect. Measuring 112 unrepaired unilateral complete cleft lips he showed that there was 10-20% greater growth in the transverse direction parallel with the orbicularis muscle than in the vertical direction perpendicular to this muscle. Therefore, procedures such as those of Le Mesurier and Tennison that transpose tissue from the transverse to the vertical will lead to excessive vertical growth.

Recently there has been greater emphasis in muscle reconstruction. Randall, Whitaker and LaRossa (1974) and Asensio (1974) believe that the abnormal insertion of the orbicularis oris muscle fibers to the bony margins of the cleft lip must be detached and rotated to the proper transverse direction in both primary and secondary cleft lip reconstruction.

Randall (1971) introduced a technique combining the Millard skin incision with a triangular muscle flap to help reconstruct the orbicularis oris muscle. The vermillion and mucosa is trimmed off

the lateral cleft margin and a sizeable flap of muscle is directed from the margin and sutured into a tunnel made with fine scissors at the level of the vermillion border. The tendency of the Millard incision to contract is countered by the tendency of the rebuilt orbicularis oris to lengthen the lip.

This technique (Randall, 1974a) has made a significant contribution to cleft lip repair. The operation adds bulk to the lower third of the lip, achieves a good rotation of the alar base and relieves much of the tension on the suture line. No significant depression of the alar on the cleft side has been noted.

Lip adhesions and two-stage lip repairs were first described by Randall (1965.) It is advocated in exceptionally severely distorted cases. It creates a narrower cleft thus facilitating repair, i.e. under less tension and with a minimum of tissue undermining. It moulded the dental arch to a more normal position and was considered by Walker, Collito Mancusi - Ungaro and Meijer (1966) as a form of orthodontic management replacing presurgical orthopedic appliances. However, Maisels (1967) prefers the same objectives to be achieved by orthodontic appliances.

In an unpublished 1979 study, Peat analysed different methods of lip repair and their effects on occlusion. He measured the crossbite frequency in primary and mixed dentition stages as shown in Table 5.

He concluded that the Millard rotation-advancement method produced the highest frequency of crossbite and should be avoided if possible. Where possible a Z-plasty is recommended. Generally, the lip repair does not significantly effect skeletal growth whether it be a Z-plasty, quadrilateral or rotation-advancement repair.

Table 5. The frequency of crossbites in complete unilateral cleft lip and palate patients with different methods of lip repair.

Lip Repair	PRIMARY DENTITION				MIXED DENTITION			
	Incisor Segment	Buccal Segment	Total Cases	Incisor Segment	Buccal Segment	Total Cases	Incisor Segment	Total Cases
Davies Z-plasty	23%	100%	30	66%	93%	29		
Millard rotation- advancement method,	50%	96%	24	92%	100%	13		
Le Mesurier quadrilateral method.	27%	87%	15	67%	100%	12		

UNILATERAL ANTERIOR PALATE REPAIR

Management of the alveolar cleft has been a point of contention over the years.

Broca and von Langenbeck (1904) and Lexer (1904) freshened the bony edges to encourage union between the alveolar arches.

Lane (1908) utilized soft tissue flaps from the alveolar ridges and palate to bridge the alveolar defect.

Brophy (1923) forcibly wired the alveolar arches. This procedure resulted in midfacial deformity, malocclusion and relative prognathism apparently due to interference with tooth bud growth. Veau (1922) advocated closure of the lip, alveolus and anterior palate in one stage using nasal mucosal flaps.

However, others believe that the anterior palate should not be closed at the same time as the lip repair. (Steffensen, 1949; Tennison, 1952; Slaughter and Pruzansky, 1954 and Millard, 1957.) Bernstein (1972) and Harding and Mazaheri (1972) feared that if the alveolar cleft was closed early growth interference of the vomer-premaxillary suture may account for maxillary growth retardation.

The Veau (1938) closure involved a septal flap sutured across to the nasal mucosa on the far side of the palatal plate. A flap of buccal mucoperiosteum can be used to reinforce the suture line of the nasal mucosa when the hard palate is closed at the time of lip operation, but cannot be made to cover the region immediately behind the alveolus. This is represented in Fig. 8.

As early as 1933 Burian started to place a flap of lip mucosa within the alveolar clefts as a covering for the oral aspect of the reconstructed floor of the nostril. This is represented in Fig. 9. The aim at the time was to prevent perforations in the anterior part

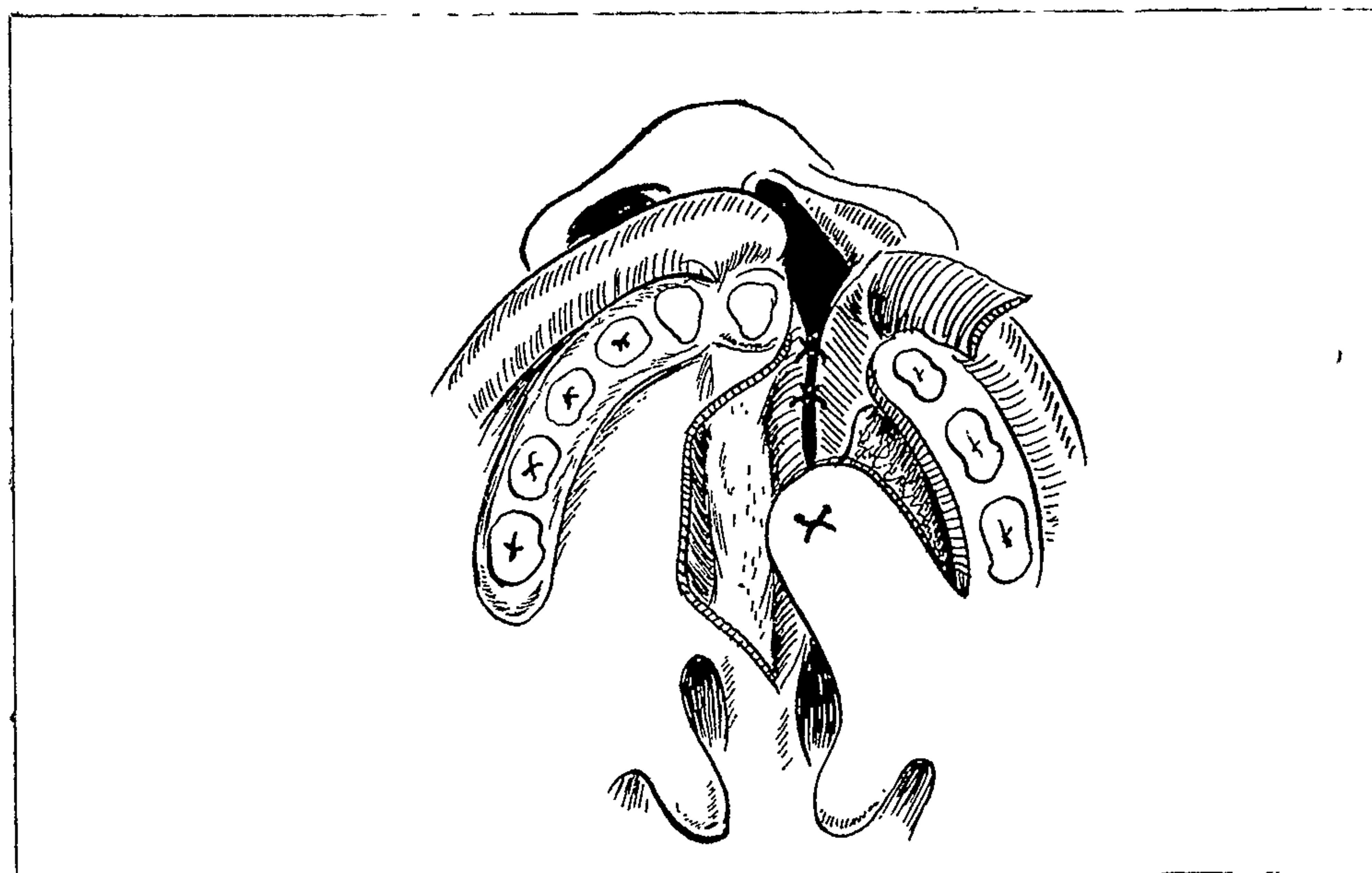


Fig. 8. The Veau Operation

A flap of buccal mucoperiosteum can be used to reinforce the suture line of the nasal mucosa when the hard palate is closed at the time of the lip operation, but cannot be made to cover the region immediately behind the alveolus. (Holdsworth, 1970.)



Fig. 9. Closure of the anterior palate during the primary operation in complete unilateral cleft lip and palate as described by Burian (1933.) The placement of a flap of lip musoca within the alveolar cleft acts as a lining for the oral aspect of the reconstructed floor of the nostril. Closure of the hard palate was by means of a palatal flap. (Burian, 1955.)

of the palate which occurred frequently in the Veau operation. Burian later dropped this interposition method after discussion with Veau who considered the gap in the alveolar process as an imperfection much worse than the nasobuccal communication.

