I. Basics
1. Pertinent Embryology

There are many missing links, and much is still unknown about the embryology of the human face and head. This deficiency is not surprising with so much happening in such a short period of time. Anatomist Robert Bean calculated:

If the rate of growth of the first month of embryonic life were to be maintained until adulthood, the resultant individual would be $128,350$ to the $1,100$ power light years in length.

The Dicksons clarified the perspective of this figure when they determined that such a rate of growth, if continued, would produce a person who at 4 years would span the galaxy, by 6 years would span the universe and as an adult could hold the universe in his hand like a grain of sand.

Veau

In 1938 Veau proposed—and he was seconded by Streeter in 1951—that masses of mesoderm migrate between two continuous sheets of ectoderm covering the face and roof of the primitive oral cavity. Stark added in 1954 that unless this ectoderm is supported by an intervening layer of mesoderm it will eventually break down and give rise to various degrees of clefting.

The secondary palate posterior to the incisive foramen is formed by fusion of the two palatal processes which are vertical outgrowths of the maxillae. Lying at first vertically at each side of the tongue, these palatal folds ascend as the neck extends and the tongue descends. Then, between seven and eight weeks, if all goes well they fuse with each other and with the inferior border
of the septum from before backwards, to form most of the hard and all of the soft palate. Failure of fusion, of course, produces various clefts of the secondary palate.

STARK

In 1971 at the Melbourne Congress Richard Stark reviewed the embryological development of the face, including the palate. He noted:

The secondary palate develops as the result of the positional change of palatal processes or shelves, then by their growth and adherence and finally merging of their mesoderm.

At the seventh week the head is acutely flexed and turned to the right. The tongue is thus pushed cephalad between the palatal shelves, which hang downward on either side of the tongue like the ears of a hound dog. Slowly, as the head begins to extend the tongue begins to drop, starting first at the base of the tongue posteriorly. The palatal shelves seize this opportunity to overcome the tongue resistance and start to rise, first posteriorly, then forward as a wave until the anterior portion completes the positioning of shelves above the tongue.

Now the shelves grow, meet and, if they are sufficiently adhesive, fuse, first at the anterior one-third of the hard palate, then forward to the incisive foramen and lastly backward to the uvula.

In the Transactions of the Fifth International Congress of Plastic and Reconstructive Surgery Stark postulated:

A number of things can occur which can cause this series of events not to take place, with a resultant cleft palate. (1) Increasing upward resistance of
the tongue, such as in Pierre Robin syndrome where the jaw is small and the
tongue is pushed upward between the palatal shelves. There is no opportu-
nity then for the shelves to rise over the tongue to meet and fuse. (2) The
shelves themselves may be so deficient of mesoderm that they cannot grow
and meet each other. (3) The force to lift the shelves up may not be present,
as is true in animals treated by excessive doses of cortisone, vitamin A, or
X-ray. (4) A broad head as in oxycephaly may prove too wide for normal
shelves to meet. (5) There may be postfusion rupture, as suggested by the
presence of epithelial pearls found along the cleft margin by Kraus. (6) The
head may not extend or stay flexed, in which case the tongue is pushed
upward and the palatal shelves are unable to get into proper position for
fusion. This would be true in Klippel-Feil syndrome. (7) An encephalocele
may hang between the shelves, proving an insurmountable obstacle.

PATTEN

Bradley M. Patten of the University of Michigan noted in *Cleft
Lip and Palate* (edited by Grabb, Rosenstein and Bzoch in 1971):

The palate is contributed to by the nasomedial processes. From their deeper
portions, the small, triangular, median part of the palate is formed. It is to
this portion of the palate that I would restrict the use of the term *primary
palate*. The main part of the palate is derived from that portion of the upper
jaw which arises from the maxillary processes. Shelflike outgrowths of these
processes arise on either side during the seventh week, and grow toward the
midline. These *palatal shelves* form the *secondary palate*. When the palatal
shelves first start to develop, the tongue lies between them, and they are
directed downward so that their margins lie along the floor of the mouth on
either side of the root of the tongue. As development progresses, the
position of the tongue is shifted downward and the margins of the palatal
shelves are free to swing upward and toward the midline. . . . Much more
information is needed concerning this process. . . . On the basis of the best
available age-length data, this places the withdrawal of the tongue from
between the palatal shelves as occurring toward the end of the eighth week,
presumptive fertilization age. . . .

When they first move up, the shelves are not sufficiently developed to
meet each other. Their growth is vigorous, however, and by the eighth
week, they have made contact. Thereafter fusion progresses from the rostral
part toward the uvula. Burdi is convinced that the typical fusion of the
palatal shelves with the characteristic incarnation of epithelial remnants does
not extend to the uvula end of the soft palate. He regards the absence of
epithelial remnants in this territory as indicating that the lengthening of the
soft palate and the formation of the uvula is brought about by merging rather than fusion. At the same time that the palate is thus being formed, the nasal septum grows downward toward it and soon becomes fused to its upper face.

Patten championed the theory that growth of mesenchymal masses beneath the surface brings originally separate structures together in the lip by pushing the epithelium out from between the elevations in the process of merging. Yet in the palate he accepted the fusion theory, except in the uvula.

One of these is familiar in the formation of the palate. For this process the usual term fusion is entirely appropriate. As the two processes come together, the covering epithelial layers are brought into contact. Shortly thereafter, the epithelium that no longer has an external exposure begins to regress. The epithelial changes in this process have been well described by Barry.

POSWILLO

David E. Poswillo has long been interested in the pathogenesis of cleft lip and palate. Upon completion of his oral surgery training in England, he returned to New Zealand in 1953 as senior oral surgeon at the Plastic Surgical Unit, Christchurch, and co-director of the cleft palate clinic. He explained how he got started:

I got into palatal study in New Zealand because I was attempting to find an animal model in which I could do control experiments on the surgical repair of the palate. Ironically, some years after I had been struggling to find a reasonable model of cleft lip and/or palate, my work became well known. As
a result of a newspaper article, a lady who lived only a few hundred yards from my hospital rang me up. All the time I had been working to produce an animal model of cleft lip and palate, she had been trying to breed the same malformation out of her pedigree colony of dachshund dogs. Luckily she had not succeeded and then for a year or two my plastic surgical colleague, John Roy, and I carried out further experiments on the dog model at home at our expense.

Poswillo’s work was recognized by Sir Harold Himsworth, Secretary of the Medical Research Council of Great Britain, during a search visit to New Zealand. His report to the British government stated that the most exciting research he had seen on his one-month visit was being carried out at the bottom of a garden in Christchurch by a young man called David Poswillo.

In 1968 Poswillo devoted his first Hunterian Lecture to the isolated cleft palate and particularly the form that is associated with postural molding, entitling the lecture “The Aetiology and Surgery of Cleft Palate with Micrognathia.” Poswillo eventually became professor of teratology in the Royal College of Surgeons of England and consultant oral surgeon at Queen Victoria Hospital, East Grinstead.

In the May 1974 Proceedings of the Royal Society of Medicine he wrote:

For thousands of years mankind has been intrigued by disturbances in the physical development of the human body. Even before descriptions of such deformities were recorded on the clay tablets of Babylon, in 3000 B.C., they were illustrated in the rock drawings of primitive cave dwellers. But it was not until the time of Harvey, in the seventeenth century, that the scientific study of malformation began. After the development of the cell theory the significance of the foldings and invaginations of the three germ layers came to be understood, and it was possible to comprehend many anomalies of development in terms of mechanical difficulties. For example, cleft lip and palate became recognized as a failure of fusion of the maxillary processes.

Clefts of the posterior palate may be classified into two principal groups. In one there are those clefts, both unilateral and bilateral, which accompany cleft lip. In the other are the solitary clefts of the secondary palate. Clinically these two groups may easily be distinguished. Most authors agree that these two groups are distinct entities. The difference in incidence, sex predisposi-
tion and prevalence of associated anomalies all support this division into cleft lip and isolated cleft palate.

The frequent association of cleft palate with the cleft lip anomaly has been investigated by many workers. As has already been described, animal embryos susceptible to cleft lip have a large median nasal process. Trasler & Fraser (1963) have shown that in such circumstances, at the commencement of palatal shelf closure, the tongue does not move forward between the lips as is usually the case. Instead the tip of the tongue remains pressed against the median process and arches up into the nasal cavity between the palatal shelves. Thus movement of the shelf or shelves towards the midline is impeded. Therefore in an embryo with cleft lip it is likely that cleft palate results because movement of the shelves from vertical to horizontal is delayed by the intervening tongue. If eventually the shelves do become horizontal it is unlikely that they will meet each other or the nasal septum; thus fusion fails to take place.

The prevalence figures for isolated cleft palate are lower than those for cleft lip and palate, but the ratio of racial incidence is much the same. In Caucasians it occurs once in 3000 live births. An excess of females over males in the ratio of 60:40 exists in isolated cleft palate. Associated congenital anomalies occur twice as often with isolated cleft palate as with combined lip and palate clefts. Micrognathia has a very high association with isolated cleft palate due in part to the simultaneous occurrence of the two anomalies in the Pierre Robin syndrome.

Normal palate fusion involves synchronized interaction between growth and convergence of the palatal processes, tongue withdrawal and muscular activity, mandibular growth, changes in cranial base and cranial flexure, and steady increases in the width of the developing head. It can be postulated that any significant interference with these time-specific interactions could lead to incomplete fusion of the palatal shelves, both with each other and with the nasal septum. In addition, changes affecting the fusion and subsequent breakdown of the epithelial seam could induce malformation. Shelves which merge and fuse could be later disrupted, either by abnormal mechanical pressures or by growth traction if mesodermal bridging is incomplete. Such phenomena could lead to palatal fistula, submucous clefts or even complete rupture of the palate. One can hypothesize, therefore, that cleft palate may arise from one or more of the following causes: interference with the intrinsic shelf force; excessive head width, or diminutive palatal shelves; excessive resistance from the tongue; non-fusion of the palatal shelves; and fusion of the palate with subsequent breakdown.

Walker and Fraser (1956) were the first to propose the existence of an intrinsic shelf force which they ascribed to the presence of elastic fibres.

8
within the shelf mesoderm. Poswillo and Roy (1965) believed that the intrinsic shelf force arose from a combination of the expanding fibrillar mesoderm and increased mitotic activity along the lower margin of the shelf. This latter hypothesis was reinforced by the work of Andersen and Matthiessen (1967) who showed that increasing mitotic activity was an important factor contributing to the overall rise in tension of the shelf tissues. Verrusio (1970) proposed that a gradual decrease in the angulation of the cranial base could provide the "internal shelf force." It is likely in a multifactorial system such as palate closure that interference with the cranial base, be it mechanical or biochemical, will contribute to failure of palate closure.

Small palatal shelves may also contribute to palatal clefting; X-irradiation produces reduced mitotic activity and small palatal shelves in both rodents and primates. Other teratogens, including glucocorticoids, have been shown to do likewise. Mesenchymal deficiency, however it may arise, will obviously affect the developing palatal shelf mechanism with consequences leading towards malformation.

The role of the tongue in palate closure is still a matter for debate. Complete tongue obstruction over a time-specific period can produce 100% cleft palate in rodents, accompanied by a high proportion of moulding defects of the Pierre Robin type when induced by amniocentesis (Poswillo & Roy 1965).

Harris (1967) has shown that glucocorticoids produce oligohydramnios in mice, with postural-type defects of the palate caused by interference with angulation of the cranial base and subsequent tongue withdrawal. The role of corticosteroids in the induction of cleft palate is still not clear. . . . It has often been demonstrated that pregnant mice exposed to starvation, noise, cold or transportation near the critical time for palate closure will have a high incidence of cleft palate in their offspring.

Disturbances of fusion are believed by Smiley (1972) to be responsible for cleft palate. . . .

Submucous cleft palate, in association with bifid uvula, is likely to be a microform of posterior cleft palate. Submucous cleft palate can be induced in the mouse by the administration of phenobarbitone on Day 12 of development. It results in a delay in the centripetal flow of palatal shelf ossification of increasing magnitude from before backwards, which leaves an unreinforced palatal vault prone to rupture under growth traction or tongue pressures. The absence of bone reinforcement across the midline of the vault, combined with a deficient osseous inductive force in the midline of the palate, contributes to the failure of the velar mesenchyme to merge and elongate. Thus bifid uvula, either alone or combined with submucous cleft
palate, may result from disturbances in the processes of ossification and merging which take place between the seventh and tenth weeks of human development.

**S M I L E Y**

Gary R. Smiley, research orthodontist at the University of North Carolina at Chapel Hill, who raises ringneck doves as a hobby, worked as a laboratory technician with an embryologist during the summer of his freshman year in dental school. After graduate training in orthodontics he was invited by D. W. Warren to join the University of North Carolina Oral, Facial and Communicative Disorders Program and was thus provided research time for study of the secondary palate. He began work with anatomist A. D. Dixon and from him learned electron microscopy, which aided his investigation of palate fusion. He noted that, as the palate halves come together, the epithelial edges at their union must break down as nasal and oral accumulation of epithelial cells with lysosome bodies gives way by lytic activity to allow mesenchymal union across the midline. The epithelial plate, which is four to six cells thick at time of contact, separates the approaching mesoderm and, by degeneration and desquamation, goes to one-cell thickness at the presumptive area of fusion.

It does not take contact to cause this epithelial breakdown. Smiley notes that epithelial death is programmed before contact, so if timing is off for any reason, the keratinized edges of the cleft will not join each other. If, for instance, the palatal plates are late getting up to their horizontal position, programmed epithelial death along the cleft edges may proceed according to its own schedule, but it will be too late for union to be achieved by the time the palatal halves actually touch each other.

In 1968 M. Pourtois ascertained that

Fusion of palatal processes is time-critical. That is, if the palatal shelves meet after the critical period for fusion, fusion will not take place.
In 1972 Smiley noted:

Studies which indicate that the soft palate forms by a process of merging could easily explain the occurrence of a bifid uvula, but cannot explain the typical submucous cleft (which has an intact oral and nasal epithelium covering, muscle failing to reach the midline of the velum and usually a deficiency of bone at the posterior border of the hard palate). . . . There is no satisfactory single explanation for the etiology of the submucous cleft, or even for all clefts of the secondary palate. Nevertheless, an abnormal epithelial seam either in its formation or breakdown seems to be a most likely explanation for submucous cleft palate.

In the Archives of Oral Biology Smiley noted in 1975:

There are many studies on the midline epithelial seam during palatogenesis but few have distinguished between the presumptive hard and soft palates. Burdi and Faist (1967) and Burdi (1968) in man, and Bollert and Hendrickx (1971) studying baboons, described the development of both the hard and soft palate suggesting that the soft palate develops by mesenchymal merging rather than fusion because epithelial remnants are not found in the soft palate. However, Shah and Chaudhry (1974) indicated that the soft palate in hamsters formed by the process of fusion of the opposing epithelia. This inconsistency and lack of studies on soft palate formation in mice prompted this investigation on normal palatogenesis to determine whether the soft palate forms primarily by epithelial adherence and mesenchymal fusion or by merging.

He concluded:

A midline seam of epithelium was observed in the region of the future soft palate [in rodents and man for a relatively short period of time] indicating that epithelial adherence and mesenchymal fusion was occurring, and not merging. Epithelial breakdown was more rapid and complete in the presumptive soft palate and along the junction of the nasal septum and palate than in the midline of the future hard palate.

In his 1975 histological study in Archives of Oral Biology Smiley presented some convincing microscopic sections of human fetuses with and without the midline epithelial seam:
"Human 10-week foetus. (A) Future hard palate—midline epithelium is intact except for early breakdown nasally and epithelium is present between the nasal septum and palate. Note glossopalatine epithelium adherence and partial separation of the nasal septum from the palate. (B) Future soft palate—midline epithelium is present and appears to be in clumps in some areas."

"Human 12-week foetus. (C) Potential hard palate—midline epithelium is intact near the oral groove and has broken down nasally. Epithelium between the nasal septum and palate is absent. (D) Potential soft palate—midline epithelium is absent and the uvula has a furrow."

Based upon these observations . . . the hypothesis is proposed that the medial palatal epithelium is different and/or is acted upon differently in the anterior and posterior regions of the developing secondary palate. . . . Besides the palatal epithelium covering the oral and nasal surfaces, the hard and soft palate oral epithelium itself is different . . . Differential development also occurs in the underlying palatal mesenchyme, e.g., bone forms in
the hard palate and muscle in the soft palate. The palatal mesenchyme that develops into these different tissues could significantly influence the midline epithelium of the respective regions, suggesting that the difference in epithelial breakdown may not reside in the epithelium per se.

IN VITRO STUDIES OF PALATAL DEVELOPMENT IN MICE

Information on the embryological development of the palate is being gleaned from laboratory studies, and, of course, even greater knowledge will eventually be gained from such investigations.

Comparison of secondary palate development with different in vitro techniques, reported by Gary R. Smiley and William E. Koch in 1975, suggest:

The mesenchyme may play an important role in epithelial degeneration along the medial edge of palatal processes, since epithelial disruption did not occur in the absence of a viable underlying subjacent palatal mesenchyme.

Mary S. Tyler and William E. Koch of the University of North Carolina School of Medicine found that palatal processes removed from 12-day mouse embryos under particular culture conditions were able to continue their differentiation. The discovery enabled these researchers to make certain interesting deductions.

1. They confirmed the assumption that the epithelium at the tip of the vertically oriented palatal process was indeed the future medial epithelium of the horizontally positioned process because "it was always this epithelium at the tip of the vertical process which underwent regression."

2. Their results also confirmed for 12-day palatal processes the 1972 report by Smiley and Koch for 14-day mouse palates that "cellular contact between palatal processes is not a prerequisite for midline epithelial disruption."

3. They clarified the probability that "from the time it becomes identifiable as two ridges projecting from the maxillary arch, the mouse palate is capable of in vitro development and eventual fusion."
4. Finally, as suitable in vitro environmental conditions may be provided which are fully adequate for supporting morphogenesis and histogenesis of early palatal tissues, "it seems appropriate to suggest, therefore, that the concept of the 'acquisition' of a 'potential for fusion' does not identify a specific in vivo period of differentiation in the ontogeny of the mammalian secondary palate."

The administration of excess vitamin A to pregnant laboratory animals has been used extensively to produce a high incidence of cleft palate in offspring (A. Giroud and M. Martinet; H. Kalter; D. H. M. Woollam and J. W. Millen). Explanations differ, however, as to how maternal vitaminosis A leads to fetal cleft palate. Ravindra Nanda, formerly of the University of Nymegen, the Netherlands, and now at the University of Connecticut, Hartford, reported in the 1974 *Cleft Palate Journal* that his recent studies suggest:

Vitamin A retards the growth of the palatal process in vivo and subsequently the processes do not come in contact with each other at the morphogenetically determined time. The growth of the head subsequently moves the processes apart and fusion does not take place. However, the palatal processes retain their potential to fuse in vitro in the absence of cranio-facial structures and maternal metabolism and environment. This further suggests that vitamin A probably does not irreversibly disturb normal in vivo events of fusion mechanism.

Of course in vitro studies in the human are more enlightening since there are variations from development of the rodent primarily related to differences in behavior of the epithelium and the area of fusion.

Alastair N. Goss of Adelaide, South Australia, noted in the *Cleft Palate Journal* in 1975:

Only foetuses obtained by open surgical methods are suitable for palatal culture.

His five successful human cultures indicated that it is possible to stimulate in vivo fusion and in vitro epithelial pure formation and to investigate aspects of postfusion rupture. His case I demonstrated that in vitro fusion of human palates does occur,
the fused area showing rapid mesenchymal penetration. Cases III and IV demonstrated that if the intact palate is ruptured, normal healing by epithelial covering of the exposed mesenchyme occurs. As rupture of the previously fused palate was postulated as a mechanism of cleft palate in humans by H. Kitamura in 1966, Goss is in the process of showing that cleft palate can be induced in vivo in the rat by rupture of the intact palate. He reported:

With some types of palatal rupture continued growth of the face distracts the ruptured palate, thus increasing the width of the cleft. Other sites and sizes of palatal rupture heal with time so that the palate has reformed by birth.

Mark W. J. Ferguson of Queen’s University of Belfast (center of the troubled zone), who is interested in philately and paleopathology, has studied normal Wistar rat fetuses and those with cleft palate induced by 5-fluoro-2-desoxyuridine to elucidate the mechanisms of palatal shelf elevation and the pathogenesis of cleft palate. In 1977 he wrote:

The following theory is advanced to account for shelf elevation. The gradual build up of mucopolysaccharides, predominantly hyaluronic acid in the palatal shelves from day 14 to day 16.3 produces an increasingly powerful elevating force because of the turgor associated with the strong water binding tendencies of these substances. At 16.3 days this turgor reaches a threshold level and the elevating force becomes sufficient to overcome the resistance offered by the tongue, so enabling flip up to occur. The tongue is passively depressed, flattened, and its tip protruded out of the oral cavity, so making room for the common nasal passage. Other factors aid the transposition of the palatal shelves. Firstly, the undercutting of the underside of the shelf base by epithelium provides a fulcrum for shelf elevation. Secondly, maxillary and palatine osteogenic blastemata are present just exterior to the shelves and afford a firm base for flip up. The subsequent rapid invasion of the elevated shelves by these blastemata, and the ensuing ossification, soon consolidate the elevated palate. . . .

The present theory of shelf elevation postulates a confrontation between shelf elevating force and tongue resistance, and so it is not surprising that depression of the tongue should lead to premature shelf elevation (even at 14 days). It follows that cleft palate is theoretically producible in at least two ways: (1) by decrease in shelf force (as in F.U.D.R. fetuses); (2) by increase in tongue resistance (as seems probable in Pierre Robin-like anomalies.
produced by amniocentesis and contraction of the fetal membranes—Poswillo, 1968).

The avoidance, at least during the first twelve weeks of pregnancy, of drugs known to depress mucopolysaccharide synthesis is recommended.

In 1978, as the Winston Churchill Fellow lecturing at the University of Miami School of Medicine, Ferguson added:

The posterior one-fifth of the palatal shelves (i.e., the future soft palate) are horizontal from their first appearance at day 15 and so do not have to elevate. Furthermore these "soft palate shelves" do not approximate each other till day 17.5, whereas the future hard palate shelves have fused within five hours of flip up (which occurs at day 16.4).

All the F.U.D.R. induced abnormalities are readily explicable by depressed mucopolysaccharide synthesis and it is interesting to note that cleft palate in man is frequently associated with such anomalies.

### Ossification Centers of the Maxillofacial Region

According to Patten,

The primary support of the mandibular arch is Meckel's cartilage, which appears late in the seventh week. By the early part of the eighth week the mandibular bone starts to be intramembranously formed as a number of trabeculae lateral to Meckel's cartilage. As ossification spreads, the newly formed mandibular bone tends to enclose Meckel's cartilage.

The maxilla also appears early in the eighth week as intramembranously formed trabeculae in the mesenchyme of the maxillary process. It expands rapidly, but its extensions into the palatal shelves do not ordinarily appear until toward the end of the eighth week when the palatal shelves have moved up from their initial position on either side of the tongue. During the ninth week ossification progresses rapidly, and by the end of the tenth or beginning of the eleventh week, trabeculae extending from the primary ossification areas of the maxillae have laid the foundation for the bony support of the hard palate.

The more medial portion of the maxillary arch which carries the incisor teeth arises, during the late eighth or ninth week, from separate ossification centers formed in the part of the upper jaw which is of nasomedial origin. This independent origin of the incisive portion of the human maxilla emphasizes its homology with what, in lower forms, is a separate bone known as the premaxilla.
The cartilage primordia of the nasal septum and the nasal capsule are clearly differentiated by the end of the seventh week (embryos of 18 to 20 mm. C-R). The paired ossification centers for the vomer appear on either side of the lower part of the cartilaginous septum toward the close of the eighth or the beginning of the ninth week (embryos of 28 to 32 mm. C-R). By the eleventh week the two ossification centers are united below the septal cartilage. A week later the progress of ossification extends so that the periosteum of the vomer merges with that of the palatal bones just above their meeting with each other in the midline.

Here are schematic diagrams showing in the horizontal plane the changing relations at lip level in the developing upper jaw. (From Patten, Normal Development of the Facial Region, in Pruzansky's *Congenital Anomalies of the Face and Associated Structures*.)

As noted by Patten:

The youngest stage represents relations at the level of the originally shallow stomodeal depression before the rupture of the oral plate. The extent to
which the stomodeum is overhung by the frontal area is indicated by a broken line. . . . The downward component of the growth, which is particularly active in the nasomedial processes, soon brings them into the plane of section. Their union is normally a matter of merging rather than of fusing . . .

Of basic importance in understanding the development of the maxillofacial region are the relationships of the nasomedial processes. Their merging in the midline is readily seen in face views. . . . Their deep relations to the nasal septum are best seen when the developing upper jaw is looked at from below. In such views the deep continuity of nasomedial processes with the primordial nasal septum is clearly evident. Because these primordia lie at slightly different levels, this important relationship can only be suggested in diagrams of horizontal sections at the level of the upper lip by dotting in the position of the nasal septum, as has been done in [the accompanying diagrams].
2. Anatomy of the Palate

BONY SKELETON

The bony skeletal scaffold for the palate consists of the bones adjacent to the base of the skull, including the sphenoid and temporal, the premaxilla, the maxilla proper, and the palatine bone. These bones offer the origins and insertions of the muscles and provide the exits of the nerves and blood vessels serving the palate.

These bones, of course, are covered with a periosteum before receiving the palatal muscles and aponeurosis. The entire area is covered finally with a mucous membrane. In the hard palate area the mucosa is grayish pink and tightly adherent to the perios-
The mucous membrane of the oral surface of the soft palate is highly vascular and red in color, covered by a non-keratinized, stratified squamous epithelium. A layer of elastic fibers separates the lamina propria from the submucosa, which contains many mucous glands. At the free border of the soft palate, the oral mucosa changes to pseudostratified, ciliated, columnar epithelium of nasal mucosa.

**PALATAL MUSCULATURE**

Gustavo Sanvenero-Rosselli of Milan, a historical scholar who accumulated an extensive plastic surgery library, reminded cleft specialists at the 1973 Copenhagen International Cleft Palate Congress that Leonardo da Vinci understood the function of the soft palate in using vowels in speech and was cognizant of the varying levels attained by the palate during speech.

The first true anatomical descriptions of normal anatomy of the palate and pharynx were published by H. von Luschka in German in 1868.

**Veau**

Victor Veau of l’Hôpital des Enfants Assistés, Paris, published his classic book, *Division Palatine*, in 1931. Here he described the anatomy and pathology of cleft palate, gleaned from his vast number of cases and his hundreds of surgical procedures. In an explicit diagram of the palatal anatomy, he compared the normal aponeurosis and musculature with that in a cleft palate, showing some of the displacement and misdirection of fibers. One of his important contributions in palate surgery was the metallic suture used in gathering the fibers of the displaced muscles of the cleft edges parallel to each other.

**Whillis**

In 1930 anatomist James Whillis of Guy’s Hospital, London, showed that some fibers of the superior constrictor were inserted into the palatal aponeurosis and constituted a lamella he referred to as the “palato-pharyngeal sphincter.” It is possible in the
normal palate that Whillis' lamella may narrow the lateral pharyngeal recess, thus helping obtain ultimate palatal occlusion of the nasopharynx during contracture of the levator.

**Browne**

In the December 1935 *British Medical Journal* the eccentric, innovative Denis Browne of the Hospital for Sick Children, Great Ormond Street, London, postulated an orthopedic operation for cleft palate, justifying his design by his evaluation of the muscular activity during nasopharyngeal closure. He diagramed two overlapping muscle slings involving the levator palatini and the superior constrictor and noted the efficiency of this sphincteric action, which avoids the necessity for the large amount of contraction required by a simple, single muscle ring sphincter.

**Oldfield**

In 1941 Michael Oldfield of the General Infirmary at Leeds, England, noted that the muscular elements of the soft palate, apart from the uvular muscle, consist essentially of four slings. These are actually bilateral muscles which effect the sling-like function through their common insertion into the tissues of the soft palate. Superiorly, these muscles are the levator and tensor palatini; inferiorly, they are the palatoglossus and palatopharyngeus.
Braithwaite

Fenton Braithwaite of Newcastle upon Tyne received his master's in mathematics at Cambridge prior to his study of medicine and training with McIndoe at East Grinstead. He collected such exquisite antiques that it has been said his furniture was either just coming back from or just going off on loan to the Victoria and Albert Museum. Braithwaite was the first to realize the importance of correcting the malposition of the levator muscle in cleft palate surgery.

At the 1964 Cleft Palate Congress in Hamburg, Braithwaite noted:

Of the four muscular slings [of Oldfield] . . . , the levator descending on each side and passing downwards and forwards and the palatopharyngeus passing upwards and forwards, are more intimately concerned with speech.

He presented a diagram of the anterior view of the normal upper levator and lower palatopharyngeus muscle slings (heavy line) and the superior constrictor (fine line) from its attachments at the base of the skull to the hamulus and pterygomandibular raphe. He explained:

The levator sling elevates the soft palate in a backward and upward direction whilst the palatopharyngeal slings on contraction will approximate the posterior faucial pillars and narrow the pharyngeal aperture. When these two slings act together, each loop will afford counter purchase for the other and the interposed palati steadied by simultaneous contraction will convert each group into a "V," and the whole mechanism partakes of the features of an "X," as suggested by Podvinec (1952).

When this mechanism is examined from an anterior view through the open mouth, it may be seen that an isthmus is produced at the level of the soft palate by pulling in the lateral walls of the pharynx. It is obvious, therefore, that if the slings are not intact or indeed if the soft palate is scarred, this will not be effected.

As early as 1949 Braithwaite advocated constructing the levator muscle sling.
Kriens

Energetic Otto B. Kriens, professor of plastic and maxillofacial surgery, Bremen General Hospital, Germany, started his training in cleft lip and palate in 1963 under Karl Schuchardt in Hamburg. This was like commando training for combat because his teacher was a taskmaster whose students had to be strong to survive. I once heard Sanvenero-Rosselli refer affectionately to his friend Schuchardt as "der Führer." The pressure pushed Kriens into the study of palatal anatomy, and this is what he wrote to me in 1973:

When I started to study the anatomy of the palate, astonishingly there was hardly any literature on the cleft velum! Although the first reports about veloplasties date back prior to 1820, the first detailed anatomic descriptions of the normal anatomy of the palate and pharynx were given by Luschka in 1864. Thus the beginning of cleft palate surgery had to be without knowledge about the pathology to be corrected. No wonder the operations were morphological ones and in all methods the velar halves were used as entities, as architectural units, which had to be moved medially and/or dorsally so to achieve integrity.

Veau had postulated a closure in layers and he drew sketches of what he had seen during interventions. Braithwaite followed Veau's principle even further and dissected "muscles," apparently not exactly knowing which were where.

The dissections I did on normal palates and pharynges trained my three dimensional perception but did not reveal the essential pathology. Thus Professor Schuchardt was not wrong in rejecting an article speculating what seemed to be wrong in the velum. His dissent was not too polite, which probably prompted Professor V. Karfik to spontaneously invite me to Prague for an anatomical study (1966), which revealed characteristic findings (O. Kriens, Anatomische Untersuchungen am gespaltenen weichen Gaumen. Chir. Plast. Reconstr., 1967). The necessary changes of the velar closure were only too evident after the pathology had been exposed to sight!

Here is his 1967 diagram of the dissected soft palate from the oral aspect:
Also shown are two of his actual dissections with labels:
(1) Total bilateral cleft; fresh specimen. On the right can be seen the pars palatina/palatopharyngeus with its insertion on the posterior edge of the palatine bone, as well as the pars pterygopharyngea with its insertion on the hamulus, continuing to the aponeurosis. Between these, the belly of the levator is visible. (2) Total bilateral cleft; material fixed for some weeks in 10% formalin. On the left side of the specimen the levator belly with all its insertions is dissected and the pars palatina of pharyngeus, freed from its hard palate insertion, has been turned laterally.

In Plastic and Reconstructive Surgery in 1969, Kriens summarized the pathology of the cleft palate anomaly as the basis for planning reconstruction.

1. The forward and transverse displacement of muscular insertions (but the origins are normal).
2. The synergistically interwoven anterior portions of the levator veli palati and of the longitudinal portion of the palato-pharyngeal muscle (Veau's cleft-muscle).
3. The palato-salpingeal fascia forming a functional boundary between the constrictor pharyngeal and palato-pharyngeal muscles (levator space of cleft palate).
4. The plane of loose connective tissue between the palato-pharyngeal sphincter and the palato-salpingeal muscle (plane of blunt dissection in the velum proper).

5. The altered insertion of the hypoplastic horizontal (palatine) tendon of the tensor veli palati (no aponeurosis in the cleft velum).

His explicit drawings, presented in the *Cleft Palate Journal* in January 1970, show the normal palate, the basic pathology of cleft palate and the ideal goal of corrective surgery:

A and B show the normal condition of the three main muscle slings posterior to the interhamulus disk and palatal apron. C presents the levator palatini and palatopharyngeus muscles of the normal soft palate (arrow) in repose and contraction. D

presents the levator palatini and palatopharyngeus muscles of cleft soft palate in repose and contraction. E portrays Veau’s cleft muscle. F shows the major portion of Veau’s cleft muscle being joined to form the levator sling.

Always interested in controversy, I asked David Dickson to review Kriens’ outstanding 1967 article. These are his 1976 comments:
His statement that the tensor attaches to the Eustachian tube cartilage is debatable, if one views the literature, and probably wrong. The lack of aponeurosis in the cleft velum is a consistent finding of other investigators, but one in which we do not concur, based on our own histologic sections. His statement that the uvulus muscle is not involved in velopharyngeal closure is without support and is very likely wrong.

DIVIDENDS OF MODERN RESEARCH

Closure of palate clefts has been in progress for over two centuries, and with reasonably good functional results in a majority of cases during the last half century. Yet only in the last few years has the sophisticated knowledge of palatal anatomy and physiology been clarified to the extent that the plastic principle “return normal to normal position and retain it there” has finally been appreciated and applied to cleft palate surgery.

Velopharyngeal function, as its name implies, is a combination of movement patterns of the velum and of the pharynx. The principal components of the velum are a single intrinsic muscle, the insertions of several extrinsic muscles, a large amount of glandular material in the anterior inferior portion of the velum and an anterior aponeurosis. The single intrinsic muscle is the azygos uvulae. Muscles typically referred to as extrinsic muscles of the velum include the tensor veli palatini, levator veli palatini, palatopharyngeus, palatoglossus, and fibers of the superior constrictor. Pharyngeal muscles usually described as having a functional role in velopharyngeal movement include the superior constrictor and the salpingopharyngeus. Research over the past 40 years presents us with a view of the anatomy and physiology of this system which is frequently at variance with descriptions found in modern textbooks and old wives’ tales.

Dickson

As a guest speaker at the Ohio Valley Society of Plastic and Reconstructive Surgery in 1970, I was impressed by a presentation on “New Studies on Velopharyngeal Musculature” by David Ross Dickson, professor of anatomy and speech at the University of Pittsburgh. Dickson, dedicated, sensitive, tireless, is a teacher who chose speech pathology because of a long-standing interest.
in human communication. He has an inborn quirk that compels him to attack ideas simply accepted, without reason or because of tradition. He believes in the scientific method, not just as an exercise in academia, but also in personal conduct, social rules and political viewpoints, searching to know the "real question" and considering unconsidered options. Yet he can be found in all weather collecting English brass rubbings, made by a technique developed in eleventh-century Netherlands, atop famous British sarcophagi, such as those of Sir Thomas Bullein, father of Anne Boleyn, Lady Margaret Chayne, and especially the Black Knight of Canterbury.

In 1972 Dickson was invited to Miami to lecture and at that time helped place the palatal muscles in plasticine on the base of a skull. Now, as professor of pediatrics at the University of Miami School of Medicine and director of speech and hearing at the Mailman Center for Child Development, he will describe the current knowledge on the palatal musculature with its variations from the previous standards in *Gray's Anatomy* and even in *Cleft Lip and Palate*, published in 1971. The accompanying drawings were made from dissections, aided by the microscope, of embryonic heads by Wilma Maue-Dickson, previously of the University of Pittsburgh and now associate professor of anatomy at the University of Miami School of Medicine.

Comparisons of muscles of all the usual types of clefts in 18 mature stillborn children with those of four normal stillborn children have been obtained from Miroslav Fára's and Jiří Dvořák's dissections at the Charles University, Prague.

Dickson will describe muscles in the normal palate; Fára and Dvořák, muscles in cleft palate.

Dickson:

The *tensor veli palatini muscle* arises from the angular spine of the sphenoid bone, the scaphoid fossa, and the pterygoid fossa. Medial to these attachments, fibers of this muscle arise from the lateral membranous wall of the Eustachian tube. These fibers converge on a medial tendon which passes around the hamulus of the medial pterygoid plate. At this point the tendon is covered with a synovial sheet. The tendon then bends into a horizontal plane and enters the velum as the anterior aponeurosis, which is composed of a flat tendinous sheet in the anterior third of the velum, superior to the...
Adult lateral head dissection shows the tensor veli palatini muscle.

glands and muscles which are found in that portion of the velum. It has been thought that since the notch of the hamulus through which the tendon passes is slightly inferior to the velum, muscular contraction of the tensor palatini muscle would place tension on the velar aponeurosis. There is no evidence whatever that such tension exists as a function of contraction of this muscle. Nor is there any convincing evidence that tension placed on the aponeurosis would have any particular functional significance. It has been well documented, however, that this muscle, upon contraction, opens the Eustachian tube, and that no other muscle serves this function. In 1862, Henle disclosed that the tensor palatini muscle exerts a force predominantly effective on the auditory tube with only a minor part of its activity directed to the normal palate. In 1923, Rich confirmed this in dogs and in 1964 Ruding claimed that the tensor palatini muscle had only an auditory function in the cleft palate. It is interesting to note that the superior part of the tensor palatini muscle forms a tendon which passes through the cranial base and forms the inferior tendon of the tensor tympani muscle, the other end of which attaches to the malleus. Thus these two muscles, the tensor tympani and the tensor palatini, form a two-bellied muscle. There has been speculation that these two muscles may have a conjoint function in aeration of the middle ear; the tensor veli palatini by opening the Eustachian tube, and the tensor tympani by increasing middle ear pressure by drawing the tympanic membrane medially, which would, in turn, possibly lower the threshold for Eustachian tube opening. This speculation has arisen from the evidence that three factors are necessary for Eustachian tube function. The first is contraction of tensor palatini muscle, the second is production of surfactant to release surface tension within the Eustachian tube, and third is air pressure differential between the middle ear and pharynx.

Fára and Dvořák:
The tensor [in the newborn cleft] was somewhat thinner than in a normal newborn child . . . [with] a few bundles attached to the hamulus and the tendon itself . . . The front part of its bundles extended along the rudimentary palatine aponeurosis, toward the posterior nasal spine, and was partially attached to the spine or (laterally) to the posterior edge of the palatine bone. Some of the tensor fibers radiated into the aponeurosis. The main part of the tendon, however, arched backward to the cleft edge of the velum . . . [and thus became either] partly dispersed, and then a triangular area passed into the front bundles of the levator muscle, or [in two-thirds of the autopsies] . . . not disperse [d] at all, but passed into the anterior bundles of the levator muscle as . . . a thick and free single muscular-tendinous bundle.
APONEUROSIS IN CLEFT PALATE

This is one area where Veau, Fára and Dickson have some disagreement. As indicated by Veau in *Division Palatine* (1931), the classic schema of the normal palate described by Fára consists of an anterior third (aponeurosis), a middle third (muscular) and a posterior third (mucous membrane). Veau stated:

In cleft palate, the anterior third (aponeurosis) is lacking and there remains only a solid fibrous fasciculus for the aponeurosis; this fasciculus is attached to the bone. *There is no real palatine aponeurosis in the palatine division* [in cleft palate].

In 1970 Miroslav Fára and Jiří Dvořák discussed the deficiency of the palatine aponeurosis in the cleft palate. They wrote of the tensor veli palatini muscle:

It has no proper chance to function fully; thus, it does not develop as it should. The absence of a fixed point in the midline (which is necessary for the insertion of the fan-shaped tendon) causes not only an incomplete and atypical growth of the tendon itself, but a marked hypoplasia of the palatine aponeurosis as well. Indeed, the very existence of this aponeurosis is due to the extension and penetration of the tensor tendon into it. Thus, the very aponeurosis in its lateral area is now very short; as it approaches the cleft margin, it practically disappears.

David and Wilma Dickson of the University of Miami take a different stand and present microscopic sections of a fetus with cleft palate which clearly demonstrates the palatal aponeurosis.
Dickson:

The levator palatini muscle is a cylindrical muscle which has its origin from the petrosal portion of the temporal bone anteromedial to the entrance of the carotid canal. From that point the muscle courses inferior to the Eustachian tube and occasionally gives off a few fibers to the cartilaginous wall of the tube. Just before entering the velum, the muscle passes lateral to the torus tubarius, which is the enlarged inferior end of the Eustachian tube cartilage. From that point the muscle passes into the soft palate with fibers spreading over the posterior three-fourths of the velum. These fibers cross toward the midline where they join with fibers from the opposite levator palatini muscle. Within the velum the levator palatini muscle is the most superior muscle with the exception of the azygos uvulus.

The levator palatini muscle displaces the velum in a superior and posterior direction. The left and right levator muscles form a sling widely separated superiorly and interlacing in the velum inferiorly. From studies of this muscle it is probable that the course of the muscle from velum to cranial base is in a direct line with motion of the velum toward velopharyngeal closure for speech.

Fará and Dvorák:
[In the newborn cleft this muscle was] considerably hypoplastic bilaterally; sometimes, [it] did not exceed half the muscle thickness in normal newborn children. The thinner the muscle belly, the thicker the layer of loose connective tissue in its bed [Veau, 1931] . . . . The posterior bundles ran posterolaterally toward the . . . palatopharyngeus, penetrat[ing] the posterior palatine arch [near] the base of the uvula . . . . The medial bundles radiated like a fan into the margin of the cleft. The anterior bundles were either . . . attached by a triangular tendinous area coming laterally from the posterior nasal spine to the posterior edge of the palatine plate ( . . . [and also] into the tensor tendon), or . . . directly linked up with the compact part of the tensor tendon. . . . In the less serious forms of clefts . . . some anterior bundles of the levator advanced for some millimeters along the cleft margin of the palatine plate as a part of Veau's "cleft muscle."

Dickson:

The palatoglossus muscle is slender and arises from transverse bundles of the tongue. It passes through the palatoglossal arch and into the inferior middle portion of the velum. It would appear capable of lowering the velum or raising and retracting the tongue. However, electromyographic evidence suggests that the palatoglossus muscle is active in tongue function but not in velar lowering. It may also act to narrow the opening between the mouth and pharynx during swallowing.
Fára and Dvořák:

[This muscle in the newborn cleft] passed [forward] in the cleft margin to the posterior edge of the palatine plate . . . [and] extended, in many cases, beyond the posterior edge . . . [to become] inserted more frontally (3 to 5 mm) in the oral periosteum of the hard palate.

Dickson:

The palatopharyngeus muscle arises from the lateral and posterior pharyngeal walls and inserts into the velum. Its superior fibers arise from a complex intermingling with the superior constrictor muscle. These fibers arise from a level just below the most superior fibers of superior constrictor and pass horizontally into the posterior three-fourths of the soft palate, inferior to the fibers of the levator palatini muscle. Lower fibers of the palatopharyngeus muscle arise from the inferior part of the lateral wall of the pharynx, medial to the middle and inferior constrictor muscle. Some fibers may arise from the thyroid cartilage. These lower fibers pass through the palatopharyngeal arch to insert into the velum.

While there is little electromyographic evidence regarding the function of this muscle, it is felt that it decreases the distance between the palatopharyngeal arches during swallowing and also acts to lower the velum.

Fára and Dvořák:

[This muscle in the newborn cleft was] relatively well developed. . . . Even though the smaller part of its fibers ended in the cleft margin, most of its bundles passed forward along this margin and inserted on the posterior edge of the palatine plate. . . . Some fibers advanced along the cleft margin, together with the bundles from the levator as a part of the “cleft muscle.” . . . The circular fibers of the posterior pharyngeal wall were difficult to distinguish from the bundles of the superior constrictor. . . . (50 percent of our cases) had condensation and even some thickening of the circular fibers . . . cross [ing] in the Passavant pad, which bulged visibly in the autopsy material. This was not seen in any case of sectioned normal newborns.

The powerful insertion of the pars pterygoidea extended from the hamulus across the medial plate of the pterygoid, as far as the lateral portion of the aponeurosis.

Dickson:

The azygos uvulus muscle is a well-developed unpaired muscle traversing the velum in an anterior-posterior direction superior to all other muscles of the velum. Its origin is apparently from the anterior velar aponeurosis with occasional tendinous fibers passing forward to the posterior nasal spine. It
passes posteriorly through the superior midline of the velum to insert into the uvula.

The function of this muscle has never been studied. However, this muscle is thickest as it passes superior to the main mass of the levator palatini muscle, approximately two-thirds of the way back in the velum from the hard palate. This is the portion of the velum which is most displaced in velopharyngeal closure. During velopharyngeal closure, particularly in the male, the superior surface of the velum become convex from anterior to posterior, creating a considerable hump in the superior portion of the velum above the level of contact of the velum with the posterior pharyngeal wall. This superior hump in the velum has been called the "levator eminence" but is more likely to be created by contraction of the uvulus muscle which could thicken the velum in this area. Therefore, at least until some further evidence develops, it is probably more reasonable to call this area the uvular eminence. This could create some confusion but no more so than is presently available in velopharyngeal terminology.

It is of interest that as early as 1880 R. Falscon commented on the bulk of the musculus uvulae. In 1969 R. Pigott confirmed with nasendoscopy the presence and importance of the musculus uvulae in speech:

The enormous bulk of the musculus uvulae could be seen. The majority of subjects had a large ridge down the soft palate, occupying the central $\frac{1}{3}$, rising to a height almost equal to its width. In other subjects, it was less prominent, but it was never absent. . . . During speech movements . . . the levator sling could be seen to tighten into a bar throwing the convexity of the musculus uvulae bulge up and back to fit into the concavity of the posterior superior pharyngeal wall. . . . The possible role of the musculus uvulae in giving a suitable convexity to the upper surfaces of the levator at the point of contact . . . is of particular importance in the light of Broomhead's finding that this muscle is not supplied by the pharyngeal plexus, but by the lesser palatine nerve.

As part of the fruit from David and Wilma Dickson's research on the morphology of the musculus uvulae, Nabil A. Azzam and David P. Kuehn of the University of Iowa reported their findings, confirming the Dicksons' observations in the 1977 Cleft Palate Journal. They noted:

The morphology of the musculus uvulae was studied utilizing detailed gross anatomical dissections and histological sectioning of the soft palate in seven adult human cadavers. The results indicated that the musculus uvulae is paired. . . . Each bundle takes origin lateral to the midline from the
tendinous palatal aponeurosis posterior to the hard palate and just anterior to the insertion of the levator veli palatini muscle. The two bundles converge in an area overlying the sling of the levator muscle and course along the dorsum of the soft palate, terminating as two separate bundles which subdivide and insert between the mucous glands of the uvula proper into the connective tissue and basement membrane of the mucosa. Because of its location and size, it appears that contraction of the musculus uvulae would add bulk to the dorsal surface of the elevated soft palate [arrow], thus aiding in occlusion of the velopharyngeal portal during speech and deglutition.

Fára and Dvořák:

[This muscle in the newborn cleft] passed in the cleft margin and its bundles intermingled with those of the palatopharyngeus and the levator.

A more detailed description of the uvular muscle in cleft palate was given by Elizabeth Ann Latham at the Third International Congress on Cleft Palate in Toronto, based on serial histological sections of two postmortem infants and a Plexiglas reconstruction.

The Musculus Uvulae was divided by the cleft into its bilateral component muscle bundles which were seen on the medial border of each palatal shelf. The M. Hemiuvulae originated anteriorly in relation to the border of the hard palate formed by the palatine bone. Here it blended with other velar muscles. It stayed in the medial border as it coursed posteriorly beneath the mucous membrane and turned more onto the inferior border prior to entering and dispersing in the hemiuvula. A coronal section through the cleft soft palate at the level of the pterygoid hamulus showed the hemiuvular muscle on the medial border of the palatal shelf between the mucous membrane and deeper glandular tissue. This was clearly seen at a higher magnification which showed the muscle fibers sectioned rather obliquely.

PHARYNGEAL MUSCLES

The constrictor pharyngeus muscles consist of superior, medial and inferior portions. The superior portion of the constrictor complex may be involved in velopharyngeal function. The superior constrictor muscle fibers have their origin at the hamulus and the adjacent pterygomandibular raphe. Fibers pass around and through the lateral pharyngeal wall and "join corresponding fibers of the opposite side in a tendinous strip, the pharyngeal raphe, which runs in the midline from the pharyngeal tubercle of
the occipital bone throughout the entire length of the pharynx.”

To complete the circle of this “U,” Whillis found that some of the upper fibers inserted into the palatal aponeurosis formed an actual palatal-pharyngeal sphincter. In 1948 Whillis at Guy’s Hospital patiently pointed out to me in detail in cadaver specimens the muscles involved in the “palato-pharyngeal sphincter.” He emphasized the importance of the superior pharyngeal constrictor’s lateral attachments into the soft palate, which he had found in 1936 under Wardill’s none-too-gentle prodding.

Dickson:

While the superior constrictor muscle undoubtedly has an important function in narrowing the pharynx during swallowing, its function in velopharyngeal closure is debatable. While Passavant’s ridge has never been studied electromyographically, it is quite probable that this inbulging of the lateral and posterior pharyngeal walls seen in many patients with cleft palate and other forms of velopharyngeal insufficiency is a function of the most superior fibers of superior constrictor which enter the velum. Some writers have suggested that the superior constrictor is also responsible for the lateral wall motion seen in normal velopharyngeal closure. However, the fact that Passavant’s ridge occurs below the normal site of velopharyngeal closure, the fact that the lateral pharyngeal walls move medially and posteriorly rather than medially and anteriorly in normal velopharyngeal closure and the fact that the superior constrictor lies at the level of the hamulus, rather than higher in the nasopharynx, suggests that it probably is not involved in velopharyngeal function during speech. Available electromyographic evidence supports this contention. The continuing debate regarding the role of this muscle in lateral wall movement during speech has been reinforced by findings of considerable variability in patterns of motion of the velum and pharynx from subject to subject in radiographic research.