However, he followed up these patients and observed that with the interposition method there was less deformation than in those operated on without interposition, except for the premolar segment of the jaw. This premolar constriction was due to scarring in the foremost part of palate where a defect resulted after shifting the Veau palatal flap.

Campbell (1926) described a procedure using a lateral cleft flap and a septal vomer flap. This technique has been combined with bone grafting. To avoid the effect of scarring, Burian omitted the formation of palatal flap and inserted the vomerine flap as advised by Campbell. This is represented in Fig. 10.

Georgiade (1974) also found it advantageous to create a separate nasal floor and palatal roof during the primary repair of lip alveolus and anterior palatal area. A maxillary flap is brought across the cleft and sutured to the denuded septum establishing a nasal floor. The nasal septal mucosa flap is turned down and sutured to the lateral maxillary shelf, establishing a palatal roof and closing the defect. This method has been also described by Wassmund (1939), Schmid (1955) and Widmaier (1959.) This double-layer closure of floor of nose and of the alveolar cleft is suited to cover a bone graft. The method used depends on the width of the cleft and shape of the alveolar stump. The nasal floor is formed using the Veau-Axhausen method (Veau, 1931 and Axhausen, 1952) and the formation of the oral flap can be either:-

- (i) The vestibular mucosa. (Trauner, 1959.)
 - (ii) The mucoperiosteum of a premaxilla with an alveolar stump protruding laterally. (Pfeifer, 1962.),
- or

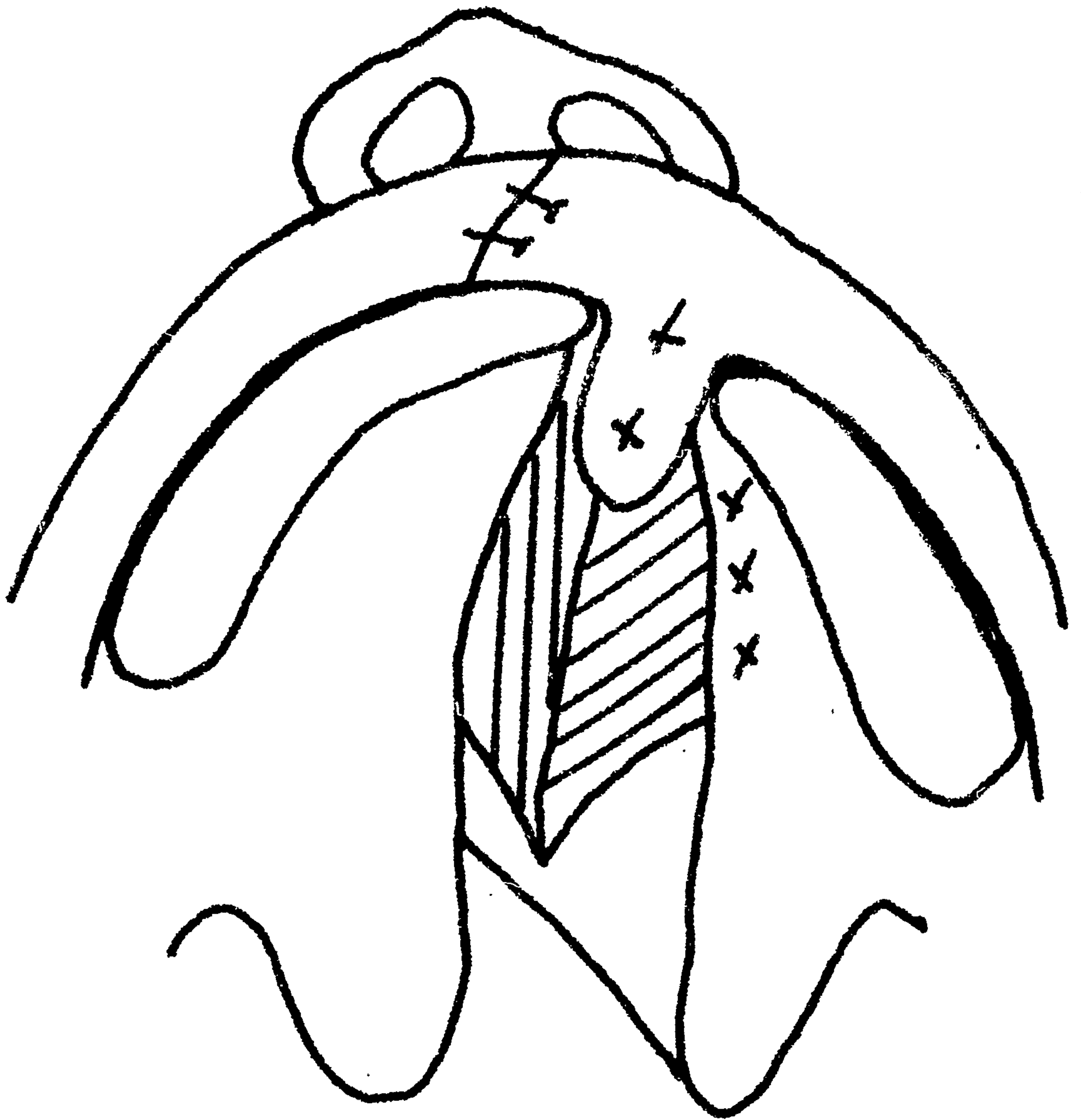


Fig. 10. The interposition-operation using a vomerine flap as advised by Campbell (1926) for anterior palatal closure. (Burian, 1955.)

- (iii) The muco-perichondrium of the nasal septum in wide clefts. (Stellmach, 1959.)

In 1967 Skoog described the boneless bone grafting technique which involves elevating local mucoperiosteal flaps for closure of the alveolar cleft.

Muir (1966) and Battle and Whitfield (1970) noted that with the standard method of repair, the cleft in the alveolar gap is usually closed by a single nasal layer, leaving on the buccal aspect a raw surface which is often 1 cm wide. It is left to granulate and finally to heal by secondary epithelization which results in scar tissue formation and ultimately its contraction and the in-drawing of the lesser segment. A lining should always be added whether it be a Burian flap (1955), Muir's soft tissue flap (1966), Stellmach's flap (1959) or Dey's triangular flap (1974).

It was considered that the flap from the upper labial sulcus that Burian used should not be sacrificed during primary lip closure. Muir (1966) observed that there is a source of tissue which appeared not to have been tapped, namely, mucosa of the free edge of the lip which is usually discarded at operation. This tissue can be retained and used as a soft tissue flap to provide a buccal layer for the repair of the alveolar cleft. This is represented in Fig. 11.

Similar to Muir's method, Dey (1974) has added to his primary operation a bulky soft tissue from the inner surface of the lip which is carried through the alveolar gap. However, his flap differs in detail, size and proportion. The flap consists of muscle tissue and red lining which is mobilized from its attachment to the alar cartilage at its base. This is represented in Figs 12 (a-h).