The salpingopharyngeus muscle consists of a few fibers arising from the inferior portion of the palatopharyngeus muscle which passes through the lateral pharyngeal wall superiorly to attach to the torus tubarius. These muscle fibers are few in number and frequently absent in normal specimens. Commonly, the salpingopharyngeal fold through which these fibers pass consists entirely of gland and loose connective tissue.

Fára and Dvůrak:

Fára noted no difference in the pharyngeal muscles in the newborn cleft and the normal.
Dickson:

In summary, from a functional standpoint it is apparent that the levator veli palatini muscle is the principal and quite possibly the only muscle to function for elevation of the velum in speech. In addition, the medial and posterior motion of the lateral pharyngeal wall during velopharyngeal closure for speech must be accounted for. Since the salpingopharyngeus muscle is frequently absent in the normal, and since the superior constrictor muscle is probably too low to account for motions seen in the nasopharynx, and because available electromyographic evidence is not supportive of the contention that this muscle acts during velopharyngeal closure for speech, some other mechanism must be found. A likely answer is to be found by noting the slinglike arrangement of the entrance of the levator muscles through the pharynx and into the velum. As was noted above, the levator passes lateral to the torus tubarius prior to entering the velum. Thus, on contraction it would be quite likely that the levator muscles would not only lift the velum but would displace the lateral pharyngeal walls and tori tubarius in a medial and posterior direction. It is, therefore, a most reasonable hypothesis that both the pharyngeal and velar components of normal velopharyngeal closure for speech are a function of the levator palatini muscle.

With regard to the cleft condition, here are a few of Fára's deductions:

The differences between the normal and the cleft arrangement of the muscles of velopharyngeal closure are considerable, but . . . occur because the muscles extending toward the central line of the soft palate cannot attach themselves to the punctum fixum in the midline of the velum; so they insert at some substitute points. These points, however, prevent the muscles from becoming fully functional; therefore, their development is retarded. With the preservation of normal origins, the atypical insertions and the hypoplasia of the muscles are the main pathological features in the cleft velum . . . .

The levators in clefts illustrate clearly the effect of a morphological disorder on function, not only from the point of view of quantity but also of quality. Indeed . . . the effect of the activity of these muscles in a cleft palate is almost opposite to that in a normal one. While the muscles of both sides normally join in the raphe to form a sling lifting the palate upward, in cleft palates each muscle pulls its own half of the soft palate in an entirely different direction (i.e. supero-laterally), causing a further widening of the cleft.
Robert Shprintzen of Montefiore Hospital, the Bronx, New York, of Spanish descent with a Russian rinse, made an interesting study, with McCall, Skolnick and Lencione.

The frontal and lateral cinefluorographic views of five normal subjects performing speech, blowing, and whistling tasks were synchronized in order to observe where the greatest degree of medial movement in the lateral aspects of the pharyngeal walls was occurring in relation to structures observed in lateral view. The results . . . indicate that for all five subjects, maximal medial excursion in the lateral walls of the pharynx occurred at the level of the full length of the velum and hard palate, well below the levator eminence. It is hypothesized that the observed interaction may be due to the select contraction of those fibers of the superior constrictor muscle which enter the velum via the lateral walls and those fibers attached to the pterygoid plates, as well as levator muscle activity.

This led to their suggestion of the possibility that the kinesiological observations of this study would seem to indicate that both the levator and the superior constrictor are necessary to closure.

**NASOPHARYNX**

The pharynx is related to the sphenoid bone and to the basilar part of the occipital bone above, and descends into the esophagus. Anteriorly, it opens into the nasal and oral cavities and the larynx; posteriorly, from before backward, it is related to the prevertebral layer of fascia, prevertebral muscles and upper six cervical vertebrae. Laterally, such structures as the styloid process and its associated musculature, the medial pterygoid muscle, the carotid sheath and its contents and the thyroid gland are present.

The nasopharynx is actually the posterior portion of the nasal cavity communicating with the oropharynx through the pharyngeal isthmus or hiatus and bounded by the palatopharyngeal arches, the soft palate and the posterior wall of the pharynx. Embedded in the mucous membrane of the posterior nasopharynx is the mass of lymphoid tissue known as adenoids. The pharyngeal isthmus is encircled by a lymphatic ring, the nasopharyngeal tonsil (adenoids) above, the palatine tonsils laterally and the lingual tonsils below.
Dissected sagittal view—normal adult palate and pharynx showing auditory tube, pharynx and musculature of the velum (palate).
Inferior view—newborn palate and related structures

- Premaxilla
- Palatine process of maxilla
- Palatine bone
- Hamulus
- Pterygoid plates
- Petrous part of temporal bone
- Mandible
- Hard palate
- Soft palate
- Palatopharyngeal fold
- Palatoglossal fold

NORMAL
Mucosa reflected and palate dissected.
Palate dissected to show auditory tube and related structures.
Superolateral view of the palate and pharynx, showing position of auditory tube and the tensor veli palatini, levator veli palatini and superior constrictor muscles.
The triangular pocket in each lateral wall of the nasopharynx is called the tonsil and is formed by the diverging pillars of the tonsil, the anterior pillar formed by the palatoglossus muscle and the posterior pillar formed by the palatopharyngeus muscle. Cradled between the pillars in each fauces lies the palatine tonsil.

The most significant structure in the lateral wall of the nasopharynx is the auditory, or Eustachian, tube.

THE EUSTACHIAN TUBE

Dickson

Wilma Maue-Dickson of the University of Miami School of Medicine, a compulsive, analytical perfectionist, played violin in the Exeter Symphony Orchestra in England, climbed Kilimanjaro, and saw her first severe unoperated unilateral cleft lip while in the Peace Corps in Ethiopia. David Dickson was her major professor in head and neck physiology at the University of Pittsburgh. Their mutual love of teaching, research and human communication overcame this obstacle to their relationship and resulted in marriage. Her compulsion not just to observe craniofacial pathologies but to speed their extirpation was probably "fueled" most effectively the day she walked into the storage room for some 15,000 human embryos and fetuses at the University of Pittsburgh’s Cleft Palate Center in 1969 and recognized that a gold mine of information for living children with facial anomalies lay in that 15' X 15' room.

Since then she has carried out numerous microscopic dissections of fetal heads which have placed her among the foremost head and neck anatomists. She has a special affection for the Eustachian tube. This is her 1976 position:

In the sixth century B.C., a man named Alcmoeon became interested in how goats "breathe through their ears" and gave us our first description of the structure of the auditory tube, which he felt served this function (Macbeth, 1959). Later, during the sixteenth century, the tube was described in further detail by the Italian anatomist, Bartolomeo Eustachio, for whom the tube was named. He was one of the first to describe accurately its structure, course, and relations. He compared the tube to a quill pen and divided its bony and cartilaginous parts, but did not hazard a guess as to its function.
The anatomy of the adult auditory (Eustachian) tube has been carefully documented, as has its histology, and large strides have been made in our understanding of its embryologic and fetal development. It courses from the nasopharynx to the middle ear in a posterior lateral superior direction. The anteromedial two-thirds of the tube, called the cartilaginous portion, consists of a superomedial cartilaginous wall and an inferolateral membranous wall. The posterosuperior one-third of the tube passes through the petrous portion of the temporal bone and is referred to as the osseous portion, protympanum, or semicanal. The division between the cartilaginous and osseous portions is marked roughly by a constriction called the isthmus. It has been well documented (Sucheston and Cannon, 1971) that the membranous part of the tube is lined with pseudostratified, ciliated, columnar epithelium, while the medial lamina and roof are lined with cuboidal epithelium. Muco-serous glands and goblet cells are present at the pharyngeal orifice and in the mid-portion of the tube, but are absent at the tympanic orifice. Lymphoid tissue is present at both orifices but is far less abundant in the mid-portion of the tube.

The auditory tube is of particular interest because it provides a dynamic link between the nasopharynx and middle ear. The middle ear has no direct outlet via any other route than the auditory tube. The auditory tube, therefore, provides a mechanism whereby pressure can be equalized across the tympanic mem- brane. The cartilaginous part of the tube is roughly elliptical in configuration and is normally collapsed, but opens during swallowing, coughing and sneezing. The osseous portion is obligatorily open.

The tube is also of clinical interest because it provides an avenue for the outflow of fluids from the middle ear. Unfortunately, it also provides an access route for the spread of infection from the pharynx to the middle ear and mastoid area.

The specific mechanism for opening the auditory tube involves an interaction of muscular force, pressure differential, ciliary action, and possibly the aid of a surface tension-reducing substance such as surfactant. Muscular activity associated with tubal opening has been a source of debate for years. Normal tubal function has been associated at one time or another with at least six different muscles, including the superior constrictor, the palatopharyngeus, the medial pterygoid, the lateral pterygoid, the levator veli palatini, and tensor veli palatini. Of this group, only the latter muscles have direct attachments or important spatial relationships to the tube. Fortunately, this debate about the function of the muscles associated with the auditory tube has been settled by an elegant study conducted by Arnold Rich [experimental pathologist at Johns Hopkins Hospital] in 1920, which indicated clearly that the tensor veli palatini is the sole muscle responsible for tubal opening at the isthmus. More recent EMG studies have supported this fact.
Another debate on tubal function concerned innervation of the tensor veli palatini muscle, which has been variously ascribed to cranial nerves V, VII, X, and XI. However, Rich (1920) demonstrated convincingly that tensor veli palatini is innervated via the mandibular branch of cranial nerve V.

Levator veli palatini may assist in opening the pharyngeal orifice of the tube by moving the torus tubarius, the cartilaginous expansion of the tube at its pharyngeal orifice, posteriorly and medially during swallowing. However, this action would not aid in tubal opening at the isthmus.

Cleft Palate: The auditory tube is of further clinical interest because of its apparent involvement in cleft palate. It has been demonstrated that babies with cleft palate have an almost 100 per cent incidence of middle ear effusion, frequently accompanied by hearing loss. Numerous studies have been conducted to ascertain why this is true; none have completely solved this puzzle, but it clearly involves abnormal Eustachian tube function. A recent anatomical study conducted by Maue-Dickson (1975) indicated that human fetuses with cleft palate consistently show the following characteristics:

As compared to the non-cleft, there are narrower and smaller auditory tube lumina, which are more widely separated than in normals.

The greatly enlarged auditory tube cartilages are also more widely separated than in normals.
There are more widely separated pterygoid plates than in the normals.

There is significantly reduced pharyngeal height, but greatly increased pharyngeal width.

One conclusion from these data is that the space between the lateral pharyngeal wall and the side wall of the cranium is substantially reduced and that the tube may suffer a mechanical disadvantage as a result. This problem may be reduced by craniofacial growth, which might relieve stress on the tube. This suggestion is consistent with the observation that children with cleft palate typically have reduced middle ear problems within the first few years of life.

Interestingly, while the interruption of the levator veli palatini muscle and of other muscles which traverse the soft palate is obvious in cleft palate, there is no indication that the extra-palatal anatomy of the tensor veli palatini muscle is abnormal in cleft palate, and even in severe cleft of the palate, the presence of a well-defined palatal aponeurosis (the tendon of tensor) can be demonstrated in the palatal tags in histologic sections.

In summary, while the adult structure of the auditory tube is fairly well understood, more information is needed on the specific morphology of the tube and associated musculature in cleft palate. Based on data collected recently, there is a strong suggestion that cleft palate may in fact co-exist with, or even be caused by, cranial base deformity occurring early in the embryogenesis of the involved structures, and that auditory tube malfunction observed clinically in cleft palate may be related to this deformity.
The internal maxillary artery gives off the descending palatine artery, which, in turn, gives off several branches to the tonsils and soft palate. It then passes through the posterior palatine foramen, just above the periosteum, and proceeds forward close to the alveolar margin on each side as the greater (major) palatine artery to the incisive fossa. At that point it sends a terminal branch through the incisive foramen to anastomose with the terminal branch of the sphenopalatine artery. The sphenopalatine artery is also a branch of the internal maxillary artery. One of its branches descends to the incisive canal and anastomoses with the terminal ascending branch of the posterior palatine artery to form the anterior blood supply to the palate. The posterior septal artery arises from the sphenopalatine artery in the roof of the nasal cavity and courses down the groove of the vomer to the incisive foramen. As noted by Brescia, a wide, rich anastomosis is formed between the posterior septal, major palatine and ascending septal branches of the superior labial arteries.

The blood supply to the anterior alveolar process of the maxilla comes from the arterial complex composed of the major palatine, anterior and posterior superior alveolar and branches of the sphenopalatine arteries.

In the complete bilateral cleft lip and palate, as noted by plastic surgeon W. B. Slaughter, along with J. V. Henry and J. C. Berger, the union of the superior labial arteries is lacking; thus they do not contribute to the blood supply of the philtrum. Also absent is the anastomosis of the posterior septal artery with the major palatine artery. Therefore, premaxilla and philtrum must derive their blood supply from the posterior septal artery and to some degree from the lateral and terminal branches of the anterior ethmoid vessels which pass through the columella. Fortunately, in cleft specimens, there is usually one well-developed vessel on either side of the premaxilla in the region where the incisive foramen should have been. Each of these vessels passes anteriorly and inferiorly into the philtrum and forms an arcuate anastomosis across the midline in the inferior part of the philtrum.
The scholarly plastic surgeon I. W. Broomhead dissected a fetal head for blood supply studies. In 1951, in the *British Journal of Plastic Surgery*, he reported:

The greater palatine artery [GrPalArt] supplies the oral surface of the hard palate and gives off a few fine branches which perforate the horizontal plate of the maxilla to supply the nasal mucosa. It also sends twigs to the gums and palato-glossal arch. The lesser palatine artery [LsPalArt] supplies about the anterior half of the oral surface of the soft palate. A branch of the facial artery, the ascending palatine artery [AscPalArt], is the largest vessel entering the soft palate. It ascends on the lateral side of the superior constrictor muscle to turn downwards and forwards into the soft palate between the tensor [Ten] and levator palati [Lev], giving small branches to these muscles. There are two main terminal branches, the anterior one passing along the anterior border of the levator palati and the posterior one passing through this muscle. On approaching the midline the posterior division turns backwards to run the length of the soft palate to the uvula. Small twigs from the tonsillar [TonsArt] and ascending pharyngeal arteries [AscPharArt] also reach the soft palate. The branches from the tonsillar artery enter along the palato-glossus muscle [PalGloss], and those from the ascending pharyngeal pierce the superior constrictor [SupConst] and pass along the fibres of the palato-pharyngeus muscle [PalPhar].

Broomhead concluded with:

The vascular supply of the soft palate is not endangered by the [standard third-degree cleft palate] operation.

Muriel E. Morley of Newcastle upon Tyne was the speech therapist for many years for Wardill's cleft palate cases. In her little 1962 book, *Cleft Palate and Speech*, she noted:

It is of importance in surgery that the blood supply to the palate enters through the bone and not, as in other parts of the body, through the periosteum. . . . It is therefore possible at operation to strip the periosteum from its bony attachments without interfering with the blood supply.

Stefan Demjen of Bratislava referred to the lack of knowledge of the blood supply to the bony palate:

The hard palate and its mucoperiosteal membrane are supplied by blood from nasopalatine vessels and from the descending palatine arteries. The bony palate is said to have an independent blood supply and is thus protected from necrosis following conventional palate operations.
It is important to know whether the presence of a cleft in the palate changes the vascular arrangement. The data are minimal, but in 1974 David Dickson et al. in their "Status of Research in Cleft Palate" reported:

Blood supply: no specific investigations of the blood supply to the cleft palate have been found except for Frederiks, who reported slight differences between cleft and non-cleft.

Edith Frederiks of Leiden, the Netherlands, did note in 1972:

In the secondary palate the existence or absence of a cleft makes little difference to the vascular pattern.

Yet in the 1973 "State of the Art" report on clinical research in cleft lip and cleft palate, Spriestersbach, Dickson, Fraser, Horowitz, McWilliams, Paradise and Randall proposed the likelihood of a variability in arterial arrangement in cleft and non-cleft humans. This was confirmed when, in 1977, William P. Maher of Marquette University and the Medical College of Wisconsin reported postmortem arteriographic studies of the major branches of the pterygopalatine portion of the maxillary artery in three cleft palate and nine non-cleft near-term human fetuses. The study revealed numerous variations in each facial half in both cleft and non-cleft palates. Maher noted anatomical aspects pertinent to palate surgery:

Lateral branches of the greater palatine arteries provide substantial contributions to all maxillary deciduous and permanent dental structures. Incisions made parallel to the alveolar ridge and lateral to the greater palatine artery are designed for purposes of moving the pedicle flap medially to close the cleft. These incisions completely sever nutritional supply from the palatine network to all the maxillary teeth. However, the developing dental structures are also supplied by dental branches from the superior alveolar artery and gingival-osseous branches from the great facial network. Whether these major contributions are able to provide adequate nutritional sources for normal dental development or whether temporary nutritional loss from the palatal network as the results of sectioning might be responsible for morphological defects in hard dental structures remains to be clarified.

The palatal mucoperiosteum is detached from its bony base in the preparation of a palatal pedicle flap, and as a result the recurrent osseous branches are severed. These branches vary in size, number, location, and
distribution frequency. Bleeding from their cut stumps at the bony surface may be judged . . . insignificant at the time of surgery. However, after the flap is repositioned, should bleeding continue . . . blood may pool beneath the repositioned flap.

Maher also noted:

Terminal branches of the greater palatine artery were found to anastomose variously with: 1) the lateral nasal septal artery, 2) the superior alveolar artery, 3) labial branches of the facial artery via branches of the palatal artery that pass through the maxillary fissure or via the external nares, and 4) with its companion on the contralateral side. One or more combinations of these variations may occur in both facial halves. . . . Furthermore, crossover anastomoses between terminal branches of major vessels can occur.

Maher admits to being a two-kind-of-vessel freak:

I have a canoe that is called Capillary, a nice runabout called Arteriole, and a larger boat called Ark of the Aorta.

Since 1957 he has also been involved in microvascular studies and has developed the technique of injecting blood and lymph vessels with India ink in order to examine the routes and distributions. In fact, he became known as the Wizard of Ink or Inky for short. In 1977 he forwarded this vascular maze to me and wrote on the back of it:

This is a photograph of ink replications of arteries and veins in the vicinity of the palatal raphe that has habitually been described as being relatively avascular. The preparation is that of perinatal man.

INVESTIGATION INTO BLOOD SUPPLY TO PALATE BONES

This field has long been the realm of surgery guided largely by the surgeon's supposition. It occurred to me that further study would be of interest, not only from the aspect of safety during surgery, but also for investigating surgery’s possible effect on bone growth by reduction in blood supply. Lieutenant Colonel G. Franklin Welsh, U.S.A.F., while in general surgery residency at the Mayo Clinic, visited Miami to investigate a possible residency in plastic surgery. He was challenged to study the blood supply to the bones of the palatal area and to search for and identify blood
vessels that would demonstrate how the oral mucoperiosteum could be reflected with impunity in palate surgery without fear of devascularizing the bony palate. Welsh dissected the blood supply in both halves of a medial-sagittally sectioned head from a cadaver injected with red latex. This was his 1971 report, along with a photograph of the specimen:

Soon after its origin as a terminal branch of the maxillary artery, the descending palatine artery enters a canal in the vertical plate of the palatine bone to give off an intramedullary cascade of arterioles which meander antero-inferiorly into the bony hard palate. Several branches descend through the palatine foramina and divide into an anterior group, the greater palatine arteries, and a posterior group, the lesser palatine arteries, for soft palate. Immediately upon emerging from the greater palatine foramen, these arteries give off multiple small vessels that spread out, closely adherent to the undersurface of the bony palate, yielding several perforators into the bone, rather than lifting away with the mucoperiosteum. Specimen is shown:

Neither the posterior superior alveolar artery nor the anterior superior alveolar extension of the infraorbital artery, all of which penetrate maxillary
bone cortex externally, continues medially beyond the alveolar ridges and tooth sockets into the bony palate.

**Conclusion:** With perforating branches from nasal floor mucosa, multiple intramedullary branches from the descending palatine artery in its canal, and immediate takeoff of bony branches from the emerging greater palatine arteries, the blood supply to the bony palate is well secured even as the mucoperiosteum is lifted, even if the greater palatine artery should be ligated.

Upon receipt of this fine work, I wrote to Welsh to ask if he had also studied the vasculature coming through the incisive foramen. This was his response:

Although the conventional wisdom regarding anastomosis of posterior septal branches of sphenopalatine artery with anterior terminal branches of the greater palatine artery via incisive canal is repeated throughout the texts and atlases, I was unable to confirm this fact. By sprinting back to the gross anatomy laboratory here upon receipt of your letter, I located the same specimen on which I made the earlier observations. Attention to the incisive canals revealed first that the injected latex dwindled rapidly, requiring the final vascular arborizations to be traced via minute unfilled radicles. The course proved to be as follows:

An anterior extension of posterior septal branches of sphenopalatine artery enters the incisive canal, passes inferiorly into oral alveolar ridge cortex, and terminates in the region of the incisor tooth sockets. Although there were no apparent penetrations from incisive canal into oral mucoperiosteum, there were multiple oral mucoperiosteal vessels perforating the oral cortex of hard palate and alveolar ridge. No contributions from the artery of the incisive canal back into bony secondary hard palate were observed.

Although Welsh was unable to obtain clearance from the Air Force for a complete residency, he did accept a Maytag Fellowship and finally, in 1976, returned to Miami to put the finishing touches on his bony palate vascular research. Welsh, a Harvard Medical School graduate and pithy scholar, has a remarkable depth of knowledge and an uncanny way with words. He admits to having hyperhedonia when digging through the palate bone for shriveled vessels, defining this state as "hi par bê dô' ni a, (n), abnormal pleasure from doing ho-hum things," taken from Mrs. Byrne's Dictionary of Unusual, Obscure and Preposterous Words.
NERVE SUPPLY

Sensory

The maxillary nerve, the second division of the trigeminal nerve, passes through the sphenopalatine ganglion, picking up the secretory and sympathetic fibers from the facial nerve. This composite nerve then divides into three and is distributed to the nasal cavities, nasopharynx and palate. One of the branches is the sphenopalatine nerve, which passes through the incisive foramen to the anterior hard palate. Then there is the greater palatine nerve, which comes through the posterior palatine foramen, supplying the remainder of the hard palate. The smaller middle and posterior palatine nerves, branches of the pterygopalatine nerve, emerge through the lesser palatine foramen to supply the soft palate and tonsil with sensory branches.

Motor

It has long been known that the motor nerve supply to the tensor veli palatini muscle is different from the other velopharyngeal muscles. W. A. Turner (1889), L. Rethi (1893), L. Druner (1903) and A. R. Rich (1920) all confirmed that the tensor muscle was innervated by the trigeminal nerve, actually the internal pterygoid nerve, a branch of the mandibular, which in turn is a branch of the trigeminal nerve. It is of interest that Harvey Cushing in 1905 reported movement of the tensor palatini by stimulation of the mandibular division of the trigeminal nerve.

There has been and continues to be much disagreement concerning the motor nerve supply to the other velopharyngeal muscles. Most textbooks seem to be satisfied with the general statement that the vagus and accessory nerves supply motor fibers to the muscles of the pharynx and soft palate, with the exception of the tensor palatini, and that the greater superficial petrosal nerve, arising from the facial nerve, also carries motor fibers to the sphenopalatine ganglion and thence to the palatine nerves and soft palate.
E. Cords (1910) and I. W. Broomhead (1951) described these muscles as innervated by branches of the pharyngeal plexus derived from the glosso-pharyngeal and vagus nerves.

Ivor Broomhead was house surgeon to David Matthews in 1948 and was inspired by him in 1949 to carry out research on the nerve supply of the soft palate in the Anatomy Department at Cambridge University. He later joined Matthews at the Hospital for Sick Children, Great Ormond Street, London, and worked with him until Matthews retired in 1976. In the British Journal of Plastic Surgery, 1951, Broomhead reported important anatomical findings for the palate surgeon. He presented a sketch showing the distribution of the glosso-pharyngeal nerve (IX) and the pharyngeal branch of the vagus (X) to the constrictor muscles of the pharynx, levator palatini, palatoglossus, and the nerve to the medial pterygoid muscle. He also showed the course of the nerves to the palatoglossus and palatopharyngeus on the medial side of the superior constrictor.

A. R. Rich (1920), however, reported levator veli palatini muscle contractions elicited by stimulation of the vagus and accessory nerves, but not by facial and glosso-pharyngeal stimulation.

The continuing disagreement in the face of many studies of the motor nerve supply to the velopharyngeal muscles intrigued Juntaro Nishio of Japan. He continued his family’s tradition by taking a dental degree, then furthered his studies with a dissertation entitled “The Relationship Between Velopharyngeal Movement and Its Motor Nerves,” for which he earned his Ph.D. In 1976, further excellent work was published in the Cleft Palate Journal by Nishio, with T. Matsuya, J. Machida, and T. Miyazaki, of the Oral and Maxillofacial Departments of the Matsumoto Dental College and the Osaka University Dental School of Japan. Their experiments, designed to clarify motor nerve supply to the velopharyngeal muscles, were carried out on 20 rhesus monkeys. (J. F. Bosma and S. G. Fletcher in 1961 stated that basic velopharyngeal anatomy was similar in cats, dogs, monkeys and humans, while C. G. Hartman and W. L. Straus, also in 1961, reported that the course of the cranial nerves in the rhesus
monkey is similar to that in the human being.) Evoked EMG responses of the levator veli palatini, uvula and superior constrictor pharyngeal muscles, which contributed to velopharyngeal closure, were analyzed by the Japanese workers by their stimulating the cranial nerves within the skull. Here is the summary of their results:

1. Muscle action potential (M-waves) from the selected muscles could be recognized on stimulating the facial, glossopharyngeal, and vagus nerves at the petrosal area of the temporal bone but were not noted upon accessory nerve stimulation.

2. At maximal stimulation, the vagus gave a greater increase in muscle amplitude than the other nerves studied. This was followed by the glossopharyngeal with the facial nerve producing the least in amplitude.

3. Also at maximal stimulation, latencies in the response of the levator veli palatini and uvula muscles were reduced to the greatest degree by stimulation of the vagus, to a lesser extent for the glossopharyngeal, and least for the facial nerve.

4. On stimulating the facial nerve below the stylomastoid foramen, M-waves could not be recognized.

From the present study, it was concluded that the levator veli palatini, uvula, and superior constrictor pharyngeal muscles are double innervated by the facial nerve and branches of the pharyngeal plexus derived from the glossopharyngeal and vagus nerves and that the facial nerve plays an important role as one of the motor nerves in the movements responsible for velopharyngeal closure.

They also proposed an interesting theory:

We sometimes encounter cleft palate patients who demonstrate nasal grimace during phonation. This has been considered to be a compensation to velopharyngeal incompetence (Morley). Recently the authors have applied visual training to help cleft palate patients acquire adequate velopharyngeal function. As a result of the training, it was noted that coordinating movements of lip and face, such as nasal grimace or lip-protrusion, during phonation, were useful to activate velopharyngeal movements (Nishio, Yamaoka, Matsuya and Miyazaki). Therefore, the nasal grimace may occur not only to compensate for velopharyngeal incompetence by increasing nasal resistance, but also to fire the facial nerve to complement velopharyngeal movements.
A WARNING

The dissection of fetal and adult heads by Broomhead at the anatomy school at Cambridge has particular significance to palate surgeons, as he concluded his report with this warning:

During operative repair of a third-degree cleft palate damage may be inflicted on the nerve to the tensor palati, resulting in paralysis of this muscle.

Section of the lesser palatine nerves also takes place, and will result in some anaesthesia of the soft palate and paralysis of the musculus uvulae. Whether the mucous glands suffer any damage following the section of these nerves is not known.

The nerves to the palato-glossus, palato-pharyngeus, and levator palati muscles do not, in any part of their course, enter the operative field.
3. Growth in the Normal and the Cleft Palate Patient and the Effect of Surgery on Growth

L E S T there be any doubt as to the effect of natural growth, view for a second the comparison that Gillies and I presented in 1957 of a father and son with their noses switched to emphasize the difference. As this is just the nose tip of the iceberg, we must beware that our actions do not interrupt any essential part of the complex facial skeletal process of normal growth.

In 1778 John Hunter proposed that resorption was as determinative of bone growth as apposition. Since bone remains in a continuous state of apposition and resorption along periosteal and endosteal surfaces, the mass and shape of bones are always subject to change.

Donald H. Enlow, then (1971) of the University of Michigan and now of the University of West Virginia, summarized normal growth and development of the craniofacial complex for Cleft Lip and Palate:

Just as the mandible becomes displaced in a forward and downward manner as it actually grows in a predominantly upward and backward direction, several major growth sites in the maxilla similarly grow posteriorly and superiorly but become transposed in an opposite anterior and inferior course.

Enlow used an overlay to show familiar downward and forward manner of facial enlargement, taking the sella as a fixed landmark. The mode of growth shown represents a composite of
actual growth in addition to displacement produced by translocation of the different facial bones away from the cranial base.

An important aspect of maxillary growth—one to be aware of in considering the timing of cleft palate surgery—was established by J. C. Brash in 1924 and T. W. Todd in 1931, when they proved that five-sixths of the total maxillary width is complete by the end of the fourth year of life. In 1935 B. H. Broadbent carried out accurate measurements of changes in various components of the face by cephalometric roentgenography. In 1941 A. G. Brodie, using the same method, outlined a complete picture of cranial and facial growth from the third month to the eighth year of life, confirming that the lateral width of the maxilla is accomplished early, but pointing out that downward and forward growth is not complete until the end of the second decade of life.

In 1958 Samuel Pruzansky expressed the situation succinctly in the *American Journal of Orthodontics*:

The skull is a community of bones. Although the several bones may have diverse phylegetic origins and vary in their individual rates of growth, they are all bound together to give shape, size, and function to the community. When one part suffers in the course of growth and development, it seldom does so in isolation, for the community at large may also reflect, in one way or another, the misfortune of its member.

**THE EFFECT OF CLEFTS ON GROWTH**

As pointed out by David O. Maisels of Liverpool in his Kay-Kilner prize-winning essay in 1966, a complete alveolar cleft will be present by the end of the eighth week of gestation. According to Scott, growth of the interorbital cartilaginous system is responsible for much of the early forward and downward growth of the maxilla. Attached to the septum, the maxillae are carried with it. Latham, Burston and Sarnat have suggested that the potential spaces at the surrounding sutures are “filled in” by bone.

In complete unilateral clefts, the cleft side of the maxilla is separated from the nasal septum, sometimes leaving this lesser segment deprived of some of the usual growth impulses. Thus it may lag in development, be small and retroposed. The premaxilla
on the greater segment tends to spurt forward and rotate to the unclert side, taking the nasal tip with it, so that the septum is bent and the alar arcing over the cleft is flattened. In complete bilateral clefts, the unrestrained growth of the septum projects the premaxilla forward like a figurehead on a ship’s prow, leaving the disappointed lateral segments behind. In both unilateral and bilateral clefts there may be some slight collapse of the lateral segments at birth, which appears to increase during the next few months, even in the absence of surgery. In 1960 and 1965 orthodontist W. R. Burston of Liverpool claimed this to be more apparent than real because of differential growth rates between the maxilla and mandible. A varying degree of retrognathia is usually present at birth, and, as the mandible catches up and grows forward, it may outgrow the maxilla and give the false impression of increasing maxillary collapse.

As septal growth is maximal during the last six weeks of gestation, premature babies usually show less marked deformities than those that go to full term. After a short neonatal pause, there is another growth spurt for about six months, which accounts for the increasing deformity taking place in untreated babies before our eyes. Thereafter a fairly stable condition is reached in the upper dental arch with only minor changes in the maxillomandibular relationship.

THE EFFECT OF SPECIFIC TRAUMA ON GROWTH

Bernard G. Sarnat of Cedars-Sinai Medical Center, University of California, Los Angeles, was head of oral and maxillofacial surgery at the University of Illinois for many years. He worked with William Logan while a resident at Cook County Hospital, Chicago, and was first assistant to Vilray Blair for three years in St. Louis. He watched Blair consider Brophy’s work and eventually become disenchanted because of the poor results. This early clinical experience no doubt stimulated him to study bone growth, and his findings are important. In 1969, in Alpha Omegan, he wrote:
Primary Growth Centers

Primary endochondral centers . . . in the skull are the sphenoid and spheno-occipital synchondroses, and the septoethmoidal and septo-presphe-occipital synchondroses, and the mandibular condyle. These centers contribute to the downward and forward growth of the face. . . . The loss of anatomical continuity with changes in muscle balance must also be considered as a contributory factor. The truth of this fact was demonstrated by the severe deformity of the jaws and face that resulted after extirpation of the mandibular condyle in growing monkeys.

Secondary Growth Sites

Growth of bones is also active at secondary or accommodating growth sites. Appositional growth, as well as modeling resorption, occurs on the surfaces of bones (periosteal and endosteal) and contributes to growth in all directions. Sutural growth is only in the skull.

It was demonstrated in growing rabbits that considerable growth of bones occurred at the frontonasal suture. The nasal side contributed approximately twice the amount that the frontal side contributed. Extirpation of this suture, however, did not affect grossly the growth of the snout. Similarly in growing monkeys, extirpation of the midpalatine and transpalatine sutures resulted in no gross alterations in either facial or jaw growth.

Growth of the cartilaginous nasal septum contributes to the downward and forward growth of the face and palate and thereby influences sutural growth. The contents of certain other cavities of the skull likewise influence the growth of a complex of adjoining bones and sutures. Examples are the brain and the neurocranium, the orbit and the orbital contents, the tongue and the oral cavity. Muscle activity, both local and regional, also plays an important role.

At the International Congress of Plastic and Reconstructive Surgery in Rome, 1967, Sarnat summarized the differential effects of surgical trauma to the nasal bones and septum upon rabbit snout growth:

Although it was found that the frontonasal suture was a site of active growth, extirpation of it did not affect grossly growth of the snout. Dislocation of the cartilaginous nasal septum likewise did not affect grossly growth of the snout. In contrast however, resection of cartilaginous nasal septum produced a severe and striking growth arrest of the snout.

He presented lateral, frontal and dorsal views of rabbit #4, in which a minor amount of the nasal septum was removed, dem-
onstrating a relatively normal, long, tapered face. Similar views of rabbit #18, in which a major amount of nasal septum was removed at 21 days of age, reveals a short, stubby, rounded face with an indentation above the nostrils and an over-erupted lower incisor (from B. G. Sarnat and M. R. Wexler, *Amer. J. Anat.*, 118:755-767, 1966).

Sarnat stated in summary:

From these experiments it is concluded that the frontonasal suture is a secondary or accommodating site of growth whereas the cartilaginous nasal septum is a primary site of growth.

Here are Sarnat's 1969 thoughts on clinical application of his research:

In a child with a complete bilateral cleft palate, the upper jaw may be unable to obtain a full expression of downward and forward growth because of lack of contact of the palatal shelves with the ventral-free actively growing septovomeral region. Furthermore, trauma to the septal region, during cleft palate or septal surgery, might have an untoward effect upon growth of the nose, upper jaw, and face. Injury to the midpalatine or transpalatine sutures, which are secondary growth sites, is of less importance.

Functional and cosmetic treatment of growth deficiencies of the face is more difficult than treatment of growth excesses. . . . Even though the deformity may not be progressive, it is not self-correcting and there is no way to compensate for lost or retarded growth. Orthodontic, prosthetic and surgical procedures offer functional and cosmetic improvement. The operations commonly used are directed toward altering malposition and contributing bulk. Osteotomy with or without a bone graft and cartilage, bone, or alloplastic materials, as a masking procedure has been utilized. Certain aspects of treatment may be undertaken when the patient is still growing, but the final result cannot be attained until growth of the face has ceased.

OTHER PERTINENT ANIMAL STUDIES

As early as 1958, Sarnat reported no growth arrest in the palate or face of young monkeys that had unilateral removal of palatal mucoperiosteum or palatal mucoperiosteum and bone. Yet Herfert's work directly contradicted Sarnat's research.

Wolfgang Rosenthal founded a maxillofacial surgery hospital in a charming old castle at the village of Thalwitz near Leipzig, Germany. Here 130 primary cleft lip, 150 primary cleft palate and
350 secondary operations were carried out annually. Oskar Herfert, with both dental and medical degrees, joined Rosenthal and was stimulated by him to examine 350 postoperative cleft lip and palate cases. He discovered that patients in whom the palate was operated on between 2 and 5 years of age showed restriction of growth of the upper jaw. Sixteen had a lip closure but no palate operation until 12 years of age, and their upper jaw deformities were minimal. This finding confirmed what Rosenthal had already stated in 1927:

Operations on cleft palates of children from 2 to 4 years of age retard to a greater or lesser extent the subsequent development of the upper jaw. If such operations . . . are withheld until the child is 12 years of age, the upper jaw can develop normally and intermaxillary occlusion is more satisfactory.

Herfert was now prompted to experiment on a litter of terrier puppies. He published the results in 1954 and then again in the British Journal of Plastic Surgery in 1958. His experiment utilized an incision on the right side of the palate from the canine incisor to the second molar tooth, the raising of a mucoperiosteal flap from the bone, excision of a small strip of this flap and division of the posterior palatine artery. The narrowing of the palate on the operated side averaged 19 percent. A second series was done lifting the mucoperiosteum but not ligating the posterior palatine artery. Herfert concluded:

It can surely be stated that limitation of growth does in all cases appear to have taken place but to a greater extent in those cases in which the palatine artery was ligatured.

Herfert was director of maxillofacial surgery at Rostock on the Baltic Sea in East Germany until 1960, when for political reasons he was forced to become a refugee to West Germany and had to start all over again, eventually becoming a professor at Johannes Gutenberg University. His important early findings in dogs gave fuel to the orthodontists and even concern to some surgeons.

By 1967, confirming data had been reported by Charles R. Kremenak of the University of Iowa, who showed in puppies that the unilateral excision of a 4 mm. wide strip of mucoperiosteum just medial to the posterior teeth caused a definite decrease in palatal width (27 percent narrower on that side). Mere elevation
of a unilateral mucoperiosteal flap or ligation of the palatine artery each caused only a 3 percent narrowing of the palate. This information was sufficient to cause some surgeons to make their lateral relaxing incisions in the mucoperiosteum more medially and farther away from the teeth.

In 1977 in Toronto Kremenak noted:

We learned, after Herfert, that surgery leaving bare bone next to teeth hindered jaw growth but did not understand why. Earlier reports by Billingham, Grillo and Gross and others supplied a clue: data on contraction in healing of full thickness skin wounds resembled data on postsurgical jaw growth in our animals. Could contraction in early healing be the reason for surgical interference with jaw growth? The answer in our animals was at least a partial “yes.” Could the contraction phase of healing be prevented? . . . Majno’s group reported observations of newly recognized myofibroblasts in granulation tissue; their work and that of Wessel’s group suggested that in vivo pharmacologic regulation of contraction might be possible. Madden et al (1974) reported an animal trial; we began similar work.

Kremenak reported that immediate split-skin grafts to the denuded bone prevented much of the usual growth lag.

Yet it is somewhat difficult to evaluate dog palate experiments in relation to man. Scholarly M. J. Jurkiewicz of Emory University is one of the Pied Pipers of plastic surgery in academic medicine, drawing outstanding students into our specialty with his exciting teaching of fundamentals. While at the University of Florida, he had a mixed colony of cleft lip and palate dogs which were being used in an experiment in genetics. As he explained to me in 1972 and 1976, during operations on canine clefts—both isolated cleft palate and cleft lip and palate—he found extending forward from the greater palatine foramen the descending palatine vessels, which freely anastomosed with the anterior branches of the descending palatine vessels emerging from the incisive foramen, much as in the human. He noted important differences:

The incisive foramen in the dog is approximately three times the diameter of the greater palatine foramen. All along the canal accommodating the vessels are a number of tiny foramina which admit tiny nutrient vessels to the palate which appear to come from the bone the whole length of the palate. Basically, therefore, my impression is that there are many more lesser
palatine vessels than there are in the human, and the anterior descending palatine vessels, which emerge through the incisive canal, carry a much greater volume of blood than do the posterior vessels. I can say from experience that the standard von Langenbeck repair in complete clefts in the dog is fraught with complications in healing, often resulting in slough of the anterior portion of the flaps. I think this is because we did not pay enough attention to the contributions from the anterior vessels emerging from the incisive foramen. . . . Thus I would tend to agree with you that it would be difficult to transpose dog experiments into the human condition.

Similar difficulties have been encountered in the cleft palate of the horse.

STUDY OF THE EFFECTS OF SURGERY ON HUMAN GROWTH

For centuries surgeons obsessed with closing the hole resorted to drastic surgery on the lip and palate, eventually causing dentists faced with unbelievable dental distortion to start an anti-surgery war cry.

J. Daniel Subtelny, orthodontist and researcher in Rochester, New York, has been a leader in the attempt to get to the truth. His original work with frontal plane tomography (Subtelny, 1957; Coupe and Subtelny, 1960) added insight into the anatomy of the cleft palate beyond that presented by plaster casts. In 1962 in Plastic and Reconstructive Surgery he gave an excellent review of cleft palate studies during the previous 10 years. He first cited two monumental landmarks in the chronology of cleft palate growth studies, one by T. M. Graber in 1949, cross-sectional in nature and dealing with the past, and the other by S. Pruzansky in 1957, which was longitudinal in design and dealt with the future.

VOTES AGAINST EARLY SURGERY

Graber was one of the first to study a large number of postoperative cleft palate individuals using cephalometric x-rays to evaluate objectively the skeletal structures of the faces of the postoperative group for comparison with those of non-cleft individuals. He came to the startling conclusion that the maxillary jaw in post-
operative cleft palate cases is deficient in all dimensions—that is, in vertical and lateral, as well as anteroposterior, dimensions. The most marked reductions in the size of the maxilla were apparent where an early surgical closure of the cleft palate or a great number of surgical procedures had been performed. His findings seemed to suggest that surgical injury to growth centers of the maxilla and palatine bones was responsible for skeletal deformities. M. W. Buck of the University of Iowa confirmed Graber’s maxillary findings in 1951 and agreed with Graber that the mandible also was smaller than in normal patients. In 1954 Snodgrasse found retardation in growth, but more in the maxilla than the mandible. By 1954 Graber had reported on a larger cleft palate sample of 250 patients and strongly stressed that facial growth in unoperated cleft palate patients closely approximated that in the normal. In his view there was no real deficiency of tissue in cleft palate infants, and if no surgery was performed, they would show little or no growth disturbance. Graber took the stand that surgical closure of cleft palate should be postponed until 5 years of age, emphasizing that the maxillary dental arch had been found to be more normal in patients whose palates had been closed after 4 years. In 1954 W. Krogman advocated postponement of palate surgery to 4 to 6 years of age, justifying the delay with the fact that the major portion of maxillary width growth has been attained by 5 to 6 years of age.

Impressed with Graber’s early awareness and intrigued to know more, I wrote him in 1976 at the University of Chicago and was highly rewarded. Thomas M. Graber has four sons who are eagle scouts, and if he had done nothing else in life, he would deserve a whole sash of merit badges! Yet he has been a pioneer in the cleft field and his reminiscences are both fascinating and provocative:

I attended Washington University Dental School from 1936 to 1940 where the dental and medical schools are contiguous and there was a fair degree of interchange. Dr. Jorstad, our pathology teacher, pointed out we had one of the world’s best surgeons on our staff—Dr. Vilray Papin Blair. Almost of the same stature was Dr. James Barrett Brown. I was lucky enough to be admitted to the amphitheatre a number of times when they were operating. Dr. Blair was a great showman and made his operations “live.” Barrett,
more taciturn, was a master craftsman, and I was much impressed by the ability of these men to work in such a small field, with so many tissues, and achieve an apparent success. Naturally, I read all they had written and got hold of Dorrance's text. The controversy in the field of cleft palate rehabilitation became apparent. I did a survey article, "Cleft Palate and Hare-lip," for the Washington University Dental Journal right before I graduated, and it was apparent by then that immediate surgical success did not mean that everything was normal forevermore. Seeing a number of cleft patients in the dental clinic with tight lips, mid-face deficiencies, poor speech, high caries incidence, deformed maxillary arches and psychological problems after the holes had been closed in the face and the mouth made me wonder what was happening in the growing face, since these problems seemed to get worse as the child matured.

In World War II, I saw a number of severe facial injuries and found them depressing. . . . But the cleft children seemed different. They looked so normal after Drs. Blair, Brown and Byars were finished and seemed to grow into a deformity. I wondered if there was some possible way we dentists could guide such growth and prevent the developing facial deformities? With this in mind, I entered a graduate program in orthodontics at Northwestern University and later joined the staff at Children’s Memorial Hospital in Chicago. Dr. Louis Schultz was doing the cleft palate surgery according to the approach of Truman Brophy. You know the results as well as I—really depressing. Fred Merrifield, head of Oral Surgery at Northwestern, shared my concern. We were aware of the work of Wayne Slaughter and I had a chance to visit with him, to see his patients and to know of his concern over early traumatic surgery. Finally, Merrifield got a grant and we set up the Northwestern University Cleft Lip and Palate Institute in 1947. It was then I began my growth research and had ample cleft material both at CMH and Northwestern. Most of it was patients treated by the Brophy technique. The Blair-Brown-Byars cases had never looked that bad. Why the difference? How to prevent or correct the surgical results? I recall one case vividly. The patient was the young wife of a dentist who came to our clinic about 1952. The maxillary arch was collapsed, totally contained within the mandibular arch, and the usual midface concavity and short, tight upper lip were present. We had already spread the maxillary buccal segments in a 14 year old girl earlier in the year when the palate repair had broken down and obtained two centimeters of basal bone repositioning, in addition to moving some teeth. With a tight, unyielding and scarified palate, I saw no way of doing the same for her and timidly suggested we cut the repaired palate to enable the spread. Tears came to her eyes and she exclaimed, "You mean you actually want to open the cleft, after I have gone through so many operations to close it?" It was totally incomprehensible for her to understand our concern over jaw growth, jaw size, jaw position. The be-all and end-all for
her . . . and for so many surgeons and patients was the mechanical closure of the hole. Never mind the function, the resultant deformity and growth arrests of contiguous structures—close the hole at all costs!

With new surgical techniques, with grafting, with orthopedic procedures, things have improved. But I still see the need for better dissections of muscles, for means of relieving lip tension on the sensitive and responsive maxillary bone and teeth. Perhaps we may be able to help with some sort of plastic splints that fit in the oral vestibule and prevent lip contact with the alveolar bone and teeth—that actually stretch the soft tissue. This could be one advance for the future.

In 1954, in the *British Journal of Plastic Surgery*, A. Jolleys reported on a study of 254 children with cleft palate treated by a variety of surgical procedures at different ages. He observed a reduction in maxillary development regardless of operation or time of surgery and blamed this retardation on fibrosis. It is interesting that speech was found to be worse in the patients who had undergone surgery after 3 years of age. This finding led Jolleys to suggest that the simplest surgical procedures be used, the soft palate be closed as early as possible, leaving the hard palate till the eighteenth month of age.

At the Hamburg Congress in 1964, Longacre noted that the difference in final results depended on the time of the palatal surgery:

It is a well-known fact that the premaxilla unites with the maxilla to establish the maxillary arch between the age of four and five years. As I mentioned, we have run two parallel series using exactly the same technique. (1) An early age group before two years of age, and (2) a group at the age of four. As we have carefully analyzed these, we have noted a degree of cross-bite due to collapse of the maxillary segments in the younger group; the degree of cross-bite in the older group is definitely less. Also the degree of deformation of the anterior face and the degree of contraction of the maxilla in all three directions is definitely less.

**A Search for Unoperated Adult Clefts**

A possible control population with a potential for throwing some light on the effects of palate surgery on facial growth was, of course, unoperated cleft palate adults. Both surgeons and dentists
began scrambling around in search of these cases to help settle the argument of *just when the patient is really ready for surgery*. Yet finding unoperated adult cases was not so easy in the more advanced areas of the world for, as Claire Straith said over 25 years ago,

If I don’t operate early on this cleft baby, someone else will!

The courtly Fernando Ortiz-Monasterio, a pre-Columbian history scholar at General Hospital, Mexico City, is a skilled sailboat racer who represented Mexico in the Tokyo Olympics. In 1959, with Rebeil, Valderrama and Cruz, he reported cephalometric measurements on unoperated cleft palate adults in Mexico revealing that growth had not increased the deformity. From 1963 to 1972 Monasterio has had an unsurpassed experience of 450 late unoperated clefts, 250 of the patients being over 15 years of age. At the Cleft Palate Congress in Copenhagen in 1973 he reported the occlusion in both the unilateral and bilateral clefts to be normal, except in the area of the actual cleft. Also in 1973, he wrote:

We started 19 years ago with a very modest cleft palate clinic which has grown both in number and maturity of the members of the staff and patients. It is very large nowadays. Faced with a large number of unoperated adults we became aware (and corroborated by our cephalometric studies) that early or aggressive surgery was the main factor in growth deficiency and/or collapse of maxillary segments.

Further information on unoperated adult cleft palates was provided by the orthodontic team of J. Mestre, J. De Jesus, and J. D. Subtelny of Rochester, New York, in 1960. This is their succinct summary:

Cephalometric X-rays of forty-nine adults with unoperated clefts of the palate were compared with cephalometric records of thirty noncleft adults. The subjects ranged from fifteen to fifty-seven years of age and were located on the island of Puerto Rico. . . . The study showed that the mature skeletal relationships of the jaws did not differ significantly in the unoperated cleft palate adults when compared with the normal adults. Particularly, the dimensions of the maxilla and the positions of the maxilla within the craniofacial complex were found to be normal in the cleft palate subjects.
In 1967 Ivo Pitanguy of Rio, with T. Franco, went one step further to claim that unoperated palate clefts in his series of 84 improved with aging.

These findings of improvement in time were also observed by C. O. Innis in unoperated adult clefts of the Dusan tribes of North Borneo. He concluded that most deformities seen in the Western world in postoperative cleft patients were iatrogenic. I had noticed effects in adult unoperated clefts in Korea and Jamaica. At the Cleft Palate Congress in Copenhagen in 1973, R. J. Maneksha of Calcutta confirmed similar findings in his unoperated adult cleft Indian population. In 1974 Ralph Blocksmna recalled:

The consistent excellence in facial development of individuals with unoperated clefts which [I] observed in Pakistan 30 years ago . . . [confirmed] the basic truth that oral-facial development in unoperated oral cleft individuals proceeds generally in a normal way.