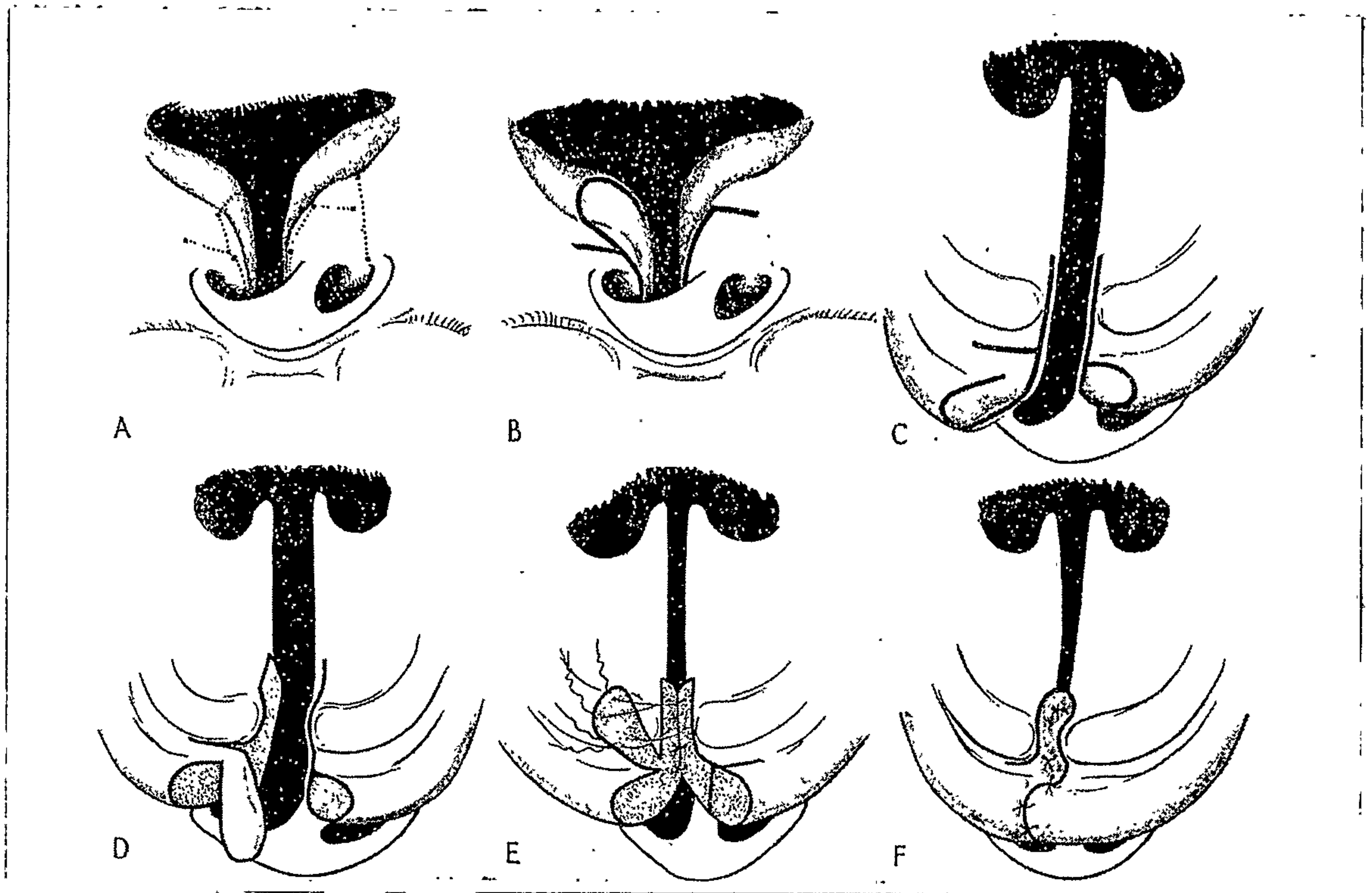


Fig. 11. Muir's Soft Tissue Flap Technique.

- A. A left cleft lip and palate showing the markings for a triangular flap type of repair.
- B. The incisions - the tissue of the free edge of the lip on the lateral (left) side of the cleft will be used for the flap to repair the alveolar cleft.
- C. View of the posterior aspect of the lip to show the design of the flap.
- D. The flap has been raised on a narrow base.
- E. The nasal layer has been sutured and two "A" stitches have been passed through the flap.
- F. Shows the flap in position at the completion of the operation.

(Muir, 1966.)

The original object of this manoeuvre was to allow the vertically disposed muscle fibers in the cleft margin to resume a more horizontal line as part of the orbicularis oris muscle and to mobilize the nostril fully. It was found that a triangular corner falls naturally into the alveolar cleft and is easily fixed there without tension, covering the nasal floor. The flap reinforces closure of nasal floor, prevents narrowing of alveolar gap and allows the incisors in particular to maintain their proper relationship with the mandibular incisors.



Fig. 12. (a) This illustrates the preoperative condition of a six weeks old child with a complete unilateral cleft at the time of the primary operation. It shows the usual appearance of mucosal lip lining.



Fig. 12. (b) The origin of the triangular labial flap.



Fig. 12 (c). The mobilization of the labial flap from the alar base with the orbicularis oris muscle.



Fig. 12 (d). Intercartilaginous alar incision to mobilize the nostril.



Fig. 12 (e). Advancement of the alar cartilage.



Fig. 12 (f). Z-plasty to secure the advanced position of the alar cartilage and to turn the alar base inwards.



Fig. 12 (g). The interposition of the triangular labial flap to reinforce the nasal floor in the interalveolar area.



Fig. 12 (h).

This photograph was taken at 3 years of age, 2½ years after the lip and anterior palate repair. It shows the interposition of the triangular labial flap and its effect. The alveolar gap has not closed perhaps due to the buttressing effect of the flap. The general shape of the arch is good.

UNILATERAL NASAL REPAIR

Congenital clefts of the primary palate are associated with a characteristic malformation of the nose. Whereas the cleft of the lip and maxilla is primary in origin, the nasal deformity may be regarded as a result of abnormal developmental forces generated by the discontinuing of the nostril circumference. (Skoog, 1969.)

To date most improvements in the nose are actually related to better lip surgery. Usually the nose, lip and palate are interrelated problems which cannot be compartmentalized. (Berkeley, 1971.) Various manoeuvres in lip repair may enhance or detract from nasal reconstruction.

Stenström and Oberg (1961) considered that nasal reconstruction must be based upon:-

- (i) an anatomical restoration of the oral sphincter,
- (ii) the rotation of the deformed lower lateral cartilage into anatomic balance with its brother cartilage,
- (iii) the reconstruction of the floor of the nose with nasal mucous membrane,
- (iv) the addition of bone on the face of the maxilla to support the deformed alar and thus offset the hypoplastic quality of maxillary component, and
- (v) the repositioning of the lower end of the septum into a midline position.

Hagerty, Hill, Mendelson, Karesh, Lifschitz and Swindler (1964) emphasized that:

".....that retrodisplacement of the alar attachment and inadequate support of the nostril floor on the cleft side are features which characteristically and necessarily accompany alveolar arch collapse."

Huffman and Lierle (1949) analyzed the anatomy of the cleft lip nasal deformity and noted moderate hypogenesis of the lower lateral cartilage and of the underlying maxilla on the cleft side. The entire lower lateral cartilage on the cleft side is rotated anteriorly and positioned below that of the normal side. This produces a flatness of the nose on the cleft side and there is a bow string contracture on the interior of the cleft nostril which inhibits elevation and rotation of the alar.

The question arises whether one is justified in doing any surgery to the nasal tip at the time of the primary lip repair. It has been noted that if any scar is left in this area it is likely to distort the very thin underlying cartilage. However, it has been argued that unless these cartilages are returned to a normal position inherent defects are bound to occur. (Wynn, 1972; Randall, 1974 b and McComb, 1975.)

Trauner (1955) observed the tendency of the cartilage to resume the same position as before on account of its elasticity. He corrects the deformity during the first operation by transposing, at the entrance of the nose, small flaps of skin obtained from the upper part of the lip (Z-plastic) in combination with the operation by LeMesurier.

CLEFT PALATE SURGERY

The two classic aims of a cleft palate operation are to obtain anatomic closure between the mouth and nose and to produce normal speech. Increasing sophistication has given us a third aim to prevent maxillary or buccal segment collapse and dental alveolar deformities.

Dieffenback (1826) was the first to suggest the separation of the soft parts of the palate from the underlying bone before suturing the edges of the cleft. He recognised the value of releasing incisions in the soft palate. The value of elevating the palatal periosteum with the mucosa when developing the palatal flaps was not recognized until 1861, when suggested by Von Langenbeck. This method has shortcomings. The dead space between the mucoperiosteal flaps and the nasal cavity with an exposed raw surface resulted in scarring and contracture of the palate and velopharyngeal incompetence was noted in a significant percentage of these patients. However, with variations the principle of this operation is still being used today.

Fergusson (1845) described the function of the palatal muscles and recognised the value of myotomy. It is the action of tensor veli palatini muscles during swallowing immediately prior to speech that pulls the flaps, created at operation, away from the midline.

Billroth (1889) fractured the pterygoid hamulus around which the tensor courses, converting it thereby into a slack muscle incapable of producing post-operative tension.

The effect of palatal surgery and specifically hamulectomy on ventilation of the middle ear has been of recent concern. Bluestone, Paradise, Beery and Wittel (1972) noted that after palatal repair ventilation, protection from unwanted secretions and drainage of middle ear secretions were improved. No differences between the ear on the side of the hamulectomy compared to the other ear were found by Noone, Randall, Stool, Hamilton and Winchester (1973).

Thompson and Harwood-Nash (1972) observed roentgenographically that the infractured hamulus returned to its preoperative position.

Originally, to accomplish complete relief of tension at the suture line, releasing incisions were extended well behind the last molars. Grabb, Rosenstein and Bzoch (1971) and many other surgeons advocate extending these incisions from the incisors to the retromolar area. Rayner (1925) recognized the functional disadvantage of this extensive surgical procedure and advised a conservative approach.

Other methods of palatoplasty include the Davies-Colley criss-cross flaps (1890) and the osteal-periosteal flaps of Davis and Brophy (1923).

Limberg (1927), Luoff (1928) and Webster, Quigley, Cuffey, Querze and Russell (1958) advocate closure of only the velum and anterior palate leaving the posterior palate to be moulded and approximated by muscle tension as the child grows.

Several methods of palatal lengthening or push-back procedures have been devised. Von Kuster (1893) lengthened the palate by means of a portion of the detached edge of the cleft. Ganzer (1917) retroposed the entire palate by applying a V-shaped incision behind the incisors. This produced a longer soft palate than that obtained with the von Langenbeck operation.

Gillies and Fry (1921) advocate closure of the soft palate at the expense of the hard palate, the resulting deficiency in the latter being closed by means of an obturator.

Dorrance and Bransfield (1946) pushed the entire palate toward the posterior pharynx in stages with split-thickness grafts applied on the superior raw surface.

Brown (1940) advocated osteotomy of the palatine foramen.