In 1972 Frank McDowell threw us a provocative curve by adding another dimension:

Having observed a considerable number of patients with wide single clefts and total double clefts who have grown up without surgery of any kind, I saw all sorts of distortions which came late in the growth of these unoperated patients. I am sure that the problem of lip repair is not alone the simple mechanical immediate restoration of contour in the very young infant, but we have to deal with all the distortions that would have developed anyway if the patient had never been operated on, plus the influences exerted on these distortions by every scar produced in the lip, nose, cheek or palate. If all these factors were ever programmed on a computer, and someone pushed the answer button, it would probably blow all the fuses.

In a slightly less dramatic presentation, the maxillary distortions of the unoperated cleft were confirmed in 1977 in Toronto by S. Bishara, W. Olin and C. Krause of the University of Iowa, when they clinically and cephalometrically compared dentofacial relations of 8 unoperated clefts of the lip and alveolus, 12 unoperated clefts of the lip and palate and 20 normal individuals matched for age, sex and ethnic background. Their findings suggested that different cleft types have different clinical, dental and cephalometric characteristics. They reported:
Some of the significant findings include a relative maxillary skeletal protru-
sion in the lip/alveolus group while the lip/palate group indicated a
relatively steep mandibular plane and more upright lower incisors.

INDIVIDUAL VARIATION

As condemnation of early surgery was gaining momentum, another controversial figure, in the form of Samuel Pruzansky, loomed on the scene. At first he appeared arrogant, impudent, and as irritating as a picador. Yet as the inflammation settled, it became evident that his confidence came from having done his “homework” and his sharp tongue was not attacking any specific group; he was only in search of the truth. As he said:

My early reading was from Fogh-Andersen and Victor Veau; their writings and conceptual approach affected me most of all.

So when others were condemning all palatal surgery, Pruzansky, from his longitudinal studies, presented conflicting opinions. As he explained:

When the longitudinal growth study of children with cleft lip and palate was initiated at the University of Illinois in 1949, it was our expectation that the collection of casts, cephalometric radiographs, photos, family and medical history, and other related data would describe and measure the initial state of the unoperated infant, document the manoeuvre in the form of surgical or other treatment, and the subsequent state through long term follow-up.

Whereas maxillary deficiency was being found in some post-
operative cleft palate patients, there were others who were growing normally. As Pruzansky pointed out in 1954,

The child with a cleft palate is first of all a child. As such, he is endowed with inherent potentialities for growth and development that reflect his genetic heritage and the metabolic climate in which he thrives.

In other words, some cleft palate patients have a potential for attaining a favorable facial appearance while others, from birth, do not. In fact, in 1954 W. B. Slaughter, plastic surgeon, and S. Pruzansky noted that surgery could actually aid and direct natural development processes through the reestablishment of more normal muscle forces. This finding caused greater attention to be
directed toward the actual surgery. Many of the cases which had caused so much concern over maxillary growth deficiency had been submitted to Brophy’s surgical maneuvers, using constricting wires to reduce a cleft mechanically by forcing the bony segments of the maxillary jaw together!

Egil Harvold of the University of Oslo and the Norwegian Dental School also resisted the stand that palatal surgery before 5 years of age inevitably leads to facial disfigurement. In the 67 postoperative cleft palate patients he studied in 1954, the deformities were not necessarily the results of reduced growth potentials. The change in the position of the separated maxillary jaw parts, he noted, can cause maxillary constriction, and deformities in the nasal septum and premaxilla are evident in fetal life. Harvold did admit:

It cannot be denied that the greatest deformities in the lateral segments of the alveolar process arise where surgical treatment has left abundant scar tissue, while the symmetry aberrations and the deformities are relatively limited where the palate has not been operated upon. It is also apparent that orthopaedic treatment may result in almost normal development in this area when it aims to eliminate the unfortunate influence of scar tissue formation in the palate and attempts to establish the most nearly normal eruptive conditions for the permanent teeth in the lateral segments.

In 1973 prosthodontist T. Ramstad of the University of Oslo, Norway, noted:

Loennecken’s introduction of improved surgery [trained by Gillies] represented a milestone in cleft palate treatment in the Oslo area, and his conservative procedure led to a marked improvement in maxillary development.

In 1956 L. T. Swanson, D. W. MacCollum and S. O. Richardson studied more than 100 children with clefts of the palate surgically closed prior to 2 years of age. The skeletal profile of the face was compared to an “ideal” concept and an average profile attained from a random sample. The cleft children did not conform to the “ideal” but were closely related to the normals selected at random. These palates had been closed gently by the MacCollum-type Langenbeck and were considered good results, not justifying delay of surgery to 4 to 7 years of age.
In 1958, in the *American Journal of Surgery*, Richard Webster, Lawrence Quigley, Richard Coffey, Robert Querze and James Russell of Brookline, Massachusetts, proposed pharyngeal staphylorraphy and speech aid as a means of avoiding maxillofacial growth abnormalities in patients with cleft palate and concluded:

We plan to delay surgical closure of the hard palate clefts until the child is between the ages of five and eight, or even longer, unless complications unknown to us at present arise.

Pruzansky has demonstrated differences in growth changes in many cleft palate patients even prior to palatal surgery; some clefts narrow with age and others do not. This may be especially the case after lip cleft closure but the same discrepancy has been observed in posterior clefts with no involvement of the lip or alveolus. The palatal shelves may be growing more in some clefts than in others. From serially accumulated plaster cast reproductions of the face and jaws of newborn infants with cleft lip and palate, Pruzansky deduced:

Cleft lip and palate do not represent a single fixed clinical entity subject to generalizations of description and classification and, least of all, rigid therapeutic formulae.

These factors he does consider important:

1. Extensiveness and width of the cleft.
2. Adequacy of parts and amount of deficiency.
3. Evaluation of cleft segment misplacement and/or distortion.

In 1960 T. B. Coupe and J. D. Subtelny studied 127 cleft palate children under 3 years of age with cephalometric laminography and found:

There was a definite tendency toward a deficiency of hard palate tissue in all types of clefts of the palate. Of course, individual variation was noted. . . . The bilateral cleft palate subjects tended to show the greatest amount of tissue deficiency . . . the greatest amount of displacement of the maxillary bones. . . . The posterior cleft palate cases were observed to have a greater degree of tissue deficiency than the unilateral cleft cases, while the unilateral
cleft cases showed a slightly greater amount of tissue displacement than the posterior cleft cases. Therefore, one of the very basic differences between cleft palate and non-cleft palate subjects is to be found in the quantity and spatial position of hard palate tissue.

Morphological differences in other skeletal areas have also been found. In 1956 M. L. Moss noted possible malformations of the base of the cranium in the cleft palate individuals, and in 1954 R. M. Ricketts noted that cleft palate cases may show some deviation in the base of the skull. In 1955 Subtelny, for example, observed that the hamular processes of the medial pterygoid plates of the sphenoid bone are farther apart in unoperated cleft palate children than in non-cleft children, indicating an abnormally wide nasopharynx in cleft palate cases. G. H. Borden of the University of Illinois studied mandibular growth in cleft palate subjects and in 1953 noted that the rate of growth in the cleft palate group was slightly below that in the non-cleft group. Pruzansky emphasized the importance of mandibular growth in bilateral clefts of the palate. Following surgical resection of a part of the vomer and premaxillary setback, it was often observed that the premaxilla did not follow the downward and forward growth of the maxilla, and gross facial disfigurement ensued. Yet in many patients with a projecting premaxilla facial appearance improved with growth after surgical resection of a portion of the vomer. It was demonstrated that the downward and forward growth of the mandible, as well as the maxilla, permitted these structures to catch up with the premaxilla, which seemed to be held in place by the tension of the closed lip. In some children this improvement in facial appearance occurred quickly, with rapid growth of jaw structures; in others growth and facial improvement progressed more slowly.

At this time in the evolution of palate surgery, the death knell had been sounded for early traumatic surgical methods, while sound, gentle procedures promised to correct anatomy, improve physiology and, in fact, encourage and direct growth.

In 1972 Toshiki Minaba of the Tokyo Dental College summarized his growth studies using lateral roentgenographic cephalograms of 291 cleft lip and palate patients and 160 normals,
concluding that facial growth is retarded in all cleft groups. He noted several points specifically. The forward growth of the lower part of the orbits in cleft lip and palate and cleft palate groups is inferior to that of the normal. Forward and downward growth of the maxilla in all cleft groups is inferior to that in the normal but is more inferior in the cleft lip and palate group than in the cleft palate group. Minaba also recorded that downward growth of the posterior part of the mandible in both cleft lip and palate and cleft palate groups is slightly less than in the normal. The mandibular plane angle, gonial angle and ramus angle are larger in cleft lip and palate and cleft palate groups than in the normal, but the mental angle is smaller, and all become more remarkable with advancing age. Finally, labial inclination of the upper incisors and lingual inclination of the lower incisors is greater in cleft lip and palate and cleft palate groups than in the normal.

His conclusion:

It seems that repairs of both cleft lip and palate affect the growth of the maxilla. Consequently, tight lip must not be reconstructed in cleft lip repair; and also techniques involving comparatively little surgical invasion ought to be adapted to cleft palate repair.

Kenneth L. Pickrell, with E. Clifford, G. Quinn and R. Massengill, reported in 1972 on 100 cleft lip and palate patients operated on by him 22 to 27 years previously, using the Wardill palatoplasty at about 18 months:

There was maxillary collapse in all instances in which the cleft involved the alveolus and the maxilla.

In 1972 Crikelair, Price and Cosman divided maxillary deformities in postoperative cleft lip and palate patients into four main categories:

1. Medial collapse of the cleft segment of the alveolus with eventual crossbite.
2. Anterior-posterior shortening of the maxilla with its retrognathism.
3. Decrease in inferior-superior height of the maxilla on the cleft side with tilting up of the cleft segment off the plane of occlusion.
4. Buckling inward, or hourglass deformity, of the lateral portions of the alveolar arch.

**Hourglass Maxillary Collapse**

The "buckling collapse" deformity was first noted by T. D. Foster of Stoke Mandeville, England, in 1962. It appeared postoperatively in 11 of 102 complete unilateral cleft lip and palate cases, 4 of 47 bilateral complete clefts, and 7 of 19 postalveolar cleft palates without cleft lip. Foster made another important observation: In the unilateral cleft lip and palate case the buckling was bilateral and essentially equal on both the cleft and non-cleft segment. In 1972 Crikelair, Price and Cosman presented two examples of this deformity which had occurred among the postalveolar clefts operated on at Columbia Presbyterian Hospital between 1958 and 1968. They entitled the deformity "hourglass maxillary collapse." One case was a standard postalveolar cleft treated by the routine von Langenbeck procedure which was followed with uneventful healing. At the age of 10 years, the child presented an hourglass collapse without anterior-posterior shortening. In the second case the operation was done at 4 years with a pushback procedure to close the anterior portion of a wide, horseshoe-shaped postalveolar cleft. At age 6 a von Langenbeck procedure closed the posterior portion of the cleft. Turnover flaps were used to close fistulae at ages 8 and 9. At age 21, dental models presented the hourglass maxillary collapse. Crikelair, Price and Cosman made pertinent comments about this deformity:

It is the only form of alveolar arch deformity found in the alveolar cleft palate patient. The defect is clearly related to surgery. The mechanism of its cause is uncertain, but may be a denuding injury to the palatal bone shelf immediately adjacent to the tooth line rather than operative site scar contracture. Elimination of releasing incisions or their placement further away from the alveolar ridge is suggested as a potential means of preventing this form of maxillary collapse.

It would seem likely that at least isolated palatal clefts with the anterior strut of an intact dental arch would not be affected by early elevation of mucoperiosteal flaps. Yet R. Hellquist, B.
Pontén and T. Skoog reported in 1978 on 99 isolated clefts operated by a V-Y or Dorrance-type pushback procedure at the age of 18–24 months. At the age of five years the frequency of anterior crossbite was 38 percent in patients with large palatal clefts, compared with 19–21 percent in patients with smaller clefts. In boys with cleft palates the frequency of anterior crossbite was 13 percent higher than in girls, in spite of the fact that the incidence of large palatal clefts was lower in boys. In cases of large clefts of the secondary palate, the incidence of anterior crossbite was 12.5 times higher than in noncleft patients of the same age.

In 1973 at the European Orthodontic Society meeting S. Pruzansky, with H. Aduss, S. Berkowitz, H. Friede and K. Ohyama, summarized the progress of their longitudinal growth studies. Their 4,000-case survey has provided a view of the wide spectrum of variation encountered in each cleft type in the unoperated state and the changes due to growth or specific therapeutic maneuvers. They noted:

As our observations expanded, it appeared that, 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. . . . In studying the effect of lip repair on the facial profile in complete bilateral cleft lip and palate, Friede and Pruzansky (1972) found that the following two factors characterizing the patient were more predictive of the ultimate result than who did the surgery:

1. The amount the premaxilla projected in the initial state was found to vary by a multiple of two. Thus, the patient whose premaxilla projected the least in the unoperated state presented the better profile at the earliest age, while those which projected most exhibited the poorest results, irrespective of who did the surgery.

2. The pattern of mandibular growth was a significant independent variable in determining the ultimate cosmetic improvement in the facial profile. A prognathic mandible could mask the characteristic midface convexity, while a retrognathic lower jaw would only accentuate the premaxillary protrusion. . . . Given two surgeons of similar competence and utilizing procedures that did not vary greatly in principle, the ultimate success or failure was less dependent on differences between them than on the variables within the patient.

In their study of the initial state, it was apparent that clefts cannot be lumped together, as noted in 1972 by A. Burdi, M.
Feingold, K. S. Larsson, I. Leek, E. F. Zimmerman and F. C. Fraser. Long conscious of this principle, Sam Pruzansky, in 1953, as the first published sentence to emerge from his research, stated:

Not all congenital clefts of the lip and palate are alike.

When asked by a National Institutes of Health site visitor what he considered his most important contribution to the cleft palate literature, he responded that he had never been able to exceed the profundity of his first sentence. Of course, he elaborated on the unpredictability of clefts when he stated in 1958:

Recognizing that certain congenital deformities will show spontaneous improvement in time, while others will remain the same and some will grow worse, is of practical value.

And later,

The same funny-looking kid looks funny for different reasons at different times.

Pruzansky is concerned with the many factors that are involved. Is there an adequacy of parts, the cleft being merely a non-union of normal parts, or is there an intrinsic inadequacy? How much distortion is present? Then there is the geometric relationship of contiguous anatomical structures. The status of the mandible, posture of the tongue, anomalies of the skull base and upper cervical column and of the anatomy of the nasal cavity—all have their influence on the final result.

These longitudinal studies have provided crucial information on craniofacial growth in children with clefts and have demonstrated that current surgical practice which does not resort to presurgical maxillary orthopedics and primary bone grafting can produce satisfactory results without interfering with growth (Aduss, 1971), contrary to reports in the 1940's of the deleterious effects of surgery on the growth of the mid-face.

Their analysis of the initial state suggested that under certain conditions surgical repair of the palate is feasible quite early, while in other instances, optimal conditions for repair will not become evident until a later age. In our experience a selected number of cases underwent palatal repair at or before one year of age without detriment to
midface growth. . . Age at surgery is not a primary variable in determining the effect on facial growth. Quantitative and qualitative characteristics of the defect, general health and genotype of the individual are determining factors.

Pruzansky concluded with this statement, having already made it clear that he stood strongly in camp 2:

It would seem that we [the cleft palate workers] are still divided into two camps: (1) Those who believe that cheiloplasty should be supported by pre- and post-surgical maxillary orthopedics with or without primary bone grafting; (2) those who hold that the effectiveness and benefits of such procedures are limited and the costs incurred are inordinately high for the value gained.

In 1975 S. Pruzansky and Hans Friede came upon some evidence to help confirm their general position. Two sisters, daughters of migrant workers, were reported with unoperated bilateral cleft lip and palate. They were 5 years 8 months, and 3 years 11 months of age and both revealed a degree of premaxillary protrusion similar to that in unoperated infants.

Pruzansky noted on the other hand,

Children operated in infancy showed less midfacial protrusion than the sisters following repair of their lips at a later age suggesting that the repaired lip has a long-acting effect in restricting growth of the premaxillary-vomerine complex. Later, forward growth of the mandible and elongation of the face also serve to minimise the convexity due to the projecting premaxilla.

About a year ago I asked Sam Pruzansky for an up-to-date summary of his feelings on cleft palate. After scanning Volume I of Cleft Craft, he hastened to respond in a manner that well portrays the man and his stand:

Cleft Craft is a scholarly tome and also a happy by-product of the increasing communication between scientists and clinicians throughout the world that is dissolving the provincialism that prevailed when I first became interested in clefts in 1949.

Over the years, I developed a central hypothesis that unifies much of our research. It is at the opposite pole to your own thinking which quite properly emphasizes the craft, even though you recognize that the final result “must depend upon the sculptor and his clay.” My mission is to analyze the clay.
As Feinstein stated, there are 3 elements in the architecture of clinical research design:


I know of no satisfactory way to control and document the manoeuvre. Therefore, there evolved the hypothesis that the initial state (the clay) is the precondition that determines the subsequent state, given a cadre of plastic surgeons of nearly equivalent competence and utilizing similar principles.

Toward that end, a combined prospective and retrospective study has been undertaken to analyze the complete unilateral cleft lip and palate, paying attention to some of the variables that Pfeiffer first discussed.

As you noted on pp. 20-23 of Cleft Craft, there are a number of variables, inconstant in their severity, that characterize the complete unilateral cleft lip nose. Some of these variables I have measured and reported in collaboration with H. Aduss. I should like to elaborate on your list and add some additional variables:


2. The turbinates on the cleft side vary in size, shape and position and have an influence on subsequent arch form in the complete unilateral cleft lip and palate.

3. The nasal floor is affected by the variable inclination of the palatal shelves. The palatal shelf on the cleft side may be superior, at the same level, or inferior to the non-cleft shelf.

4. The philtrum is variable and in a few instances, the eminence on the cleft side is absent or poorly defined.

5. The relationship of the alveolar process to the lip is variable. In some, the alveolus protrudes between the cleft in the lip and overrides the lip on the cleft side. In others it is concealed by the labial elements except when the infant cries.

6. The extension of the vermilion varies.

7. The number, size and position of the lateral incisors adjacent to the cleft varies, as shown by Dickson (1966) and confirmed by Lauterstein and myself (Teratology. 1969). This tells us something about mesodermal adequacy and is a factor in determining the arch form.

8. In examining infants, casts and observing in surgery, I became impressed that there are aberrant insertions of labial musculature or fibers onto the nasal aspect of the maxilla which may contribute to the rotation and architectural configuration of the total complex. In a sense, my observations parallel what you wrote about the vestibular lining, except that I tried to explain the dynamics in terms of muscular tensions on developing unbuttressed structures.
Finally, I must add one other item to the check list by quoting the title from an article in preparation, "Time: The Fourth Dimension in Syndrome Analysis." All of these variables are subject to change with increasing age. A static view of deformities leads to poorly timed and sometimes unnecessary surgery. As examples, many VSD defects of the heart tend to close and the micrognathia of the Robin anomalad diminishes in severity as the child grows older.

NO. 1 PRESENT DILEMMA

An extremely difficult task which is involving the concentrated efforts of a multitude of measuring researchers, both orthodontists and surgeons, continues to be that of carefully estimating and honestly evaluating whether the final discrepancies seen in clefts are inherent or induced or both. If induced, it is important to determine whether the discrepancies were caused by the injury of surgery followed by fibrosis, or by the timing of the injury and its effect on growth or both.

NASOPHARYNGEAL AND SOFT PALATE GROWTH

Growth and development in the nasopharyngeal area are important. In 1952 E. W. King demonstrated that after 2 years of age a remarkably stable relationship exists between the posterior nasal spine of the hard palate and the anterior arch of the first cervical vertebra. He concluded that with growth there is little perceptible increase in depth of the skeletal pharynx. The forward growth of the anterior arch of the atlas seems to prevent any appreciable increase in depth. The vertical dimension of the nasopharynx normally continues to increase until 17 to 18 years of age, when the maxilla itself completes its growth. As the head grows, the hard palate moves away from the base of the skull in a gradual and parallel manner. Both nasal and nasopharyngeal height increase as a result of this descent of the hard palate. Thus the floor of the nasopharynx, the soft palate, by virtue of its attachment to the posterior border of the hard palate, is also being carried to lower levels in relation to the base of the skull.
Growth of the upper face results in a constantly changing distance between the soft palate and the soft tissues of the superior and posterior aspects of the nasopharynx. From infancy to early adulthood, Subtelny found, there is an increase in the depth of the "soft tissue" nasopharynx, the dimension between the posterior border of the hard palate and the soft tissue of the posterior pharyngeal wall. The descent of the palate serves to increase the anteroposterior depth of the soft tissue nasopharynx with a concomitant increase in the vertical height of the nasopharynx.

In the normal child, the growth in length of the soft palate was most rapid between 3 months and 2 years of age, after which minimal growth was apparent until 4 to 5 years. At that point a slow and steady growth increase was noted up to late adolescence or early adulthood.

P. J. Coccaro at the University of Rochester, from records obtained from the cleft palate center of the University of Illinois, found growth patterns of the soft palate for cleft palate children quite similar to those of normal individuals. The cleft patients were observed to have somewhat shorter soft palates. It was also noted that operated soft palates grow in length as do non-operated ones.

**ADENOID TISSUE**

The comparatively shorter soft palate length with the comparatively greater dimension through which it must move points to the importance of the projection of adenoid tissue.

In 1964 J. M. Tanner, with his source of reference the work of R. E. Scammon, J. A. Harris and C. M. Jackson et al. (1930), wrote:

The lymphoid tissue of the tonsils, adenoids, appendix, intestine, and spleen, has quite another growth curve. It reaches the maximum amount before adolescence, and then, probably under direct influence of the sex hormones, declines to its adult value.

Although in general this, for the most part, is the case, there are exceptions. In 1975 Pruzansky, with cephaloroentgenographic studies of tonsils and adenoids, attacked the statement. He found
considerable variation in adenoid size in all age groups. Some children exhibited sparse development of adenoid tissue, and in some the size of the tonsils did not necessarily mimic the size of the adenoid tissue.

In 1956, using lateral cephalometric x-rays, J. D. Subtelny and H. Koepp-Baker studied the specific cycle of growth of nasopharyngeal adenoid tissue. Early rapid growth of adenoid tissue fills as much as one-half of the nasopharyngeal cavity by 2 to 3 years of age. After 2 years of age the adenoid tissue continues to grow, still in a downward direction but at a slower rate, until its peak of growth is reached at 9 to 11 years. Evidently this peak can be reached as late as 14 to 15 years. Then the adenoid tissue commences to atrophy. By adulthood it has atrophied completely and, with maxillary growth at an end, the greatest dimension between the superior surface of the soft palate and the superior and/or posterior wall of the nasopharynx is established.

In children the soft palate was observed to move upward and backward, with velopharyngeal closure occurring against the adenoid tissue. Following adenoidecotoxy, greater muscular activity on the part of the soft palate was necessary for velopharyngeal closure. Not all normal palates can adjust to this loss of cushion; certainly postoperative cleft palate patients can be affected adversely. Thus it becomes obvious that adenoid tissue should not be removed routinely without good cause in the cleft palate case.

Although many cleft palate patients are known to be able to accommodate to the gradual growth and abrupt surgical changes, unfortunately, not all are able to do so. While the level of velopharyngeal closure was found to be closely related to the level of the hard palate at all ages, the soft palate usually contacted the superior aspect of the nasopharynx or adenoid tissue in the younger ages and the posterior pharyngeal wall in the older groups. As noted by Subtelny:

The changes in site of velopharyngeal closure with growth helps to demonstrate why some youngsters could have good speech at one age and poor speech at an older age. In the younger group, a soft palate that is limited in activity could contact adenoid tissue since it is closely related to this
contiguous structure. With growth, the same soft palate may not be able to move adequately to contact the posterior pharyngeal wall.

It can be simply said that no congenital cleft of the palate is exactly like another; surgical treatment varies according to the surgeon, the technique and the timing of the surgery, but all these factors have some influence on the growth and development of the affected parts. Our goal is to find a plan of treatment that will have the least deleterious effects on growth with the best effects on development toward normal appearance and function.
4. Cooperation of Plastic and Dental Teams is Vital

There have been many times in the history of plastic surgery when the dentist was a most important member of the team.

If we allegorize cleft surgeons as the law-and-order good guys fighting with principles against the bad guys, primary congenital and secondary surgical deformities, then we can flash back to Tombstone, Arizona, October 26, 1881.

At half past two in the afternoon, the Clancy brothers, the McLowrys and a tough young killer, Billy Claiborne, waited in the shadows of the fence corral. The three Earp brothers, Virgil, Morgan and Wyatt, came onto Fremont Street heading for the O.K. Corral. Each wore a black stetson and under his single-breasted black frock coat carried two Colt revolvers. Virgil also had a shotgun. On they came, the three against the five. Suddenly, a man in a flapping gray coat and carrying a cane dashed out toward them from the sidewalk. It was dentist Doc Holliday.

"Where are you fellers going?" he asked.
"To a fight," said Wyatt.
"Fine," Doc replied, "I'm in it too!"

He dropped his cane to catch the shotgun Virgil tossed to him. As they entered the corral, Marshal Earp ordered:

"You're under arrest. Up with your hands!" and the guns blazed. When the smoke had cleared, three of the outlaws lay dead and two were on the run. Only two of the Earps had flesh wounds. Doc Holliday had evened the odds and given a good account of himself that day. Gillies' Doc Holliday during World
War I was Kelsey Fry, while mine in the cleft campaign is Sam Berkowitz.

Berkowitz received his dental degree at New York University and his orthodontic training at The University of Illinois Dental School where he studied with Sam Pruzansky for two years prior to coming to Miami. He is a scholar, artist and dedicated researcher. It is not an exaggeration to say that Sam has strong opinions, does not take kindly to argument against his beliefs and, although beginning to mellow with the years, still can be stimulated to a purple color and some ranting and raving if we insist on an immediate ideal result rather than the long-term plan. He has made original contributions to his field. As stated by his mentor, Pruzansky:

Three dimensional analysis of casts utilizing stereophotogrammetry being carried out by Berkowitz (1968, 1971) provides the most sophisticated approach currently available to measure the architecture of the palate. Studies now being completed by Berkowitz et al . . . may lead to a subclassification of cleft types based on cluster analysis of architectural characteristics of the cleft palate cast.

His dedicated record taking and longitudinal study of our cases has had a tremendous influence on diagnosis and treatment of our clefts. It is important that he have his say, now:
One's enthusiasm for a particular form of therapy should not be regarded as scientifically established when, in fact, it may not have stood up to critical scientific analysis. Treatment fads come and go; unfortunately, when they involve cleft palate it takes at least a decade to determine their effectiveness. A prior belief in a particular therapy often determines the biased selection of evidence to support a concept currently in vogue, and some clinicians show a select sample of cases to prove the theory only in part, if at all.

This chapter presents my understanding of the cleft defect and the face in which it exists. It is designed to answer some basic questions previously posed by Drs. Pruzansky and Subtelny. What is the natural history of the cleft defect? How do similarly classified clefts differ from one another? What should be done for a child with a cleft lip and palate, and when should it be done? And finally, how does the treatment vary from child to child? Although selected cases will be shown to develop our treatment philosophy, it must be stressed at the start that the concepts being presented are supported by the findings from many longitudinal facial growth studies already published. The cases offered here are somewhat unique in that they represent the results of only one surgeon who has modified his concepts according to the critical review of the results achieved. Also, no neonatal maxillary orthopedics has ever been done.

Treatment failures as well as successes are presented to develop and stress the physiological principles which are the basis of our treatment philosophies.

**DIAGNOSTIC RECORDS**

Longitudinal facial growth records such as cephaloroentgenographs, casts of the palate and facial photographs starting at the newborn period demonstrate how surgery may influence the direction and degree of palatal development. This knowledge helps explain how different results can occur even when similar surgical techniques are being employed. It exposes the biological mechanism which ultimately determines the success or failure of reconstructive surgical procedures. This information does not diminish the importance of surgical technical skill but does highlight the many other

81
factors that warrant consideration in planning treatment. It permits the clinician to appreciate what great allies or adversaries he has in time and growth.

Serial cephaloroentgenographs and casts expose the rate and degree to which a face and palate can improve or worsen with time. The face with a complete bilateral cleft lip and palate in this case improved as the profile became flatter.

GLOSSARY OF DENTAL TERMS

Classification of the anterior (incisal) and posterior (buccal) occlusion is based on the interrelationship of the teeth of one arch with those of the other arch. Malocclusion may be present unilaterally or bilaterally. Although there are three basic classes of malocclusion, there is a great variety of dental relationships within each type. For example, a class II malocclusion can have an anterior open bite and displaced teeth as well.

Neutroclusion (class I)

The maxillary canine (cuspid) occludes between the mandibular canine (cuspid) and first deciduous molar or first premolar (bicuspoid), and all the other teeth occlude in their proper interrelationship.
**Distoclusion** (class II)

The maxillary cuspids are forward of their ideal position as seen in neutralocclusion. The posterior teeth are out of alignment in the same direction as well. Distoclusion is often associated with severe maxillary anterior overjet and/or overbite.

**Mesioclusion** (class III)

This is the reverse of distoclusion. The maxillary buccal teeth are posterior to their ideal position with the mandibular teeth. The anterior teeth are usually in crossbite.

**Open Bite**

The teeth of opposing arches do not meet. This may occur in either the anterior or posterior portions of the arch and may be due to orofacial habits or skeletal dysplasia.

**Crossbite**

Crossbite may be due to malposition of the teeth alone and/or be coupled with bony displacement. Buccal crossbite in cleft palate is often caused by medial displacement of the lesser palatal segment. Anterior crossbite may be the result of dental dysplasia, mandibular prognathism and/or maxillary hypoplasia.

**Pseudo class III** (associated with anterior crossbite)

A bilateral cleft lip and palate in Class II dental relationship. Note that the buccal teeth of the upper arch are forward of their proper position when related to the lower teeth.

Class III malocclusion with buccal and anterior crossbite

Left buccal crossbite due to medial bony displacement

Class I malocclusion with open bite

Class III mesioclusion
This state exists when the mandible is of normal size and in normal relationship with the maxilla but the anterior teeth are still in crossbite. It may be due to the *retrusion* of the maxillary teeth, to premaxillary and/or maxillary hypoplasia or to the forward *posturing* of the mandible (false prognathism) which places the lower anterior teeth forward of the maxillary teeth when in occlusion.

*Dental dysplasia*

Individual teeth are malpositioned within each arch.

*Skeletal dysplasia*

Prognathism with class III malocclusion

Retrognathism with class II malocclusion

Prognathia is a condition in which the mandible is located anteriorly within the skull or is enlarged. It is associated with class III malocclusion. The maxilla may be posteriorly positioned within the skull or be underdeveloped as well.

Retrognathia is due to a small mandible (micrognathia) or to a posteriorly located normal sized mandible. It is associated with class II malocclusion. In some cases a relatively small mandible can coexist with a forward-positioned maxillary complex.

**Is the Neonatal Unoperated Cleft Lip and Palate Deficient in Mass and/or Displaced in Space?**

While Stark stated in 1958 that all cleft palates show mesodermal deficiency, and therefore adult cleft palates are destined to be small in size, Graber (1954) reported that growth deficiencies in all dimensions were due to early closure and to many surgical procedures. Pruzansky (1956), Slaughter, Pruzansky and Harris (1956), and Mestre et al. (1960) wrote that each cleft...
palate patient had the genetic potential for adequate facial growth. Coupe and Subtelny (1960) demonstrated that tissue deficiency can exist in variable degrees according to cleft type. The complete bilateral clefts of the palate had the greatest degree of tissue deficiency and lateral displacement of the palatal shelves. My own findings suggest that the neonatal cleft palate may be smaller in size than a normal palate of similar age, but it has the potential of reaching normal size at a later age. Also, in complete clefts the palatal segments are displaced from their normal position because of the pull of aberrant external muscular forces and the action of the tongue within the cleft pushing the segments farther apart.

The composite of normal and complete unilateral cleft lip and palate casts shown here graphically portrays the lateral displacement of the palatal shelves as a result of clefting and uncontrolled muscular systems.

**Influence of Aberrant Muscle Forces on Palatal Segments—Displacement of Tissue**

Cleft of lip and alveolus

Complete unilateral cleft of lip and palate

Complete bilateral cleft of lip and palate (Slaughter et al., 1958)
In the molding of the facial skeleton there are complex muscular systems which influence the spatial relationships of the facial bones. The superior constrictor of the pharynx, the buccinator and the orbicularis oris muscle complex are largely responsible for molding the dental arch and opposing the expansile forces of the tongue. Muscle function is initiated prior to complete ossification of the skeleton and may manifest itself as early as the second month of life.

Since development of the palate involves two separate embryonic processes—the lip and alveolus derive from one, and the palate posterior to the incisal canal from another—it is possible to have any number of variations in degree and locations of clefting.

**Incomplete clefts of lip and palate**

Any soft tissue bridge (Simonart's band) across the cleft will prevent the aberrant muscle systems from being really effective in distorting the palatal segments. For this reason it is inadvisable to lump together all types of clefts when reporting on surgical results and planning treatment procedures.

**Complete clefts of lip and palate**

When there is a complete cleft through the lip, alveolus and hard and soft palate, continuity of the outer muscular ring and the underlying skeletal structures is lost. Thereafter, the musculature of the cleft lip develops and functions around an aberrant skeletal foundation. The cleft musculature acts in a disproportionate and possibly asymmetrical fashion on the unfused maxillary structures and is unable to restrain the expansile forces of the muscular tongue.

**Complete unilateral cleft lip and palate**

In complete unilateral clefts of the lip and palate there is an anterolateral displacement of the non-cleft segment with an outward and lateral rotation of the premaxillary area adjacent to the cleft.

In the complete bilateral cleft of the lip and palate, this disturbance of normal muscular balance results in extreme forward projection of the premaxilla in relation to the facial complex. The projection is due to excessive growth at the premaxillary vomerine suture which occurs in an
Complete bilateral cleft lip and palate—original distortion corrected by molding action

Both palatal segments with their connecting perpendicular plates of the sphenoid have been displaced laterally as a result of the aberrant muscular forces. As shown in this basilar view, closing the lip brings the palatal segments, with their respective pteryoid plates, together. (Subtelny, 1955)

Environment of abnormal force fields resulting from a release of lip muscular restraint. The patient whose premaxilla projects least at birth often has a better profile at the earliest age. A severely projecting premaxilla will yield the poorest immediate results irrespective of who does the surgery. This comment should not be interpreted to mean that a severely protruding premaxilla at birth is to be surgically retropositioned.

Not all palates within the same cleft type are alike. Since a single cleft type exhibits many variations, no rigid formula of treatment applicable to all clefts should be expected. Several kinds of isolated cleft palate are illustrated. The cleft may extend in varying degrees as far forward as the nasal palatine foramen, and in its lateral dimensions the cleft may be wide or narrow. Each case poses different surgical problems and therefore may influence the timing for surgical repair of cleft palate.

Clefts of hard and/or soft palate

Isolated clefts of the palate can vary in shape and extent from a slight visible notching of the hard palate to cleft extending up to the incisal canal. There may be a cleft of the uvula alone or it may include the entire soft palate without involving the hard palate.

Molding action—Movement of displaced segments into a more normal relationship

Variations in isolated cleft palate (Lis et al., 1956)

Cleft of the soft palate only (Pruzansky, 1955)
The new force field created as a result of closing the lip cleft induces striking and relatively rapid changes in the architecture of the palate.

**Cleft of the Lip and Alveolus**

The alveolus is brought into normal approximation within a few months.

**Unilateral Cleft Lip and Palate**

The palatal segments in almost all instances move medially, resulting in the narrowing of the cleft space throughout its entire anteroposterior length. The posterior width of the hard palate as measured from the base of the alveolar crests is reduced even before soft palate repair is performed. Some additional molding action can occur after soft palate repair which will reduce the cleft space further, but the subsequent lessening of the cleft width is mainly due to appositional bone growth at the medial border of both palatal segments.

**EFFECT OF COMPLETE CLEFTS ON THE NASAL CHAMBER**

Subtelny (1955), Coupe and Subtelny (1960) and Aduss and Pruzansky (1967) showed that wider nasal chambers are due to lateral displacement of the maxillary bones in the oronasal area, and that the nasal septum is displaced in varying degrees toward the non-cleft side. Closing the lip cleft causes medial movement of the lateral processes, carrying the end of the septum closer to the mid-sagittal plane.

**COMPLETE UNILATERAL CLEFT LIP AND PALATE—3 POSSIBLE ARCH FORMS WHICH CAN OCCUR AS A RESULT OF ESTABLISHING UP CONTINUITY**

Pruzansky and Aduss (1964) described the three possible resultant arch forms which can occur: (1) Alveolar segments butt jointing into end-to-end contact producing a symmetrical arch form. (2) Overlap of alveolar segments produces a collapsed arch form. (3) Alveolar segments approximate, but without contact. The last may be a transient relationship; either it can end up in the overlapped relationship or the segments can approximate as a butt joint.
Newborn; unilateral complete cleft lip and palate

1. Noncontacting palatal segments
2. Overlapping palatal segments
3. Abutting palatal segments

1 year

2 months

2 months

8 months

1 1/2 years

Noncontacting palatal segments

Overlapping palatal segments

Abutting palatal segments
Approximation without contact of alveolar borders

The lateral segments may fail to touch because the inferior turbinate of the lesser segment makes contact with the nasal septum. (Aduss and Pruzansky, 1967). When the inferior turbinate was removed by surgery, the lesser segment moved more medially. When the surgery was done at the age of 4 years with the maxillary buccal teeth in overjet relationship, the occlusion became more nearly ideal. The medial movement of the lesser palatal segment brought the teeth into better relationship. This result supports the belief that in complete clefts of the lip and palate the lateral segments are overexpanded and not collapsed.

Overlapped palatal segments

This geometric relationship does not necessarily signify that the teeth in the lesser segment will be in a crossbite relationship at a later date when teeth have erupted. The force of the tongue acting within the vault space can exert lateral pressure against the palatal segments causing them to move outward. Spontaneous correction of overlapped segments has occurred as long as there was an absence of restraining scar tissue. Should a crossbite
develop, it can be corrected in two or three months by means of tooth-borne expanding appliances.

**Approximation with contact of alveolar borders resulting in excellent buccal occlusion**

The lip was closed at 1 month and the soft palate repaired at 11 months. The hard palate cleft was closed at 20 months of age.

The tip-to-tip buccal occlusion of the lesser segment, first seen at 2 years of age, was still in evidence three years later. The palatal cleft was closed at 16 months.
Approximation with contact of alveolar borders even with very wide complete clefts

The initial cleft geometric form is not predictive of the subsequent palatal form.

INFLUENCE OF MOLDING ACTION AND PALATAL GROWTH ON ARCH FORM WITHOUT THE USE OF MAXILLARY ORTHOPEDICS

Palatal growth

Palatal tissue has the potential of growing to normal size even though at the newborn state it may appear to be excessively small. However, it must be stressed that although the potential does exist for the palate parts to catch up and become the size one would ordinarily expect at later ages, such growth does not always take place either because of surgical disturbances of growth centers or in rare instances because of deficiency in osteogenic tissue.
There are no known geometric or quantitative parameters that can be used to predict the ultimate palatal form even when the same surgical procedures are used.
Timing of palatal closure

Much emphasis has been placed on the need to close the hole before the age of 2 years. Unfortunately age of closure is proposed as the main consideration for the achievement of proper speech production in later years. Timing of palatal closure should be governed by the size of the palatal cleft in relation to the size of the palatal shelves. Although in some cases it is preferable to close the cleft space before 2 years of age, in other instances it is best to wait until 3 or 4 years of age. We believe that poor speech production is due to many other factors than the mere age when cleft closure is performed.

Longitudinal growth studies support the contention that there is no optimal age for repair of the cleft palate. "Timing is an individual matter and is related to the anatomical and functional assets present in the patient." These studies show that a well-designed surgical procedure which is ill timed may lead to long-term failure. Conversely, a technically inferior procedure performed at a proper time can yield superior results.

Rapid reduction of cleft space in isolated cleft palate

Early reduction in the cleft space dimensions makes it possible to close the palatal cleft at 10 months without creating growth-inhibiting scar tissue. In some instances it may be advantageous to postpone palatal surgery until 2 or 3 years of age in order to allow for an increase in palatal tissue relative to cleft space.
UNILATERAL CLEFT LIP AND PALATE—ADVERSE EFFECT OF EXCESSIVE PERIOSTEAL UNDERMINING PERFORMED AT AN EARLY AGE

In this case excellent arch alignment and closure of the cleft space resulted from molding action and palatal growth.

Three months after "island flap" palatal lengthening, there was a buccal crossbite of the left lesser palatal segment. Note the slight transverse palatal scar.

A fixed palatal expander was used to correct the crossbite; correction accomplished in three months. A fixed palatal retainer was necessary to maintain the new arch form. In the absence of palatal scar tissue the expansion need not always be retained by a transpalatal arch. The determining factors are the relationship of the palatal segment and the amount of scar tissue.
The retainer was lost. The transverse scar is very prominent and has drawn the involved teeth medially, causing an hourglass-shaped palate. The adverse effect is also seen in the developing anterior cross-bite and crowding. Maxillary development was adversely affected in all three dimensions.

**Correction of Medial Collapse of Palatal Segments by Orthodontic Expansion—Deciduous or Early Mixed Dentition**

In complete clefts of the lip and palate, the united orbicularis oris, buccinator and superior constrictor pharyngis muscle ring mold the palatal segments which have been displaced outwardly and upwardly by aberrant...
muscle pull. Narrowing of the palatal cleft occurs throughout the entire anteroposterior length of the palatal segments from the incisal papilla to the maxillary tuberosities. This action has been found to take place not only after lip repair but also after soft palate surgery.

When the alveolar process of the smaller segment becomes contained within the premaxillary alveolar element of the larger segment, a dental crossbite occurs between the maxillary and mandibular teeth. Dental dysplasia may or may not coexist with segmental dislocation.

Correction of a crossbite in the deciduous dentition has been shown to improve dental function, nasal respiration, maxillary alveolar development, speech development and facial aesthetics.
Orthodontic therapy is directed toward counteracting the muscular influences created by lip surgery. An outward-directed force establishes a more normal arch form and widens the nasal cavity. This movement carries the inferior turbinate with it, increasing the distance from the nasal septum (Aduss and Pruzansky, 1967).

Orthodontic therapy is directed toward counteracting the muscular influences created by lip surgery. An outward-directed force establishes a more normal arch form and widens the nasal cavity. This movement carries the inferior turbinate with it, increasing the distance from the nasal septum (Aduss and Pruzansky, 1967).

Maxillary dental dysplasia can be due to faulty eruption patterns associated with hypertonic lip and cheek musculature. A non-scarred palate permitted the expansion and advancement of the buccal and anterior teeth with less concern for future relapse.

The closed left maxillary lateral incisor space was recovered and a normal anterior overjet and overbite relationship established after two years of orthodontic treatment. The missing tooth was replaced, and the buccal segments were retained with a fixed dental bridge which spans both sides of the cleft.
The ability to advance the maxillary incisors into a proper overjet relationship is the most reliable means for evaluating basal bone adequacy and normal maxillary development.

The left lateral incisor space was opened to receive a tooth of equal size as the right lateral incisor. A maxillary fixed bridge was created to replace the missing tooth and maintain the palatal segmental relationship. Placement of teeth in their proper relationship is dependent on palatal segment relocation as well as tooth movement.

Improvement in soft tissue profile was achieved after the teeth were relocated and the lip and nose revised.
Classification based on the presence or absence of soft tissue attachments

Complete bilateral cleft of lip and palate with a severely projecting premaxilla

Complete bilateral cleft of lip; complete cleft of palate on right side and incomplete cleft of palate on left side

Bilateral incomplete cleft lip left side, complete cleft right side; incomplete palate cleft right side, complete cleft on left side

BILATERAL CLEFT LIP WITH OR WITHOUT PALATE: WHY DOES THE PREMAXILLA PROTRUDE AHEAD OF THE PALATAL SHELVES?

Which of three mechanisms is operative? (1) There is an overgrowth of the premaxillary-vomerine complex response to altered muscular physiology, the result of unrestrained growth at the premaxillary-vomerine suture. (2) The projection of the premaxilla is an illusion created by underdevelopment or retroposition of the palatal shelves associated with micrognathia or retrognathia. (3) A combination of the above factors is in effect.

Recent research has reported that the complete bilateral cleft lip and palate deformity is characterized by an overgrowth or excess of mesoderm at the premaxillary-vomerine suture (Pruzansky). Our own clinical research using serial casts and roentgenographs refutes Burston’s earlier statements (1958) that

The bilateral condition is characterized by two retroplaced and small maxillae. The central stem of the premaxilla is relatively protrusive. It should be noted that although this latter element may be somewhat rotated in an upward direction, the actual amount of true over-development is often quite limited and the major defect lies in the retroplaced maxillae.

The incomplete bilateral cleft lip and palate shows relatively less geometrical distortion to all three palatal segments, whereas the complete bilateral cleft of the lip and alveolus with or without palate is characterized by an extreme forward projection of the premaxilla and in relation to the facial complex. When the cleft includes the secondary palate as well, the palatal segments are laterally positioned to varying degrees of distortion. In complete bilateral clefts the prominent premaxilla has long been recognized as one of the principal obstacles to successful bilateral lip and palate repair. However, time is an ally, and the singular lesson that has been learned from serial growth studies is the need to be patient and allow the palate and face to grow and develop at their individual rates.

VARIATION IN GEOMETRICAL RELATIONSHIPS AND SIZE OF PREMAXILLARY SEGMENTS

Any number of geometrical variations can exist between the palatal shelves and the premaxilla. For these and other reasons the molding effect of lip repair on the palatal segments and the resulting palatal form will vary from one case to another.

The size of the premaxilla is determined by the number of tooth buds it contains. There may be one, two, three, four or even five buds, either with one or both permanent central incisors alone or with one or both lateral
Variations in size and relationships of the premaxilla to the palatal segments at the newborn period

Large premaxilla making contact with well-developed palatal segments
Small premaxilla projecting ahead of small laterally displaced palatal segments
Large premaxilla due to the presence of four deciduous and four permanent tooth buds. The lateral palatal segments have not been displaced because of their attachment to the nasal septum.

incisors. These teeth are sometimes malformed or displaced from their normal position. In rare instances there is only one central incisor, the other being malformed or even missing. In some cases the palatal segments contain extra deciduous and permanent lateral incisors on either side of the cleft. If they are present on both sides of the cleft, the premaxilla may appear to be much wider than the available space between the palatal shelves. This situation can create the illusion that the palatal segments are in crossbite when in fact they may be in excellent relationship.

A symmetrical premaxilla with four tooth buds. A wire placed through the premaxilla will devitalize the teeth, leading to their destruction.
Small premaxilla with three deciduous teeth but only one permanent central incisor. Note extra teeth developing in the line of the cleft.

Geometrical changes resulting from molding action (Rapid closure of cleft space in incomplete bilateral cleft lip and palate.)

It is not possible to predict the final form of the maxillary arch (the relationship of the premaxilla to both palatal shelves) according to the original size and/or geometrical relationship of the three parts. The premaxilla in complete bilateral clefts may in some instances fit within the

1 week of age before surgery
palatal shelves, but usually it is finally positioned forward of them. For the palatal shelves to fall behind the premaxilla does not signify that they are medially displaced and the teeth will be in crossbite. Only in rare cases will the three segments come together in perfect arch alignment.

Both cephaloroentgenographic and cast analysis demonstrate that lip reconstruction or the use of external traction brings almost immediate reduction in premaxillary projection because of ventriflexion, the fulcrum being the premaxillary-vomerine suture. Septal buckling has been demonstrated in my computerized tomography studies. Marked facial change and reduction in the anterior cleft space result from the ventriflexion.

The figures demonstrate that excellent arch alignment can occur within the first year irrespective of the degree of premaxillary projection and lateral palatal distortion. In each instance the premaxilla came in contact with the lateral palatal processes although lying well forward of the lateral palatal shelves in the initial state.

**THE CONCEPT OF "CATCH-UP GROWTH"**

The surgeon, when planning the habilitation of a newborn with a complete bilateral cleft lip and palate, must rely heavily on the following facts:

1. At birth, all three palatal segments are geometrically distorted, but after lip surgery the molding action will bring the segments into a more normal relationship.
2. Uniting the lip will reduce premaxillary projection by ventriflexion with or without septum buckling. The profile can continue to flatten because of differential facial growth.
3. Further molding coupled with palatal growth will continue to reduce the remaining cleft space to more manageable proportions.
4. The inherent facial pattern will ultimately determine the handling of the premaxilla.
SERIAL GROWTH CHANGES TO THE COMPLETE BILATERAL CLEFT LIP AND PALATE (after one- or two-stage lip repair)

When judging palatal growth changes from casts, one must visualize an imaginary line (F-F) connecting the posterior end of each alveolar crest as the baseline from which to view anteroposterior growth. This line connects palatal landmark points which are comparable to cephalometric landmarks PTM (pterygomaxillary fissure) and marks the junction between the hard and soft palate.

Some workers have said that the lateral palatal segments, since they are detached from the nasal septum, are deficient in size (McNeill-Burston). It is my belief, acquired from looking at many serial casts, that, although the palates look smaller at the newborn period, they grow very rapidly and may approach normal size by the age of 3 years.

Superimposing three palatal stages of the previous series

Outlines of the first, fourth and fifth cast of the above case are superimposed on the line representing the posterior limit of the hard palate. Point Z is the registration mark for superimposition purposes only. The following conclusions can be drawn from this and other palatal growth studies; they demonstrate the limits of predictability of palatal change.

1. The premaxilla shows little anterior movement after lip repair. The repaired lip appears to retard the forward growth of the maxillary complex, thus contributing to the flattening of the facial profile.

2. The lateral palatal segments demonstrate excessive anterior and medial growth. The anterior growth rate exceeds that at the medial border. The three-dimensional surface area of the lateral palatal segments doubled in 1½ years. Between 1½ and 3½ years there was an additional 25 percent increase in surface area.