Veau (1932) included an important modification to the von Langenbeck operation. He criticized this method and stressed the need for:-

- (i) closure of nasal layers separately,
- (ii) fracture of the hamular process,
- (iii) suture of the muscles of the soft palate,
- (iv) palatal repair in stages following primary lip and vomer flap closure, and
- (v) creation of palatal flaps based on a vascular pedicle.

He used lateral incisions meeting in the form of a "V" behind the incisor teeth and the resultant palatal flaps were displaced medially and backward.

Kilner (1937) and Wardill (1937) described a technique of palatal repair that ultimately has come to be known as the V-Y retro-position operation which is more radical than that of the Veau technique. Wardill suggested that by dividing the flap obliquely the closure might be easier and the palate longer. This technique could be applicable to all types of clefts. Wardill used a three-flap operation for shorter clefts and a four-flap operation if the cleft involved more than half the hard palate so as not to endanger the blood supply. The three-flap operation is represented in Fig. 13.

Wardill and Kilner both adopted the Veau technique for the anterior repair which resulted in the Veau-Wardill-Kilner operation.

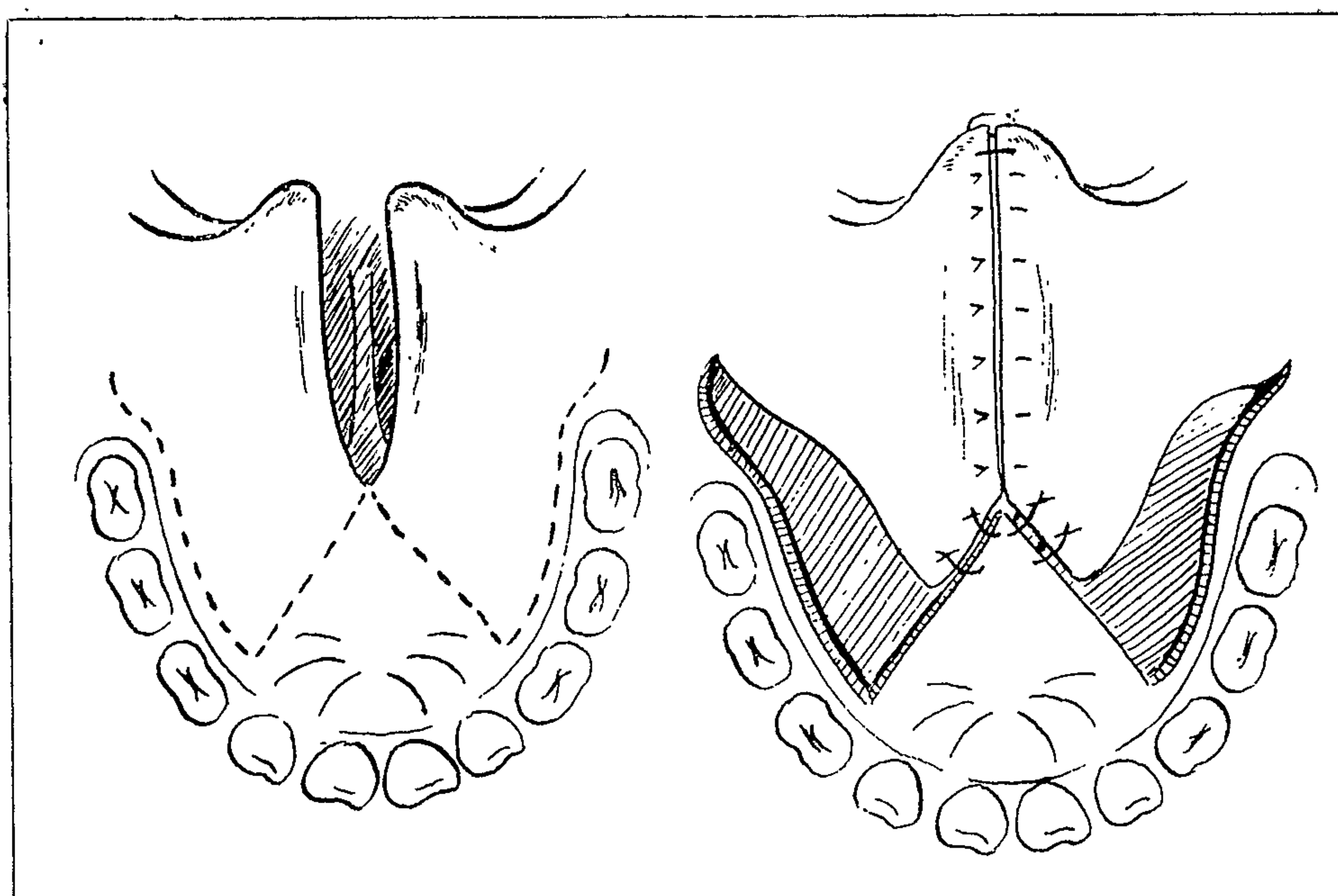


Fig. 13, The three-flap operation (Wardill)
for the shorter cleft palate.
(Holdsworth, 1970.)

Von Langenbeck's simple closure palatoplasty method (cited by Lindsay, 1974) is based on the hypothesis that scarring, secondary to surgical interference, is one of the factors responsible for the production of maxillary deformities. This procedure leaves less raw area over the palatine bone compared to the more radical push-back procedure leaving the anterior mucoperiosteum intact in the incisor area. This small modification produces quite a difference in the maxillary buccal segment and dental alveolar deformities. Lindsay (1974), comparing the two surgical techniques, observed that the frequency of incisor crossbite was much lower, but that the incidence of fistula or residual defect was only slightly higher than with the von Langenbeck operation. The simple closure (von Langenbeck) palatoplasty does nothing to improve speech results or anatomic closure, but it does decrease maxillary deformities.

The von Langenbeck (simple closure) palatoplasty procedure for incomplete cleft of the secondary palate is as follows:-

- (i) free margins of the cleft are incised,
- (ii) soft palate lateral incisions made,
- (iii) the pterygoid hamulus is fractured,
- (iv) lateral release incisions are made in hard palate up to the cuspid-bicuspid region,
- (v) the mucoperiosteal flap is elevated and posterior palatine artery preserved,
- (vi) the nasal mucous membrane is elevated and transected to allow as much of it as possible to go back with the soft palate, and
- (vii) the nasal mucosa musculature and oral mucosa are closed separately.

Blocksma, Leuz and Beernink (1975) after a long-term observation of 10 years claimed that the current techniques such as the Dorrance pushback, Wardill V-Y pushback, Millard Island flap and typical von Langenbeck operations were all to some extent implicated the gradual development of facial deformity, especially if performed within the first two years of life.

They introduced a conservative approach to the repair of complete clefts where the lip was closed at 3-4 months and soft palate closed at 18-24 months using the modified von Langenbeck technique. The hard palate closure was deferred until the age of 5 years when most of the palatal growth had been achieved. Closure involved a vomer flap.

The modified von Langenbeck operation involves:-

- (i) relaxing the bilateral incisions,
- (ii) division of the hamulus,
- (iii) exposure and division of the posterior palatine aponeurosis, without interrupting the posterior palatine vessels, and
- (iv) three-layer closure of nasal, muscle and mucosa.

Blocksma, Leuz and Møllerstig (1975) compared 309 children with the pushback operation and noted no deficiency of the middle third of face, but 49% required a pharyngeal flap. They concluded that the combination of a conservative closure of the palate combined with a pharyngeal flap or teflon posterior pharyngeal implant to correct nasal emission, where indicated, will eventuate in the highest percentage of normal speech and freedom from orofacial deformity.

Browne (1932) performed a two-stage modification of the von Langenbeck operation. Many surgeons agree that the posterior palatine artery if left intact tends to anchor the flaps and prevent the freedom and absence of tension which is so essential in the production of a mobile soft palate. Browne first incised the posterior palatal artery and then after three months performed the von Langenbeck operation with long lateral incisions from the canine area to the tonsillar region.

Nowadays, controversy has raged concerning the relative superiority of simple closure of soft palate versus the V-Y pushback technique. Recent evidence continues to be somewhat in favour of the V-Y approach (Krause, Thorp and Morris, 1976) and that the V-Y cases required fewer secondary procedures. (Musgrave, McWilliams and Mathews, 1975.)

Ruding (1964) reviewed the anatomy and physiology of the palatal muscles and advocated complete elevation and medial suture, approximation of the levator muscles combined with palatal foramen osteotomy and release of the musculature from the hamulus.

Dunn (1952) after 20 years of observation felt it was essential to discard any technique which undermined to any extent the soft tissue covering the palate or which incised the nasal aponeurosis. He employed a vomer flap to close the hard palate and a simple closure of the soft palate.