(After Slaughter and Pruzansky, 1954)
3. The premaxilla surface area changed only slightly during this same time span.

It is important to reemphasize that the geometrical palatal changes depicted here can be predicted in the majority of other patients. Exceptions are rare. Should the lateral palatal process fail to catch up to the premaxilla, then and only then should one consider surgical setback.

Why should the clinician want to set the neonatal premaxilla back to relatively smaller lateral processes when all the evidence indicates that within three years these same segments grow forward to reach the premaxilla? Friede and Pruzansky (1972) have shown that when the distance between the palatal shelves and the premaxilla exceeds 25 mm. a poor prognosis exists for conservative premaxillary handling and a retrusion of the premaxilla is in order. The timing for the retrusion is determined by many factors.

THE PROFILE IN COMPLETE BILATERAL CLEFT LIP AND PALATE

Any number of geometrical permutations may exist between the maxilla, the mandible and the cranial base which will affect the profile. Cephalometric facial growth studies have shown that the skeletal profile becomes less convex with age because of profile remodeling brought on by various growth increments to the upper, middle, and lower face.

Good facial growth occurs where the facial profile flattens with time, and the angle of facial convexity as measured by $N \alpha P_0'$ becomes more obtuse. This change is brought about mainly by the greater increment of forward growth of the chin point ($P_0'$) relative to the upper ($N$) and mid-face ($\alpha$) points. As already stated, lip repair over the projective premaxilla appears to retard the forward remodeling of the maxillary complex (Berkowitz, 1959).

The final profile characteristic will depend on whether the face is prognathic, mesognathic or retrognathic. In different individuals the same bone or group of bones may grow at different rates and at different times. In some
the same bone will grow rapidly and attain its full potential at an early age while in others it will grow more slowly but for a longer period of time.

The evaluation of success or failure of surgery cannot be made on aesthetic grounds alone and must be postponed until facial growth is nearly complete.

**Good facial growth**

The change in the profile was rapid on account of excellent mandibular growth and upper face development. The profile (angle of facial convexity) changed approximately 30 degrees within 1½ years.

**Poor facial growth pattern**

A disharmonious facial pattern represents the extreme of facial variation as seen when the premaxilla protrudes in a retrognathic face. Most faces show a growth pattern leading to reduction in profile convexity, the greatest change occurring during the first year of life. Although mandibular prognathism is the extreme of facial variation in the opposite direction, when found in complete bilateral cleft patients it may be beneficial.

SNA—85° SNP—75°

SNA—80° SNP—75°

SNA—83° SNP—90°

A. Retrognathia  B. Orthognathia  C. Prognathia

One cannot predict at an earlier age what the final profile will be. The continuing flattening of the profile until adulthood is a common occurrence. This reduction in facial convexity is mainly due to differential mandibular growth.

**Poor palatal growth pattern**

1 month  5½ years; excessive anterior cleft space  5½ years; premaxilla is in severe overjet-overbite relationship
When poor palatal growth pattern coexists with a poor facial growth pattern, the profile is placed in double jeopardy and retrusion of the premaxilla is warranted.

In complete bilateral cleft lip and palate the distance between the lateral palatal shelves and the premaxilla is a measure of the palate deformity. If it is excessive at 5 years of age—that is, if the premaxilla is not in contact with the palatal segments—one might have to consider surgical setback at some later date. The ultimate decision is made only after the profile is evaluated as well.

In this case the serial profile tracings showed that the premaxilla needed to be surgically set back since it was demonstrated that there was no further reduction in the angle of facial convexity (NαPo').

**Good palatal growth pattern**

Rapid resolution of the palatal deformity was achieved by molding action and good palatal growth within the first year.
Incomplete bilateral cleft of lip and palate left side and complete cleft of lip and palate right side. Good palatal growth pattern

Rapid resolution of the palatal deformity was obtained. The most laterally displaced palatal segment was moved medially and the premaxilla rotated into proper relationship. This movement, coupled with appositional palatal growth, markedly reduced the cleft space. The teeth on the most laterally displaced left segment ended up in a slight crossbite which will be easily reduced with a fixed expander. Correction of the buccal crossbite is dependent on the movement of the entire lateral palatal segment. The correction will have to be maintained by a fixed retainer until the permanent teeth erupt. After final orthodontics, a permanent bridge will be used to replace the missing teeth and to maintain proper arch form. Bony fixation of the palatal segment is, therefore, not essential. Facial growth studies have shown that primary bone grafting has interfered with maxillary development and has failed to maintain arch form.

5 years; after three months of orthodontic treatment. Bilateral buccal expansion using fixed toothborne appliance. Crossbite corrected by moving the palatal segments laterally.

6 years. Deciduous lateral incisors were extracted to permit improved premaxillary alignment. Palatal cleft was closed with no adverse effect to the palatal architecture.

Comments: The severe premaxillary overbite doesn’t pose any functional problems. It will be corrected by orthodontic means in the permanent dentition.
**Comments.** The possibility that a crossbite might occur at 2 years of age does not justify neonatal orthopedics.

2 years 3 months

2 years 8 months; after palate closure

---

*Complete bilateral cleft of lip and incomplete cleft of palate on both sides*

The palatal segments, which at first appear to be collapsed medially, are actually in an excellent relationship with the premaxilla. This case dramatically demonstrates the strong potential for palatal molding and growth to reduce the initial geometric disproportion.
Correction of an anterior open bite due to a superior displaced premaxilla in a bilateral cleft lip and palate patient

As the face grew and developed the surgically repositioned premaxilla, which was separated from the vomer at an early period, and detached from the lateral palatal segments, ended up displaced high in the nasal chamber. As the premaxilla failed to descend away from the cranial base along with the palatal segments a skeletal anterior open bite resulted. The buccal segments ended up in crossbite as well.

Comments. The displaced deciduous lateral incisors were extracted to permit the premaxilla to butt up against the lateral palatal segments spontaneously. The palatal cleft is almost completely closed by appositional growth. Except for a slight tip-to-tip left buccal occlusion, the dental relationship is excellent. The overbite will be reduced by orthodontia at a later date and poses no functional problem.
As soon as the permanent maxillary central incisors erupted, orthodontics was instituted to reposition them into a normal overbite-overjet relationship. The buccal segments were moved into proper occlusion at the same time.

7 years 6 months.

15 years. After final orthodontics.

15½ years. Fixed bridge inserted.
Final orthodontic treatment involved the removal of all first bicuspids in order to uncrowd the posterior teeth and to align the maxillary anterior teeth into proper overbite-overjet relationship. The left lateral incisor was moved into proper position even though a bone graft was not inserted in the line of the cleft. The right lateral incisor was congenitally missing and its space maintained for a false lateral incisor to be inserted later.

A maxillary bridge was placed between the right and left second bicuspids in order to stabilize the maxillary segments and replace the missing right lateral incisor. Relocation of the palatal segments were dependent upon the absence of restricting palatal scar tissues. Note the excellent vault space which permits normal tongue position and speech articulation.

The excellence of this result does not support early premaxillary repositioning.

Surgical Premaxillary Retrusion

Premaxilla surgically set back in late deciduous dentition

Because of the severe facial deformity and consequent malocclusion, it was decided that premaxilla setback should be immediately performed, without waiting for the permanent dentition. The objective was to eliminate the anterior cleft space and to bring the premaxilla into physical contact with the palatal segments. The premaxilla was to be stabilized in

4 years, severe overbite and overjet
three dimensions to allow for a possible bony union with the lateral segments. A removable prosthesis was used for fixation. In my experience a Kirschner wire not only fails to stabilize the premaxilla in any dimension but often causes deformations to the central incisors. Its use for any purpose should be abandoned.

Use of a wire to stabilize the premaxilla (?) Immediately after insertion and 6 months after removal. The wire penetrated the dental developmental sac, disrupting the formation of the central incisors.

Bands with lugs were placed on the deciduous molars to increase retention capabilities. A working maxillary cast was made and the premaxilla cut and repositioned against the palatal segments. Care was taken to place the premaxilla in proper geometrical position so that a normal overjet-overbite relationship resulted. A combination acrylic and steel prosthesis was fabricated, allowances being made for tissue swelling. The prosthesis was inserted at the time of surgery and worn for three months. It was removed only to maintain cleanliness. At the end of this time the premaxilla became semi-rigid because of unilateral ossification with the palatal segment.
In a case of complete bilateral cleft lip and palate the lip was repaired at 1 month and the soft palate was united at 12 months of age. The deciduous lateral incisors are displaced medially. Although the palatal segments appear to be collapsed when they are related to the wide premaxilla, the occlusion shows them to be in excellent buccal relationship. A moderate-sized palatal cleft remains.

Three months after surgical setback the overjet-overbite relationship appeared to be stable. There was some slight ventriflexion to the premaxilla which posed no long-term functional problem.

Poor palatal growth with a poor facial growth pattern

In a case of complete bilateral cleft lip and palate the lip was repaired at 1 month and the soft palate was united at 12 months of age. The deciduous lateral incisors are displaced medially. Although the palatal segments appear to be collapsed when they are related to the wide premaxilla, the occlusion shows them to be in excellent buccal relationship. A moderate-sized palatal cleft remains.
Presurgical palatal and facial evaluation and palatal preparation

The premaxilla is to be set back because the anterior cleft space has failed to reduce and is associated with a poor facial growth pattern. The anterior positioned mid-face complex, coupled with a retrusively growing mandible, has created a severe facial convexity. This profile characteristic appeared to be stable at 6½ years of age.

Even with the buccal segments in good occlusion, the arches were overexpanded prior to surgical premaxillary setback in order to permit premaxillary repositioning within the palatal arches. This buccal expansion is not permanent, and it is anticipated that the arches will return to their original position as a result of muscle-molding action.

After premaxillary setback and closure of the palatal cleft

Deciduous lateral incisors were removed and the premaxilla set back. A slight overbite and overjet was established to allow for differential mandib-
A bilateral cleft lip and palate

Arch form and size are excellent after reestablishment of lip muscle continuity. Palatal increase in size by appositional growth has further reduced the cleft space. This growth capability is inherent in all cleft palates and will occur but at various times and to various degrees.

DELETERIOUS EFFECT OF NON-PHYSIOLOGICAL SURGERY

A bilateral cleft lip and palate

Arch form and size are excellent after reestablishment of lip muscle continuity. Palatal increase in size by appositional growth has further reduced the cleft space. This growth capability is inherent in all cleft palates and will occur but at various times and to various degrees.
Extensive periosteal undermining performed in an “island flap” procedure at the age of 20 months resulted in severe scarring. The buccal crossbite and anterior incisal relationship reflect the growth-inhibiting effect. Cephalometric analysis demonstrates that growth inhibition occurs in the vertical dimension as well. This case is now an orthodontic nightmare and may require a Le Fort I advancement.

Premaxilla setback followed by palatal cleft closure resulting in scarred, deformed vault.

3 years. Good buccal occlusion, severe overjet.

1 year 10 months; after palatal surgery. Bilateral buccal crossbite with tip-to-tip incisal relationship.

7 months; after lip surgery

3 years 5 months; bilateral buccal crossbite

Vault space is obliterated due to scar contracture.
4 years 6 months. Excellent buccal occlusion. The lateral incisors were to be extracted prior to surgical setback.

5 years 6 months; after retropositioning of premaxilla.

7 years. Right buccal crossbite; asymmetrical tapered palatal arch.

Comments. The palatal cleft was surgically closed at 6 years. Palatal fistula closure at 6½ years necessitated periosteal undermining. The resulting scar contracture deformed the palate causing dental crossbite.
Comments. An island flap will not cause palatal deformation if applied on a well-developed palate with adequate tissue and if the lateral incisions are made at least 5 mm. medial to the dentition. (Charles Kremenak was the first to appreciate the need to keep the surgical incision away from the teeth.)

NORMAL PALATAL ARCH DEVELOPMENT AFTER A REVAMPED ISLAND FLAP PROCEDURE WAS PERFORMED AT FOUR YEARS OF AGE

Palate and occlusion analysis

3 years; before surgery. Ideal occlusion. Slight incisal tip-to-tip contact may be due to hypertonicity of the lip musculature.

4 years 6 months; one year after island flap surgery. Good occlusion still present. After island flap, normal arch form.

6 years 6 months; 2 years 6 months after surgery. Good occlusion and palatal development. Excellent arch form.
IN CONCLUSION

These concepts of orofacial growth present a logical sequence of treatment steps based on the natural history of cleft palate and the face in which it exists. Although the surgeon would like to get on with it and close the cleft space as soon as possible, he must temper this urge with the realization that “Time is an ally” to his reconstructive procedure.

Treatment guidelines can be listed as follows:

1. In complete cleft of the lip and palate the palatal segments are laterally displaced in space.
2. Unitilng the lip and soft palate causes medial molding of the displaced segments to a more normal anatomical relationship.
3. The palatal processes have the potential to grow and develop to normal size and shape albeit at a somewhat later date.
4. Non-physiological surgical procedures can inhibit palatal growth and cause structural deformation.
5. *Timing* of surgical procedures must be related to the assets and deficits presented by each case and varies within each cleft type, as well as with the facial growth pattern.
6. The same surgical procedure can yield different results. What might be successful in one instance can be disastrous in another.
7. The surgeon does not always have complete control of the habilitative outcome.
8. Velopharyngeal capability is related not to the degree of palatal distortion or cleft type but to the pharyngeal architecture and to the size and activity of the velum. This functional relationship can change with time.

The 1977 State of the Art in Cleft Palate report sponsored by the American Cleft Palate Association reviewed the recent dental literature and stated that there is no data to support neonatal maxillary orthopedics with or without primary bone grafting. Although it has been well established that palatal segments can be manipulated, the long term utility of the procedures has still not been proven. It has been suggested that since retention problems associated with maxillary arch alignment are a major problem, arch correction should be postponed to when tooth-borne appliances can be used. No data has been published supporting the belief that the overlapping of the palatal segments for 3 to 5 years is detrimental to palatal growth or speech development.

Since all the statements in this chapter are supported by published facial growth studies, we need not *reinvent the wheel* by redoing the same studies. It behooves the surgeon to document his own cases so that he can critically review his results and ask the right questions: Why did the case turn out well or why did it fail? Progress can take place only when the correct answers to these questions are uncovered.
FUTURE STUDIES
As a result of the development of an accurate automatic non-contact three dimensional measuring device, called an optical profilometer, I intend to investigate various research problems which, heretofore, have not been explored due to the absence of a proper measuring instrument. Stereophotogrammetry is an excellent system but it can be too expensive for our purposes. Hopefully, this investigation into changing palatal form and size under the influence of growth and surgery will lead to the development of a new cleft palate classification system which would be predictive of future palatal size and shape.

The optical profilometer concept consists of an optical system and photodetectors which observe the change in the energy distribution of an image spot of light as a function of the depth of the sample surface at the measurement point. The sample surface is scanned to provide measurement information over the complete surface. It can measure a cast within an accuracy of ±4 mm in all dimensions in 9 minutes.

By the pooling of quantitative data from a number of cleft research centers which already have a great number of palatal casts in storage we will be able to significantly increase our data base. This will enable us to arrive at more meaningful conclusions about the natural history of cleft palate development.

Dr. Berkowitz is setting a palatal cast in the moving carriage of the optical profilometer. Immediately behind the optical profilometer is the voltage adjustment console, and on a shelf above the console there is an "Intellec #4" mini-computer. To the right of the console a "silent 700" terminal-printer is used to control the instrument's operation and will print a graphic display of the data. The data is simultaneously stored on a tape for analysis by a larger computer.
5. Ear Disease and Hearing Loss in Cleft Palate

Cleft palate has long been associated with ear disease and hearing loss. In 1878 A. Alt was the first to mention a case of otorrhea in a deaf-mute patient with a cleft palate. He closed the cleft, and the otorrhea healed. The patient was able to hear again and then learned to speak.

Conductive deafness associated with cleft palate has been reported to occur in between 27 and 50 percent of patients: Halfond and Ballenger, 50 percent; Nylén, 40 percent; Skolnik, 39 percent; Pagnamenta, 30 percent; Spriestersbach, 29 percent; and Meissner, 27 percent.

Possible causes considered in this association of cleft palate and ear disease have varied among repeated infections following the lack of an intact palatal partition between the oral and nasal cavities, disturbance by the cleft of the normal muscle physiology necessary for adequate Eustachian tube function, timing and type of surgery, scarring after surgery, effect of hamular fracture, actual abnormal Eustachian tube anatomy, and cranial base deformity.

Reflux

The presence of a cleft in the palate was thought to allow reflux up the Eustachian tube causing otitis media. Even when treated with antibiotics without drainage, the purulent material was merely converted to a sterile exudate which remained as a foreign body resulting in conductive hearing loss.
LACK OF MUSCLE FUNCTION

As more thought and research were directed to this problem, blame was laid on the abnormal insertions of the musculature which, disrupted by the cleft, prevented normal action and adversely affected the function of the Eustachian tube. Surgeons started to look to closure of the cleft in the palate as early as possible as a step toward partially improving the muscle function.

EARLY SURGERY ADVOCATED

In 1960 Frank Masters, Hal Bingham, and Dave Robinson of the University of Kansas Medical Center began to study hearing loss in cleft palate patients and added normal hearing to normal speech and normal facial growth as goals of cleft palate treatment. They deduced:

The chronic recurring middle ear infection with its subsequent audiometrically detectable hearing deficit is a manifestation of altered eustachian physiology which in the cleft palate child is almost inevitable as the extrinsic musculature of the eustachian apparatus is not intact. Thus, early restoration of the dynamic physiology of the eustachian apparatus by creation of a normal muscular insertion appears to be the best method available to reduce the appalling incidence of hearing loss.

TREATING THE EAR DISEASE ITSELF

In 1954 Beverly W. Armstrong of Charlotte, North Carolina, first described insertion of pressure equalization (PE) tubes to maintain aeration following myringotomy, aspiration and tympanostomy for middle ear fluid.

In 1966, in the Cleft Palate Journal, James Donaldson of the University of Washington, Seattle, published an illustration of insertion of the Silastic tube after myringotomy, with the inner flange passing through the tympanic membrane and the outer flange securing it in place. He reported that, out of 702 cleft palate patients examined, 98 (13.9 percent) had middle ear pathology requiring one or more myringotomies. The infant was not suspect, but ages ranged from under 1 up to 15 years, with the largest number in the 3- to 4-year period. To establish middle ear aeration in these patients with inadequate Eustachian tube
function he advocated insertion of Silastic tubes. Although the tubes tended to be extruded sooner than desired, temporary middle ear aeration frequently improved the appearance of the tympanic membrane, allowing it to assume its normal position rather than a retracted one.

EAR PATHOLOGY IN THE INFANT

Still no one suspected ear pathology in the infant. In 1958 Skolnik had stated that the incidence of pathology was 6 percent in cleft palate children younger than a year old and increased to 60 percent in preschool children.

In 1967 Sylvan E. Stool and Peter Randall at the Children's Hospital, Philadelphia, reported on bilateral examination of the middle ears of 25 cleft palate infants under general anesthesia through a binocular operating microscope after myringotomy. Ninety-four percent of these ears contained mucoid material, and each of 10 biopsy specimens revealed the presence of granulation tissue. They suggested:

A reasonable approach to the problem of otitis media in the cleft palate infant is to examine these patients with magnification whenever they are having reconstructive surgery. The removal of abnormal material from the middle ear and the ventilation of this cavity should allow pneumatization of the ear to proceed in a more normal manner and should aid significantly in preventing subsequent hearing loss.

Interested in how it all began, I asked Peter Randall, who had long been concerned with the miserable problems of draining ears in children with clefts and the difficulty of finding cooperative otolaryngologists. Here are his reminiscences, written to me in 1973:

On September 11, 1963, Sylvan arrived on the scene. As you know, he is a somewhat rotund, jolly genius who had gone from medical school in Texas through his Pediatric training in Seattle, Salt Lake City and Boston and through a complete Otolaryngology residency at the University of Colorado in Denver. Sylvan arrived in Philadelphia fresh out of his residency being Board qualified in both Pediatrics and Otolaryngology with a lot of enthusiasm, absolutely no practice whatsoever and a perfectly beautiful, brand new Zeiss operating microscope. The day after he arrived we had our big monthly Cleft Palate Clinic and Sylvan—who had wondered how long it
would be before he saw his first patient—found one ear after another with problems beyond description. He described it as an "Otolaryngologist's Paradise." Within a very few days, he was in the operating room pushing his new Zeiss scope from one room to another asking me if I would mind if he looked at the ears of a child whose cleft lip I was about to tackle. This was the first time we had even thought of looking at the ears in an infant this young (about three months of age), and I thought he was really just trying to get experience with his new operating room toy. The child had had no known trouble with his ears, but Sylvan immediately said, "Oh my gosh, look at this!" which I did but frankly I didn't see very much. Then he asked if I would mind if he made a little hole in the eardrum and I asked how little was a little hole and why he wanted to do it, and he said that it was very little, would heal very quickly, and the reason was that he was sure there was a lot of trouble behind that particular drum. I shudder to think of my lack of informed consent, but under the circumstances it seemed that this would be the best thing to do for that child at that point. After myringotomy he began sucking out thick, inspissated, mucoid "glue." I thought that he had opened into a mucocele. He then asked if I would mind if he looked at the other ear which I readily agreed to, and the same problems were found on that side as well.

As luck would have it we had a second cleft lip to follow in the next room, and it wasn't long before Sylvan was asking if I would mind if he looked at that child as well. At that stage of the game we were taking Silastic intravenous catheters and cutting them into small pieces to use for insertion in the myringotomy openings. The various "buttons" were not yet available at Children's Hospital.

We were soon misled by a child whom we examined at about six weeks of age and actually found granulation tissue within the middle ear space, which led us to believe that much of this material was infected. However, Charles Bluestone has since shown that virtually all of it is not infected in these infants. We set out to try to study the incidence, and couldn't really figure out how to get a "control group." Then one day, I pulled a dirty trick on Sylvan and asked him to look at the ears of a child who had a cleft of the lip but no cleft of the palate. After he did the myringotomy and found that this was a perfectly normal drum with an air-containing middle ear space, he asked, "What is the matter with this child? His ears are perfectly all right." Then it was obvious that our control group should come from those patients with clefts of the lip only. We found that 87% of our infants with cleft palate had thick inspissated mucus in their middle ear space and only about 28% of those with clefts of the lip only had any kind of fluid in the middle ear space and this was usually quite watery.

Buddy Bluestone had been working along the same lines with Jack Paradise at Pittsburgh, but I'm fairly sure it did not antedate our work in
1963. It's interesting that they came up with the same incidence figures exactly that we had, but they carried it one step further in that they brought back the children who did not have the mucoid material in the middle ear on one and even two successive occasions and showed that eventually 100% of these children get into trouble with collection in the middle ear space. Bluestone has done a very careful study of the fluid, its characteristics and studies on the Eustachian tube.

Almost simultaneously, the team of Paradise and Bluestone had directed its interest toward this ear problem. Jack L. Paradise, pediatrician at the University of Pittsburgh, in an occasional escape from crying babies, soothes his nerves by restoring antique oriental rugs. Enticed into the Cleft Palate Clinic by Betty Jane McWilliams to look into the problem of feeding and nutrition in young cleft infants, he consistently discovered bilateral secretory otitis media.

C. D. Bluestone, Alpha Omega Alpha at the University of Pittsburgh School of Medicine, became interested in the cleft palate problem in 1959 by Sam Pruzansky, while interning at the University of Illinois. In 1964 he joined the cleft palate team as otolaryngologist at the University of Pittsburgh and by 1966 was confirming Paradise's findings by myringotomy with an operating microscope. In 1969 Paradise, Bluestone and Felder reported the universality of otitis media in infants with cleft palate:

Bilateral secretory or suppurative otitis media was found without exception in 50 infants with cleft palate 20 months of age or younger. By contrast, in a group of 100 infants without cleft palate chosen at random, otitis media was present during 22 percent of 274 clinic episodes they experienced during an 8-month period. . . . Infants with cleft palate who received myringotomy and suction, but without insertion of plastic tubes, developed early re-accumulation of fluid which required repetition of surgery. The course of infants who received myringotomy with tubes was generally satisfactory; but in some, otorrhea, early extrusion of tubes, or both occurred.

Chronic secretory or suppurative otitis media probably develops in all infants with cleft palate, and usually within the first month of life. In such infants, myringotomy accompanied by the insertion of tubes appears to be the best method of treatment now available. This procedure may be performed promptly and repeated as often as necessary to maintain middle ear aeration. It is hoped thereby to avoid the scarring, ossicular damage, cholesteatoma, or suppurative intracranial complications which otherwise
might eventually develop in some patients. Also, if surgery is not performed, hearing impairment will probably persist throughout infancy or longer, with untoward effects on well-being and function and with serious implications for intellectual, speech and emotional development.

Then Jack Paradise in 1970 studied 200 children under 12 months of age—100 with cleft palate, 100 without clefts. He found serous fluid in the middle ear in 100 percent of the cleft and 20 percent of the non-cleft children.

Also in 1970, Joyce Heller, Irving Hochberg and Gastone Milano of Newark State College, Union, New Jersey, studied 60 cleft palate and 60 non-cleft palate children aged 3 to 12 years. They found:

Cleft palate children have significantly poorer hearing sensitivity than non-cleft children, but all threshold deviations were within normal hearing limits for both.