Stenström and Thilander (1974) modified Dunn's attempt to avoid interference with maxillary growth by using vomer flaps covered with full-thickness grafts to reduce flap contraction. The graft was taken from the groin. An incision along the vomer edge allowed the skin graft to be buried with the raw surface facing the raw area of the flap. Twelve days later the vomer flap with the skin graft covering were sutured to the cleft edge. The soft palate was repaired later. In ten patients followed for ten years, they found almost no constriction of the maxilla in the transverse direction

with only crossbite in the unilateral complete cleft lip and palate cases where tomograms showed bone bridging. They have since modified the technique using split thickness grafts and soft palate closure, all performed in one operation.

Friede and Johanson (1977) observed maxillary retrognathism with their three stage soft tissue closure including vomer flaps. The most common type of vomer flap is based cranially according to the method of Pichler (1934) where the vomer flap is sutured to the maxillary lateral segment with the raw tissue facing orally left for secondary epithelization. They used this method to close the hard palate. In order to reduce restricted midfacial growth, they have excluded the vomer flap and since 1975 are delaying hard palate closure until the patient has reached the stage of mixed or, preferably, permanent dentition.

Posteriorly based vomerine flaps were used by Horton, Irish, Adamson and Mladick (1973) to cover the raw area on the nasal surface in order to avoid shrinkage in palatal pushback flaps.

Tongue flaps have been used for primary closure of the alveolus and the anterior hard palate and for secondary closure of nasal palatine fistulae with good results by Guerrero-Santos and Fernandez (1973.)

In 1944 Schweckendiek (1951 and 1955) started to perform primary veloplastic operations with the objective of creating a soft palate which could function normally. Slaughter and Pruzansky (1954) followed the same idea. They closed the soft palate at 14-16 months of age. At five years, the hard palate was closed by suturing the nasal layer and raising a unilateral palatal flap on the cleft side and placing it to the rim on the uncleft side.

Nowadays, Schweckendiek Jnr. (1978) performed primary veloplastic operation between 6-8 months and, three weeks later, lip repair and reconstruction of the floor of the nose. During the primary operation he mobilized the soft parts at the posterior edge of the hard palate by means of lateral incisions in order to bring together the muscle of the palate along the midline without tension. He also endeavoured at this stage to achieve an elongated palate by the inclusion of pharyngopalatine arch. The hard palate was closed at 12-14 years when normal growth was virtually completed.

Perko (1974) describes a new method of closure of isolated cleft palate. Basically, it is derived from the Widmaier technique (1959) for the closure of the soft palate. However, it utilizes mucosal palatal flaps without denuding bone for closure of the hard palate. This is represented in Fig. 14. This method represents an attempt to prevent a growth impairment in the palatal shelves. He utilizes two-stage closure in complete clefts whereby the soft palate is closed at 18 months. In this two-stage closure he used a modification of the Widmaier technique (1959) combined with the transposition of the levator palate muscle (Ruding 1964, Kriens, 1967 and Braithwaite, 1968) and with the lengthening of the nasal mucosa using a Z-plasty in the dorsal part of the soft palate. The hard palate can be closed between the ages of about 5-8 years, depending upon the occlusion of the teeth and the stage of eruption of the remaining teeth. It will take a few years to determine the result of this technique.

Secondary surgery of lip and palate is not relevant to this study and is omitted from this review.

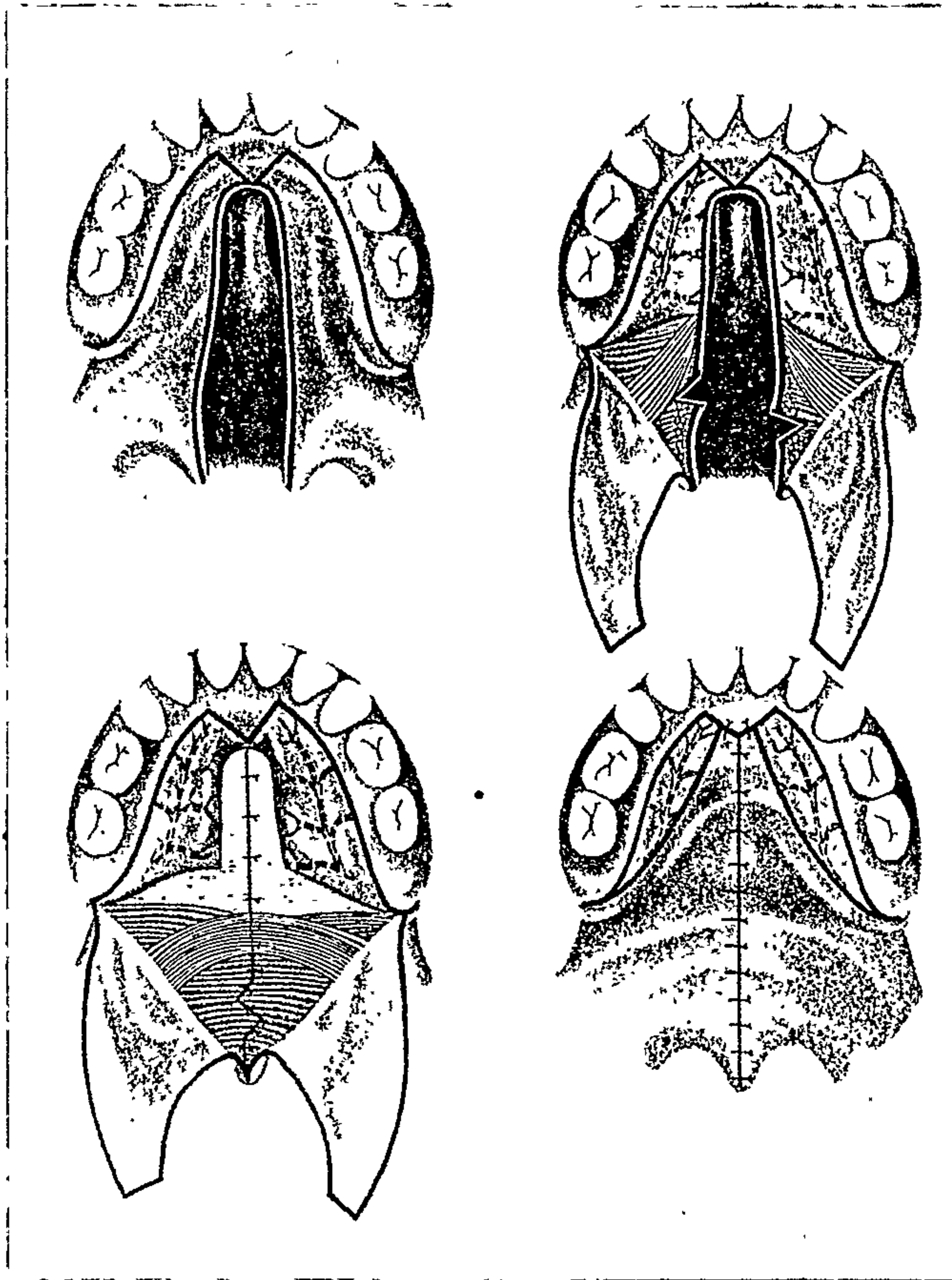


Fig. 14.

Drawing of palatal mucosal flap technique for closure of isolated cleft palate without mobilizing the periosteum. (Perko, 1974.)

SECTION 3

MATERIALS AND METHODS

The aim of the investigation was to compare a variation in the surgical procedure involved in the primary repair of the anterior palate by incorporating a triangular flap of buccal mucosa, in complete unilateral cleft lip and palate patient to a standard method of repair. The results of the treatment were evaluated by assessing the occlusion.

Selection of Subjects

The patients were selected from the public and private cases of the surgeon and cases who had been treated in the Royal Alexandra Hospital for Children at Camperdown, Sydney (R.A.H.C.). Public patients were obtained through the medical records section of R.A.H.C. where as the private patients were taken from the personal records of the surgeon.

Cases with a fibrous band bridging the cleft at the base of the nose (Simonart's band) were not excluded, as they showed significant deformity. None of the patients had undergone orthodontic treatment before or after surgery.

All the children were born between 1962 and early 1977. The average age of occlusal assessment of casts was between $2\frac{1}{2}$ to 7 years of age with full eruption of the deciduous dentition.

Method of Recording

To examine the more recent group, suitable appointments were made at the University of Sydney Dental School. Parents and patients were interviewed to explain the investigation and a short history of each patient was taken. Records comprised profile, full-face and intra-oral photographs of the children and an alginate impression of the upper and lower dental arches with a wax bite record taken in centric occlusion.

Grouping

There were 52 patients who were divided into two groups according to the surgical procedure utilized in the repair of the primary palate.

(a) The Standard (Control) Group

This included 25 patients treated by the standard surgery between 1962 and 1970. This involved a primary repair using the Tennison procedure for the lip and a medial advancement of the alar cartilage with Z-plasty and closure of the anterior palate with septal flap tucked into the cleft margin. The second repair involved closure of the residual posterior cleft of the hard and soft palate including palatal lengthening using the Wardill V-Y procedure. This group served as experimental control.

(b) Triangular Flap Group

This included 27 patients treated by the triangular flap technique between 1972 and 1977. The plastic surgeon (Dey, 1974) introduced a variation of the primary repair by incorporating a triangular flap of buccal mucosa into the alveolar cleft. The second repair of the posterior palate was the same as for the "Standard Group".

The surgical procedures were carried out at approximately 2 months of age for the lip and anterior palate repair and at 12 months for the palate repair.

All the patients were treated by one plastic surgeon and with only two-stage repairs and no other surgery.

The distribution according to sex and affected side in the different groups is shown in Table 6.

Table 6. Sex and cleft side distribution.

Group	Males Cleft Side		Females Cleft Side		Total
	Right	Left	Right	Left	
Standard Group	4	9	5	7	25
Triangular Flap Group	6	12	5	4	27

Analysis of Occlusion

The 52 sets of models from the deciduous dentition were examined and assessed for crossbites according to the numerical classification of Huddart and Bodenham (1972).

SECTION 4

RESULTS

Numerical classification of occlusion

Segment scores and total occlusal scores with their standard deviation and range values are shown in Table 7.

(i) Cleft side buccal segment.

The standard control group score of -5.44 is higher than that of the triangular flap group, -4.54.

(ii) Non-cleft buccal segment.

The standard (control) group score of -0.96 is slightly higher than that of the triangular flap group, -0.81.

(iii) Incisor segment.

The standard (control) group score of -1.32 is higher compared to the triangular flap group, -0.78.

(iv) Total occlusal score.

Again the score of -7.72 in the standard (control) group is higher as compared to -6.04 for the triangular flap group.

Statistical Analysis

A comparison between the triangular flap and standard (control) group was made using the t-test. In general, the differences in results between the different surgical procedures was not significant. The level of significance used was 1% and 5%. In both groups, means and

distributions were approximately equal. The following interpretation of the differences observed should therefore only be regarded as comments on tendencies in the material rather than on statistically significant differences.

Table 7. Comparison of primary dentition crossbites following standard and modified (triangular flap) procedures for surgical closure by one surgeon of complete unilateral clefts of lip and palate.

	Incisor Segment Score	Cleft Segment Score	Non-Cleft Segment Score	Total Occlusal Score
<u>Standard Control Group.</u>				
Number	25	25	25	25
Mean	-1.32	-5.44	-0.96	-7.72
Standard Deviation	± 2.21	± 2.33	± 1.72	± 4.61
Range	2 to -6	0 to -8	0 to -5	0 to -19
<u>Triangular Flap Group.</u>				
Number	27	26	26	26
Mean	-0.78	-4.54	-0.81	-6.04
Standard Deviation	± 1.70	± 2.39	± 1.77	± 4.05
Range	2 to -6	0 to -8	0 to -6	0 to -16
<u>Comparison of Means.</u>				
"t" value	0.98	1.37	0.31	1.39
Significance (P = 0.01)	n.s.	n.s.	n.s.	n.s.

SECTION 5

DISCUSSION

In the surgical management of maxillofacial clefts, uncertain patterns of growth and development and questionable long-term results must be taken into consideration. The lack of knowledge concerning growth and development of the cleft tissues before and after surgical intervention leads to a confusing picture with regard to the proper management of these cases. (Bernstein, 1972.)

When the known effects of operation have been excluded, there remain a group of cases in which the final state suggests that a basic lack of growth potential exists concomitantly with the cleft deformity. This has been supported by Innis (1962) who examined a number of completely unrepaired clefts showing defects which in repaired cleft cases would undoubtedly have been attributed to the effects of surgery. On the other hand, examination of a rather large similar series of patients with unrepaired or partially repaired clefts in Puerto-Rica (Mestre, Dejesus and Subtelny, 1960) suggested that the growth of the maxilla and mandible were within normal limits, so this problem remains unsettled.

Dealing with cases of major clefts, Dey (1974) has noted:-

- (i) A large variation in the degree of ultimate bony deformity, even with a standard operative approach applied by a single surgeon.
- (ii) The fact that a great deal of bony deformity may exist without any actual palatal cleft.

- (iii) The frequent deterioration of an apparently satisfactory result in childhood during the pubertal growth spurt and the maturation of the face to adult state.

From these considerations, it is evident that there is an inherent or inbuilt potential for bony deformity whether this be determined genetically or by some localized (embryonic mesodermal or vascular) factors. Failure to give weight to these factors has led to much confusion of thought in regard to the nature and timing of operative procedures and in relating these to the outcome of treatment.

Bergland and Sidhu (1974) have noted that the postnatal development and growth pattern of a cleft maxilla depend on a variety of factors, some of which are not yet understood. There has been evidence of an ever-increasing number of recordings of complete unilateral cleft subjects observed at various ages which illustrate a nearly uniform picture of a favourable maxillary and occlusal development. This fact strongly indicates that the main reasons for the maxillary underdevelopment, so frequently seen in treated cleft cases, have to be sought in the treatment procedure employed.

Dey (1974) stressed that the surgical procedures that should be avoided are:-

- (i) Interference with alveolar mucoperiosteum at operation during the early years, always leads to local dental deformity.
- (ii) Repeated operations on the hard palate undoubtedly interfere with bony growth.
- (iii) Any secondary procedure necessary for closure of a residual hard palate fistula represents a major failure.

It is clear that many of the deformities seen in the past were due to unnecessarily traumatic or inappropriate surgery.

Present opinion about the effect of operation on the skeleton may be summarized as follows:-

- (i) Repair of the lip is followed by moulding of the protruding premaxilla into a more normal position but has no effect on the growth of the maxilla.
- (ii) Repair of the part of the cleft through the alveolus causes displacement of the lesser segment of the maxilla into an abnormal position.
- (iii) Repair of the defect in the hard palate may lead to diminution of the growth of the maxilla although, fortunately, this is not often severe. (Huddart and Bodenham, 1966.)

Recently, much attention has been paid to deformities of the alveolar arch in cleft children. McNeil and Burston advocated the preoperative correction by orthodontic method of early deformities and, more recently, many surgeons have used bone grafts and periosteoplasty techniques in an attempt to fix the alveolar segments in a good position. Others, however, have recommended against early repair of the alveolar cleft. Bernstein (1972) and Harding and Mazaheri (1972) have observed that early repair of the alveolar cleft results in a very rigid fibrous or even bony union comparable to that of a bone graft. This has not only contributed to a higher incidence of anterior collapse of the posterior palatal segment but also to a larger incidence of posterior crossbite malocclusion. To overcome this problem Battle and Whitfield (1970), Burian (1933), Dey (1974) and Muir (1966) have all advocated incorporating a mucosal flap to serve as a oral lining to the Veau-type nasal floor closure.

The data from the present study is presented in Table 7.

can now be discussed. While there is no statistically significant difference between the respective measures for the two groups, it can be noted that the mean crossbite scores for the Triangular Flap Group are all less than those for the standard treatment group. The variability of the measures as shown in the range values may be related to the range of conditions presenting at primary operation.

Data from other studies can be used for additional comparison. (Table 2, page 5.5.) The age levels of the children vary, being 5 years old in Huddart's and Hellquest and Skoog's group, 8-9 years in Friede and Johanson's group and $2\frac{1}{2}$ - 7 years of age in the present study. Due to the differing age group we cannot make absolute comparisons between the studies in terms of the amount of dental crossbite present. However, from observation of mean values the triangular flap procedure may occupy a middle position in this series. Also when observing the incisor segment figure there is a definite indication of it being on the low side. Therefore we can conclude that this procedure is some way towards achieving the objective of reducing incisor segment collapse and crossbite.

An example of the effects associated with the triangular flap is shown in Fig. 15 and Fig. 16 at the deciduous dentition stage. Note the symmetrical dental arch and even bite with good occlusion.

It may be noted in Fig. 17 that the closure of the lip and anterior palate at 6 weeks initiated an approximation of the maxillary segments apparently as suggested by Bergland and Sidhu (1974), a process that seems to be completed relatively soon after the surgery. At the time of palate repair at 12 months, it was noted that the alveolar gap has not closed and the general shape of the arch is good. The closure of the residual cleft in the hard and soft palate seemed to have very little, if any, narrowing effect on the shape of the maxillary arch. This is illustrated by the fact that the segment relationship, "approximation without contact of alveolar

Table 2. Comparison of crossbite scores for repaired complete unilateral clefts of lip and palate using data from different studies.

Author(s)	Treatment	No.	Incisor Segment Score	Cleft Segment Score	Non-cleft Segment Score	Total Occlusal Score
Huddart and Bodenham (1972)	Standard surgery	34	-2.06	-3.98	-1.08	-7.33
Huddart (1967b)	Presurgical treatment	17	-1.59	-4.29	-1.18	-7.06
	Non-presurgical treatment	7	-3.00	-4.14	-0.86	-8.00
Huddart (1973)	Presurgical treatment	40	-0.6	-3.9	-1.7	-6.2
	Controls	13	-0.5	-3.3	-1.9	-5.7
Friede and Johanson (1977)	Vomer Flap and Bone graft	29	-3.2	-5.3	-2.8	-11.3
	Vomer flap	50	-2.4	-4.7	-1.4	-8.5
Hellquist and Skoog (1976)	One peri-osteoplasty	21	-0.95	-3.85	-0.48	-5.28
	Two peri-osteoplasty	15	-1.00	-4.13	-1.27	-6.40
	Control group	30	-1.07	-3.83	-0.53	-5.43
Present Study (1979)	Standard control group	25	-1.32	-5.44	-0.96	-7.72
	Triangular flap group	27	-0.78	-4.54	-0.81	-6.04

processes", persists up to the age of 4 years and 2 months. This possibly is the result of the tethering and buttressing effect of the triangular flap in the alveolar cleft. The persistence of the segmental relationship prevents any occlusal impairment from occurring in the deciduous dentition. In the cleft area the tissue maintains its soft character and will be able to act as a passive suture allowing the maxillary segments to separate during growth without the restricting effect of a rigid primary bone graft. By contrast, Fig. 18 shows one of the cases from the Standard Group treated with the standard method of repair. This case shows evidence of crossbite of the lesser and anterior segment. This collapse has been explained to be the result of the constrictive effect of the anterior palate closure. Part of this collapse is also due to the tilting of the maxillary segments and perhaps by movement which takes place at the sutures along the line of the base of the skull. (Dey, 1974.)

Improvement in cleft lip and palate rehabilitation is only established after continuous documentation and long-term follow-up of different treatment methods. (Hotz, 1973 and Subtelny, 1974.) It would be of value to observe whether this successful post-operative result in the Triangular Flap Group will continue through the pubertal and post-pubertal growth of the maxilla and mandible. There have been only two studies which have followed the pattern of malocclusion from the deciduous to the mixed dentition stage. (Bergland and Sidhu, 1974 and Ranta, et al, 1974.)

Berland and Sidhu noted considerable impairment of the occlusion in the early mixed dentition stage. They explained this apparent deterioration as not the result of resumed drifting of the maxillary segments but to be confined to the incisors and their adjacent tissue, i.e. the alveolar process. It was concluded that the premaxillary area is highly susceptible to changes in teeth relationship and that there is a considerably higher degree of stability in the lateral regions of the

maxillary arch. From observations in the present study, there was an improvement in the incisor segment relationship in the deciduous dentition stage. It is possible from only a slight variation in the surgical procedure, such as adding a triangular flap in the anterior palate repair, that the stability of the premaxilla may increase and hopefully we will not observe this deterioration of the occlusion in the mixed dentition stage. This can only be possibly confirmed by following-up the present study.

Ranta, et al (1974) observed small differences of change in the frequency of anterior and posterior crossbite between children under 5 years of age and 5 - 7 year olds. It seems probable that when crossbite develops this occurs at a very early stage. They considered the effects of scar tissue formed being marked during the period of fast jaw growth, i.e. in the first 4 years of life, allowing the anterior and/or posterior crossbite time to form.

Derichsweiler (1964) is of the opinion that malocclusion is present from the very beginning and considered that the crossbite observed in cleft patients can be seen in the new born prior to the operation and is not due to the consequence of the operation.

As has been noted in Section 2, different observers (see Literature Review : pp 2.44-47 and 2.61-74) have noted that surgery for secondary palate repair may or may not have significant effect in producing maxillary collapse and lateral crossbite. But it may be inferred from this study that the improvement in palatal surgery justifies transfer of attention to the anterior palate repair.

In fact, this study is concerned with its assessment of crossbite following surgical repair of both primary and secondary palates. In so far as the Standard and Triangular Flap Groups received identical treatment for their secondary palate cleft, their differences, particularly with respect to lateral crossbite could be related also to the difference in primary palate repair.

Godfrey (1979) considers from observations that the pattern of malocclusion, even among cleft children, does not change in any major way between 2 years and 15 years of age, unless there has been special additional traumatic

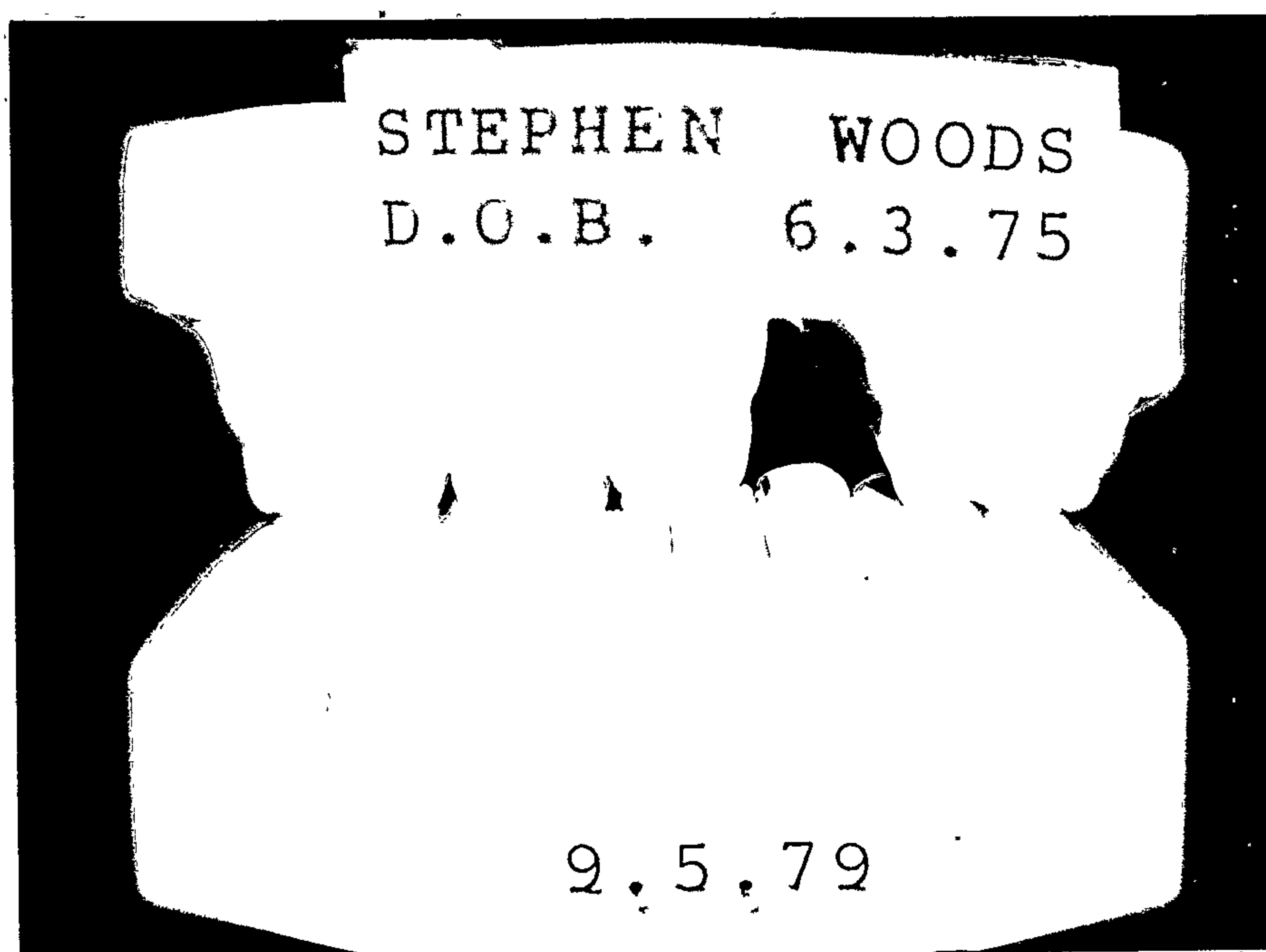


Fig. 15. Articulated study casts of complete deciduous dentition. This is one of the cases from the Triangular Flap group. It shows symmetrical dental arch and an even bite with good occlusion.



Fig. 16. An example of a Triangular Flap case at 3 years of age showing the segment relationship, "approximation without contact of alveolar processes" with good maxillary and mandibular incisor relationship.

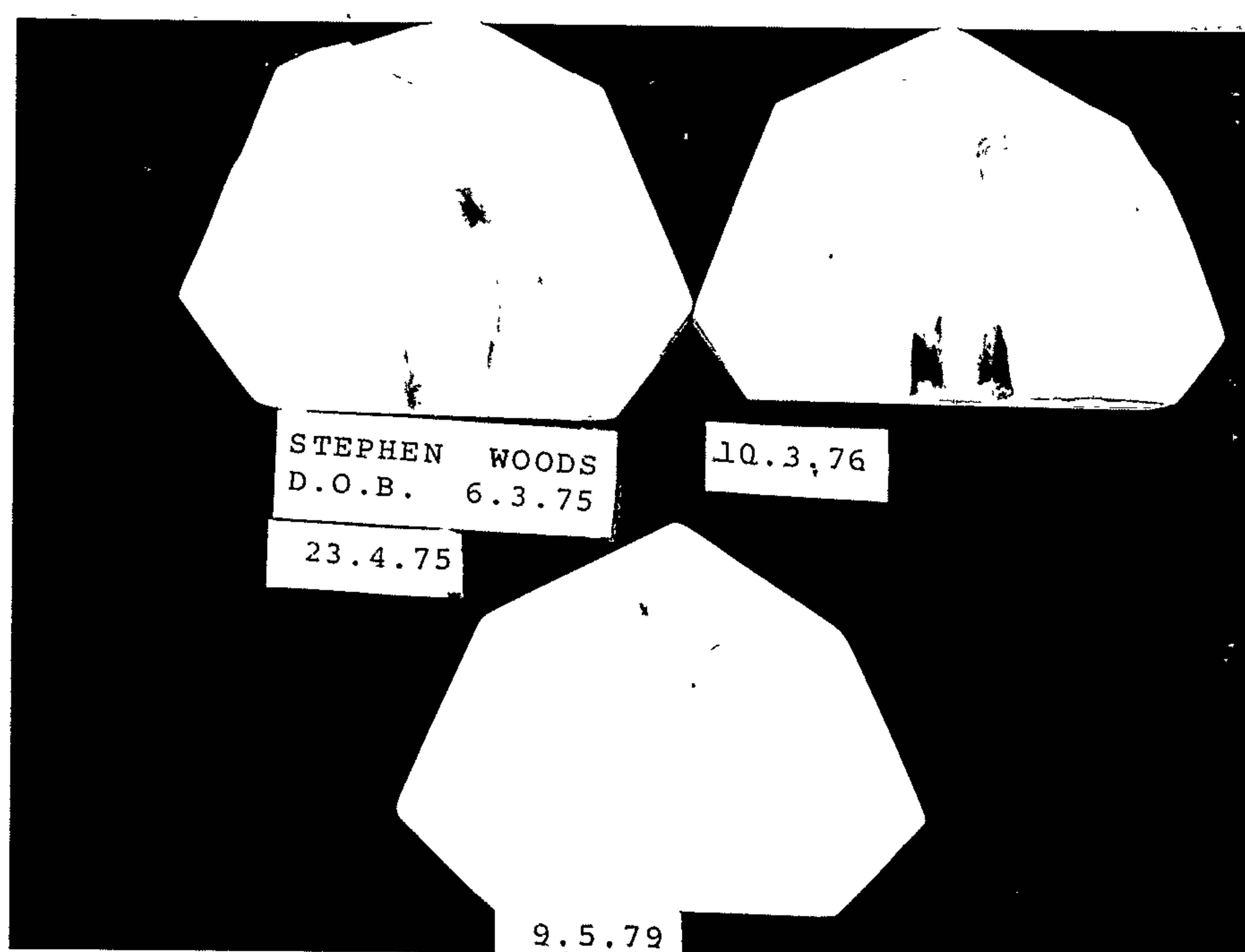


Fig. 17. Series of study casts used in the study.

Upper left, the pre-operative cast of a newborn with complete left unilateral cleft lip and palate.

Upper right, cast taken 12 months following lip and anterior palate repair using the triangular flap technique.

Lower cast, taken 3 years following palatoplasty. The alveolar gap has not closed and the general shape of the dental arch is good.

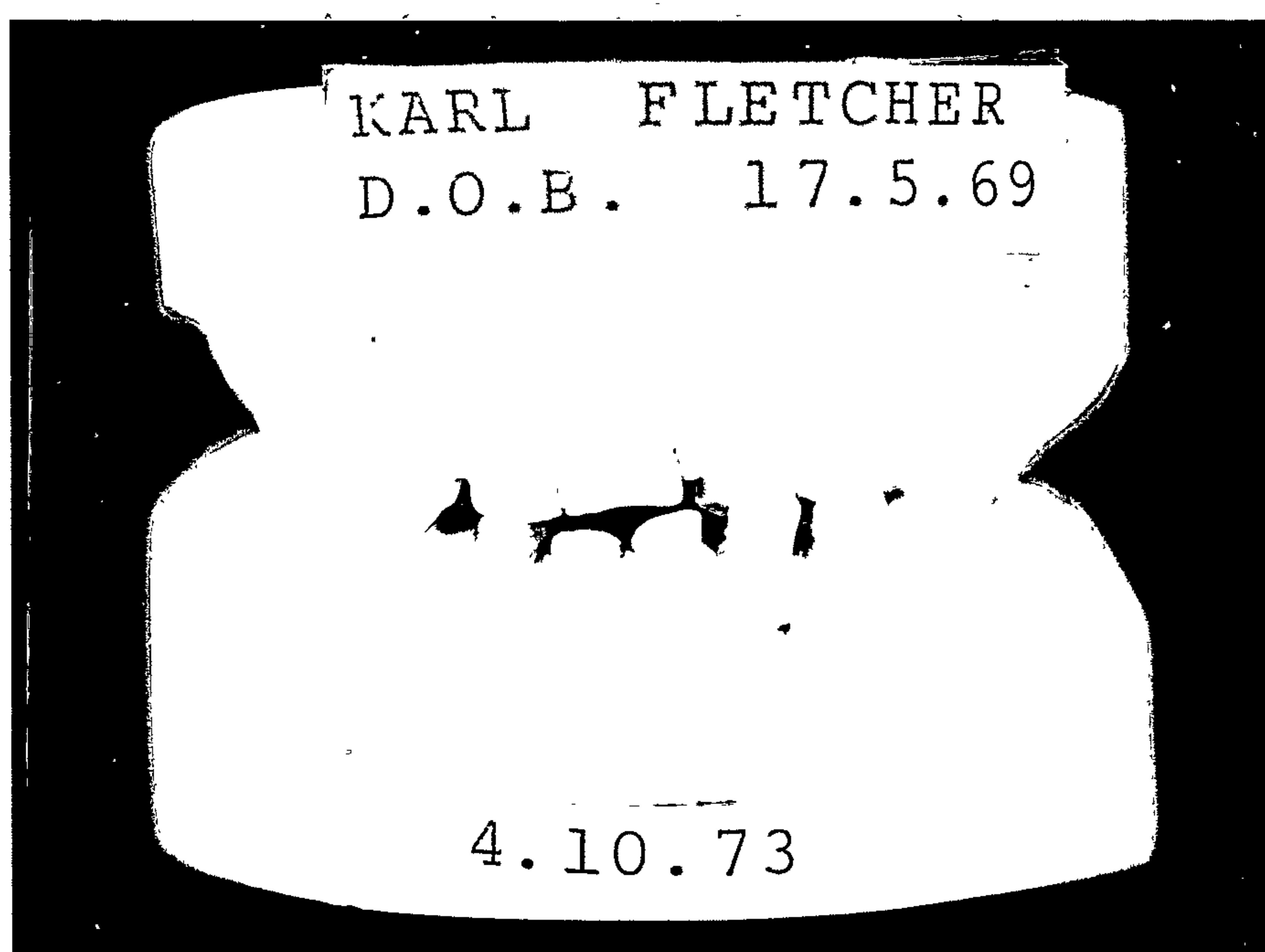


Fig. 18. Articulated study casts of complete deciduous dentition. This is one of the cases from the standard control group showing anterior and buccal crossbite. This collapse is the result of the constrictive effect of a single nasal layer closure of the anterior palate,

interference. He believes that absence of incisor crossbite during the primary dentition stage of development is a good predictor of minimal or absent crossbite when the permanent incisors erupt.

However, the outcome of surgical management cannot be appraised until maturity has been reached. This means a lead-up period of at least 15 years before one can be confident about a greatly improved therapy for repair of cleft lip and cleft palate.

SECTION 6

SUMMARY AND CONCLUSION

This thesis comprises an orthodontic assessment of primary surgery involving a modification of an existing standard method of the alveolar cleft repair.

This involved the investigation and comparison of the occlusal status of two groups of deciduous dentitions in children with unilateral complete clefts for which only soft tissue surgery had been performed. The only variation between the groups was the method used in the repair of the anterior palate. In the Standard (Control) Group closure was by a single nasal layer only, while in "the Triangular Flap group" this standard method was altered by incorporating a triangular flap of buccal mucosa.

The Huddart and Bodenham (1972) scale was used as the means for evaluating, in a quantitative manner, the degree of anterior and buccal crossbite and therefore a measure for the relative "success" of the surgical technique. Other important evaluative parameters related to speech, hearing and appearance were not considered.

This study showed that the mean incisor and buccal crossbite scores for the triangular flap group were all less than the corresponding scores for the standard treatment group, though the differences were not statistically significant. Further, the former group's incisor segment score compared favourably with studies utilizing periosteoplasty, bone grafting, orthopedic appliances and the vomer flap technique. These studies also used the Huddart-Bodenham method of assessment.

While realizing that a thorough statistical approach has failed to prove the absolute success of the triangular flap technique, nevertheless at this stage it can be concluded that the stated surgical objective of reducing incisor segment collapse and crossbite may be achieved. Thus, there is justification in continuing this form of treatment. A subsequent study at maturation is still required to enable a more conclusive appraisal of this method for repair of anterior cleft palate.

SECTION 7

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