There was an improvement in hearing sensitivity in both cleft palate and non-cleft palate children as a function of increasing age and this was most evident above six years.

There was a significantly greater incidence of conductive hearing impairment (significant air-bone gap) and aural pathology in cleft palate children than in non-cleft palate children.

Approximately 50 percent of the cleft palate children whose otoscopic findings were positive showed unilateral abnormalities.

SUBVERSIVE SUBMUCOUS CLEFT PALATE

In 1971 La Vonne Bergstrom and William Hemenway of the University of Colorado Medical Center used otolaryngological and audiometric examination to evaluate 58 patients with submucous clefts of the palate. Recurrent or chronic disease of the middle ear in 39 percent ranged from serous otitis media to cholesteatoma. Thirty-four percent had conductive hearing loss, and 25 percent had either pure sensorineural or mixed hearing losses. Half of the patients with middle ear disease did not have speech disorders, and hence submucous cleft palate might not be suspected on that basis. Submucous cleft palate should suggest the possibility of accompanying, perhaps
asymptomatic, middle ear disease, and unresolving middle ear disease might be the reason to suspect SMCP.

ADVOCATES OF EARLY CLEFT CLOSURE

In 1972 Charles Bluestone, Jack Paradise, Quinter Beery and Ronald Wittel studied 22 infants with unoperated cleft palates who received myringotomies with tube insertions during the first three months and between the ages of 18 and 24 months had secondary palate closure with a V-Y pushback with or without an island flap. They evaluated Eustachian tube protective function by roentgenographic studies after instillation of radiopaque media into the nasopharynx and noted:

Infants with unrepaired palate clefts were conditionally able to clear radiopaque fluid media in a prograde direction from the Eustachian tube and middle ear into the nasopharynx, but there was obstruction to retrograde flow from the nasopharynx into the Eustachian tubes. Following surgical repair of the palate, there was improvement in prograde clearance and, in over half the ears tested, retrograde flow appeared normal.

Abnormal distensibility of the Eustachian tube in infants with cleft palate was suggested by the results of tests following repair of the cleft. This distensibility may be a function of reduced tubal stiffness (increased compliance) which may in turn contribute to functional obstruction of the tube by rendering its opening more difficult.

Their conclusion:

Prior to closure of the palate, middle ear fluid is present in all untreated infants. Since this seems to be related to functional obstruction of the Eustachian tube, ventilation of the non-aerated middle ear cavity is indicated. A previous study has shown that closure of the secondary palate is often followed by a reduction in the prevalence of middle ear disease. The present investigation confirms this finding and suggests a relationship to improvement in Eustachian tube function. It would, therefore, seem worthwhile to reconsider repairing the palatal defect at as early an age as possible, especially when otorrhea through a tympanostomy tube has failed to respond to medical treatment. Following repair of the palate, recurrence of middle ear effusion warrants repetition of myringotomy and insertion of tympanostomy tubes. However, if Eustachian tube function is improved following palate repair, or if otorrhea develops through the tympanostomy tubes, their removal on a trial basis may be indicated.
In 1973 Paulsen of Denmark reported:

Ear disease seems a little easier to control in patients with incomplete than with complete palate clefts, and a striking finding in patients with all types of clefts has been the improvement in overall otologic status following palate repair.

In 1975 E. R. Soudijn and A. J. C. Huffstadt of the State University Hospital, Groningen, studied 132 standard cleft palate patients before and after palate surgery. They found "glue" in the ears of 94 percent of the presurgical cases and reported:

Six months after the closing of the soft palate, the percentage of glue ears appeared to have already been reduced to 65. There is of course a tendency for decrease of aural pathology with advancing years, but this does not occur until the age of four, according to Graham. In our patients the highest age was only 19 months.

The most interesting aspect of our study however is the relatively high percentage (30%) of glue ears in children with cleft lips and in children of the control group. ... In babies with a cleft palate the pharyngeal mechanism appears to be so severely disturbed, that in 94% of the cases glue cannot be drained from the aural cavity.

They concluded:

Probably all children's middle ears contain glue at birth. In the cleft palate babies glue is present in 94%.

Inspection of the eardrums, eventually followed by myringotomy in cleft palate babies is indicated periodically.

Closing of cleft palate by surgery leads to improvement of tubal function.

In 1974 Margareta Korsan-Bengtsen and Olle Nylén of Göteborg, Sweden, studied the ears and hearing of 60 children aged 8 to 15 years who had had a Wardill-Kilner palate closure at 16 to 20 months and, if the cleft was complete, had had a Johanson-Ohlsson bone grafting at 8 to 10 months. Hearing and middle ear function was normal in 33 out of the 60. Screening audiometry revealed the incidence of conductive hearing loss exceeding 20 decibels at two or more frequencies to be 3 to 5 percent. The corresponding percentage of operated cleft palate children was 20 percent. This figure was considered very favorable since 75 percent of these children had serious middle ear disease before surgical closure of the palate. They concluded:
It would thus seem justified to recommend early palate closure, even from an otological point of view, in order to prevent tubal dysfunction with middle ear changes and hearing loss.

In 1977 Lee Dellon wrote:

In 1969 the first patient we corrected the levator of told me when he awoke that he could hear better. Post-op audiograms demonstrated correction of his conductive hearing loss. All we had done was alter his levator's insertion. I believe long-term follow-up will show that if levator retrodisplacement is included in the primary palatal repair, not only will speech results be improved but also hearing loss diminished, and I have written this limerick to emphasize it.

Medical art student Susan Seif and Dellon have written a 1977 study of “Interrelationships of the Levator and Tensor Veli Palatini Muscles and the Eustachian Tube: An Anatomic Reconstruction from Serially Sectioned Fetal Heads.” It discounts the tensor as a tube opener but acknowledges the part played by the levator:

Indeed during isometric contractions the TVP increased girth will exert a pressure radially inward against the paratubal tissue. . . . The levator veli palatini muscle (LVP), as it goes from lateral and inferior to the medial plate of the Eustachian tube cartilage medially into the mobile soft palate, is in a unique position to elevate the medial tube cartilage. An isotonic LVP contraction elevates the soft palate posteriorly and the tubal cartilage medially. During this LVP muscle contraction the paratubal tissue would be compressed radially inward from below, while the radially inward “closing” pressure exerted by the “resting” medial cartilage would be relieved. . . . The incidence of hearing loss diminishes with increasing age because . . . with growth and development the LVP origin moves laterally (increasing its effectiveness in releasing the pressure of the medial cartilage plate on the tubal lumen) and the insertion moves downward and forward (increasing velar excursion and, again, increasing LVP effectiveness as a tubal “opener.”

Oh, Levator Veli Palatini,
Thou art more than wet linguini,
For when velum quakes
and Eustachius awakes
’Tis ’cause thou art in-betweeni

Not All Cleft Closures Cure

In 1962 D. C. Spriestersbach, Dean Lierle, Kenneth Moll and William Prather of the State University of Iowa studied hearing in 163 cleft lip and palate patients and found the incidence of loss and magnitude of threshold deviations significantly greater in the
youngest age groups (33 to 77 months) than in any of the older groups. They noted no significant correlation between physical management (type or time of surgery) and hearing loss.

Unfortunately, closure of the cleft in the palate, although usually of benefit, does not invariably cure the ear problem. This failure has been blamed by some on certain aspects of the surgery, such as hamular fracture, tensor tendon division or other traumatic and scarring procedures responsible for preventing the development of normal tubal function. A 1920 study by A. R. Rich of Johns Hopkins University utilized a palatal incision to allow visualization of the tubal orifices. Rich found these orifices to be closed normally at rest but opened during the swallowing, yawning and sneezing reflexes.

The levator palatini, the palatopharyngeus, the internal pterygoid and the superior constrictor muscles of the pharynx, when either cut or stimulated, were found to exert no influence whatever upon the patency of the orifice or lumen of the tube.

The tensor palatini was the only muscle functionally related to the Eustachian tube. Contraction of this muscle was always accompanied by a dilatation of the tubal orifice and lumen. Relaxation or division of the tensor palatini was followed by a passive return of the tubal walls to the condition of approximation which they normally occupy when at rest. This work, of course, eventually pointed an accusing finger at fracture of the hamulus or division of the tensor tendon during cleft palate surgery.

**HAMULAR FRACTURE**

Fracture of the pterygoid hamulus during cleft palate closure has been blamed for perpetuating hearing impairment. Newmann in 1968, Graves and Edwards in 1944, and McMyn in 1940 all demonstrated that in addition to the main tensor veli palatini, which hooks around the hamulus to insert into the soft palate, a smaller component of this muscle arises from the Eustachian tube cartilage and inserts into the hamulus and the end of the hard palate. Skolnik in 1958 and M. A. Ross in 1971 confirmed that hamular fracture is related to occurrence of otitis media. Politzer,
as early as 1862, pointed out that the muscular relations of the Eustachian tube of the dog correspond closely to those of man. In 1962 Holborow's experiment in dogs concluded that "the integrity of the tensor is essential for tubal opening," and in 1971 Odoi, Proud and Toledo, after unilaterally "expunging" the hamulus in dogs, reported development of middle ear effusions.

Yet modern human studies raise much doubt as to whether hamular fracture affects ear physiology. In 1968 M. Bennett, R. H. Ward and C. A. Tait found no such increase after tensor tendon division. In 1972 Bluestone, Paradise, Beery and Wittel, following unilateral hamular osteotomy and infracture on 12 patients, noted:

No difference in Eustachian tube function was observed as a result of this procedure, and although follow-up has been short, recurrences of middle ear effusion have also appeared unrelated.

In 1973 in the *Cleft Palate Journal* Barrett Noone, Peter Randall, Sylvan Stool, Ralph Hamilton and Richard Winchester of the University of Pennsylvania presented a sketch of their rendition of tube, tensor and hamulus anatomy. They then reviewed a randomized series of 89 patients undergoing soft palate closure between 1963 and 1969 in which unilateral hamular fracture was alternated between the right and left sides. They reported:

An evaluation of the development of clinical middle ear disease and documented hearing loss by audiogram during a three-year postoperative fol-
low-up period demonstrated no difference between the ear on the side of hamulotomy compared to the opposite ear.

OTHER SURGICAL TRAUMA

In 1966 Otto Kriens of Bremen, while studying the anatomy of palatal musculature in Prague, concluded that the levator muscle elevated the medial edge of the tubal cartilage and additional opening was effected by the pull of the tensor muscle. In the cleft palate, the pull of the tensor muscle was in deviated craniolateral motion which did not elevate the medial edge of the Eustachian tube. Kriens warned against surgical intervention near the epipharyngeal portion of the tube. He felt that fracture of the hamulus might disrupt the musculotendinous apparatus near the tube, and, even worse, dissecting and packing the space of Ernst could disrupt the equilibrium of the muscles in this area, to be further compounded by resultant scarring.

EFFECT OF PHARYNGEAL FLAP ON HEARING

Evidently pharyngeal flaps do not affect hearing. M. D. Graham and D. Lierle found in 1962 that the pharyngeal flap procedure did not aggravate an existing hearing loss. In 1966 G. Aschan concluded that surgical reconstruction by velopharyngoplasty helps to restore tubal functioning and thus hearing. G. W. Leworthy and H. Schliesser reported in 1975:

The application of a pharyngeal flap did not decrease the preoperative hearing acuity in 96 per cent of our 53 patients.

T & A

To remove or not to remove the tonsils and adenoids, that is a question even in the normal child. Recurrent nasal discharge, repeated attacks of otitis media and respiratory obstruction are accepted as criteria for a T & A. In 1965 N. I. Chalat noted improvement in hearing in 75 percent of cleft palate patients after tonsillectomy and adenoidectomy.
Yet in the non-cleft child or in the unrecognized submucous cleft case, removal of the adenoids can result and has resulted in the disastrous production of hypernasal speech. In the cleft palate child the need for any extra prominence in the posterior pharyngeal area to aid in velopharyngeal closure causes hesitation in the random removal of the adenoids. Some surgeons are enthusiastic about partial adenoidectomy in the treatment of otitis media, preserving speech by removing only the lateral portion of the adenoid under direct vision. This maneuver, however, is not so easy. As suggested by Stool, it may be possible to preserve the child's adenoid tissue if the ear disease is treated via the tympanic route first.

The removal of the tonsils is slightly less hazardous, provided the surgeon shells out the tonsil carefully, preserving the anterior and especially the posterior pillars and avoiding excessive postoperative scarring.

POSSIBLE ANATOMICAL CAUSES OF INADEQUATE TUBE FUNCTION

According to Wilma Maue-Dickson of the University of Miami, there is no indication that the extrapalatal anatomy of the tensor veli palatini muscle is abnormal in cleft palate. Even in severe cleft of the palate the presence of a well-defined palatal aponeurosis (the tendon of tensor) can be demonstrated in the palatal tags in histological sections. In 1975 an anatomical study by Maue-Dickson showed that human fetuses with cleft palate consistently have (1) narrower and smaller auditory tube lumina which are more widely separated than in normals, (2) greatly enlarged auditory tube cartilages, also more widely separated than in normals, (3) more widely separated pterygoid plates than in normals, and (4) significantly reduced pharyngeal height but greatly increased pharyngeal width—all of which were shown in cross section in Chapter 2. One conclusion from these data is that the space between the lateral pharyngeal wall and the side wall of the cranium is substantially reduced and that the tube may suffer a mechanical disadvantage as a result. The problem may be reduced by craniofacial growth, which sometimes relieves stress.
on the tube. This suggestion is consistent with the observation that children with cleft palate typically have reduced middle ear problems within the first few years of life.

**PATHOGENESIS**

It is the responsibility of the Eustachian tube to ensure that the external and the middle ear air pressures remain the same. Anything that interferes with evacuation of embryonic tissue from the middle ear and subsequent pneumatization of the temporal bone, or with the function of the Eustachian tube to prevent it from supplying the middle ear with air, will result in conductive hearing loss.

There are several clinical stages in the pathogenesis of ear disease, as noted by Stool. The *exudative stage* has acute, subacute and chronic phases.

The *viscid stage* results from failure of resolution of the exudative stage. The middle ear is filled with thick, tenacious mucoid substance. The audiogram usually reveals an average loss of 3 decibels due to failure of movement of the tympanic membrane. Treatment involves removal of the viscid material from the middle ear via a myringotomy and substitution of a prosthesis for the function of the Eustachian tube.

In the *adhesive stage* the tympanic membrane is adherent to the structure of the middle ear. It is usually seen in later childhood when the previous stage was not resolved. The membrane becomes atrophic and flaccid and collapses against the medial wall of the middle ear with a loss of 40 to 60 decibels. Desquamated epithelium gets caught and collected in the adhesive pockets of the tympanic membrane. Treatment includes myringotomy, aspiration of fluid and insertion of tubes coupled with eversion of invaginated pockets. The replacement of atrophic membrane with grafts may be of value. The best treatment for this stage is prevention.

The most serious complication is the formation of a cholesteatoma. In the cleft palate this is acquired during the adhesive stage when invaginated pockets collect squamous debris. Diagnosis may depend on x-ray demonstration of a radiolucent area.
Removal of the abnormal tissue is mandatory for cures and to prevent extension into surrounding structures including the brain.

In 1975 Jack Paradise took an international view of middle ear problems associated with cleft palate, stating:

Eighty years have elapsed since Gutzmann in Germany first observed that approximately half of all patients with cleft palate suffer from significant reduction in auditory acuity. . . . The mucous membrane lining becomes markedly thickened, the epithelial cells undergo metaplasia, and there is a great increase in the secretion of mucus.

Noting that even children with normal palates commonly experience secretory otitis media, he listed a string of international investigators studying the histological and biochemical abnormalities that characterize this sterile inflammatory process in the middle ear. Sade (Israel), Senturia (U.S.A.), Paparella (U.S.A.), Lupovich (U.S.A.), Gunderson and Gluck (Norway) and Mogi (Japan) have all contributed to a better understanding of the middle ear changes.

**PULLEN**

Fred Pullen of the University of Miami School of Medicine, while training at the Massachusetts Eye and Ear Infirmary in Boston, became infatuated with otology under Harold Schuknecht. Pullen's nasal fracture during a Michigan Golden Gloves tournament, a skilled light touch and his center-of-the-target attack on clinical problems made him the perfect otorhinolaryngology member of our Cleft Palate Team. Here is his 1977 stand:

The Eustachian tube connects the middle ear cavity to the nasopharynx. In the adult, the anterior two-thirds is cartilaginous and the posterior third bony, but in the infant the bony portion is relatively longer. The direction of the tube in the adult inclines superiorly with the horizontal plane at an angle of 30° to 40°, but in the infant this inclination is only 10°. The lumen of the Eustachian tube is shaped like two cones with the apex of each directed toward the middle. The aural orifice of the tubes is oval in shape, measuring 5 mm high and 2 mm wide in the adult. The nasopharyngeal orifice in the adult is a vertical slit at right angles to the base of the skull, but in the infant this opening is oblique due to the more horizontal position.
of the cartilage. The diameter of the orifice is 8 to 9 mm in the adult and 4 to 5 in the infant. In the newborn, the nasopharyngeal orifice lies in the plane of the hard palate, but in the adult it is situated 10 mm above this plane. The middle portion of the Eustachian tube, or isthmus, is not sharply constricted, but is relatively long, with gradual widening at each end, forming the aural and nasopharyngeal orifices. The diameter of the isthmus in the adult is 1 to 2 mm; but in the infant it is somewhat larger. The mucosal lining of the cartilaginous portion is similar to that of the nasopharynx and contains mucous glands. The mucosa in the protympanic portion of the Eustachian tube is similar to that of the middle ear and contains both mucus producing elements and cilia. The function of the Eustachian tube appears to be two-fold: clearance of secretions and ventilation of the middle ear.

Usually the Eustachian tube is closed, but it opens during swallowing, yawning and sneezing, permitting the air pressure in the middle ear to equalize with atmospheric pressure. This opening mechanism is muscular and involves the nasopharyngeal orifice. The tensor palati is the only muscle related to tubal function, and closure is the result of relaxation of the tensor palati with passive approximation of the tubal walls.

The tensor tympani muscle originates from the cartilaginous portion of the Eustachian tube and the adjoining part of the greater wing of the sphenoid, as well as from the bony canal in which it is contained. It passes backward through the canal, enters the middle ear as a slender tendon which bends laterally around the processus cochleariformis and is inserted into the neck of the malleus and into the tympanic membrane directly.

In 1970, A. J. Lupin demonstrated by dissections that the tensor palati and tensor tympani muscles have fibers that intermingle. Both of these muscles have fibers originating in the area of the greater wing of the sphenoid near the spine, and also both have fibers originating from the Eustachian tube. There is intermingling of these fibers on the undersurface
of the sphenoid spine. The common development of these muscles is from
the mandibular arch with a common nerve supply from the mandibular
division of the trigeminal nerve. There are three branches to the three
divisions of the tensor palati muscle and one to the tensor tympani. These
two muscles are, in fact, continuous and, indeed, the tensor tympani muscle
is a continuation of the muscle fibers from the anterior portion of the tensor
palati with additional slips from the base of the sphenoid bone and from the
periosteum lining its semi-canal. The contraction of the medial part of the
tensor palati muscle unrolls the tubal cartilage by depressing the lateral
lamina, thus opening the nasopharyngeal orifice of the tube.

It is postulated here that the contraction of the tensor palatini is accom­
panied by a similar contraction of the tensor tympani muscle, thereby
exerting a slight medial or inward movement of the tympanic membrane.
This combined movement thereby acts as a “pump” mechanism to help
clear the air or “push” secretions out of the Eustachian tube into the
nasopharynx. Tympanometry measurements of the ear were performed at
-150 mm of water pressure thereby causing the tympanic membrane to be
pulled laterally into the external auditory canal. Upon voluntary contraction
of the tensor palati and tympani muscles, the pressure in the outer ear canal
increased by 3 to 5 mm, proving a medial motion of the tympanic mem­
brane thereby substantiating this “pump” mechanism. The function of the
tensor tympani muscle is therefore to provide 3 to 5 mm of positive pressure
in the middle ear and Eustachian tube upon the opening of the Eustachian
tube by the tensor palati. This pressure need not be more than that when
one realizes the minute movements of the tympanic membrane which
produce hearing. Air is thereby forced out of the Eustachian tube upon
swallowing and new air then rushes into the tube and middle ear, providing
a continuous cycle of high oxygen containing air in the middle ear. The
function of the tensor tympani muscle has always been a mystery. Clearly
now the function and importance of this muscle has become elucidated.

In patients with cleft palate there is no opposing action of the opposing
tensor muscles, the Eustachian tubes remain closed and the pump mecha­
nism does not function. If the Eustachian tube fails to open, the middle ear
cannot be adequately aerated and fluid usually accumulates in it.

Identification of Serous Otitis Media

Pneumatic otoscopy as noted by J. Northern in 1976 is the accepted final
criterion and absolute reference in the evaluation of ear disease. However,
physicians vary tremendously in ability to interpret their observations during
otoscopy and pneumotoscopy. A case history can be unreliable, and hearing
tests conducted during infancy are rather complicated. Consequently, an
eyear recognition of any disturbances in early childhood mainly depends
upon an improved diagnosis, with the aid of a microscope.
In regard to audiology, traditional hearing tests are not sufficient to identify otitis media, and changes in hearing sensitivity do not necessarily relate to changes in the otologic disease process. Fifty percent of 408 ears with serous otitis media would have passed as "normal" on school hearing tests conducted at the accepted screening level of 25 dB HL.

**Acoustic Impedance Measurements**

The most sensitive screening technique for identification of serous otitis media is tympanometry. Tympanometry is defined as an objective technique for measuring the compliance (or mobility) of the tympanic membrane while varying air pressure in the external auditory canal. Tympanic membrane mobility is of particular importance since almost any pathological condition located on, or medial to, the eardrum will influence its movement. Tympanometry, compared to otoscopy, is totally objective, and eardrums noted to have normal mobility by pneumatic otoscopy examination can be shown to have abnormal mobility with tympanometry. Acoustic impedance contributes especially meaningful information about middle ear disease in cleft palate children and should be a routine part of their evaluation. In 1975, F. Bess, H. Lewis and D. Cieliczka noted that in cleft palate children identification of middle ear problems is possible with impedance when the audiometric and otologic examinations were normal.

**Management of Otologic Problems**

The treatment of infants with cleft palates must be directed toward the correction of effects as well as causes. Before repair of the secondary palate, elimination of middle ear fluid and aeration of the middle ear are the responsibility of the otologist and can be accomplished by myringotomy, aspiration and insertion of tympanostomy or pressure equalization tubes. The insertion is usually made in the anterior superior or inferior quadrant. The middle ear is then aspirated with a suction tube and the PE tube inserted.

This procedure should probably be performed soon after birth and close follow-up is necessary at least every two months. When spontaneous extrusion of tubes occurs, repeat myringotomy and reinsertion should be carried out. During the first year of life, these infants ordinarily have one or more reconstructive procedures on the lip or primary palate requiring general anesthesia. This provides an excellent opportunity for re-evaluation of the tympanic membranes, by means of the operating microscope, and for reinsertion of tympanostomy tubes if necessary. Infants should probably sleep in the prone position; when awake and supine, it may be helpful for the head and neck to be kept elevated at least 20° above the horizontal.

The tympanostomy pressure equalization tubes should be reinserted as many times as necessary until adequate functioning of the Eustachian tube is
demonstrated by a well-pneumatized middle ear upon repeated examinations. Occasional autoinflation of the middle ear may be performed to maintain the pneumatized middle ear. It is only continuous and meticulous attention to the aeration of the middle ear which will prevent serious and permanent otologic disability, and as long as air is there, I am not concerned about early closure of the palate.

**THE PULLEN PLUG**

Fred Pullen has devised a plug to allow children wearing tubes to swim, and in Florida swimming is important. He noted in 1978:

A high molecular weight polymer plastic cork (Richards Manufacturing Company, Memphis) which allows air but not liquids to pass in, is inserted into the P.E. tube. These little patients can then go swimming without fear of otitis media from getting water in the middle ears. This plug has been used for two years in over 200 cases with only one infection.
Thus, it can be said that cleft palate infants have an extremely high incidence of fluid in the middle ear, and whether this is due to the cleft in the normal tensor mechanism, abnormal tube anatomy or the relatively restrictive and compromising craniofacial relations, certain points become clear:

1. If fluid in the middle ear is ignored, hearing loss is increased, especially in low frequencies with inability to hear hypernasality. (Deaf children often speak with hypernasality.) The hypernasal cleft palate speech, not being detectable to the patient, reduces his chances of correction, even after surgical production of a competent velopharyngeal sphincter, thus setting up a **vicious cycle** for the speech therapist.

2. Early examination under the microscope, myringotomy, suction of accumulated fluid and insertion of tubes are important.

3. Repeated examinations under the microscope during the cleft surgery and reinsertion of tubes when necessary are essential.

4. As long as aeration of the middle ear is maintained, there is no hurry for palate surgery as far as hearing is concerned.

5. Craniofacial growth is probably responsible for improved tubal function, and therefore time is an ally if aeration is maintained.

6. It is possible that freeing, uniting and retrodisplacing the levator is the part of the surgery that benefits the Eustachian tube function.
6. Anesthesia in Clefts
(Gas, Tubes and Gags)

WITH SECTIONS BY S. MACMAHON
AND A. FREEMAN

ANESTHESIA IN CLEFT LIP

In ancient times cleft lip was treated by pinning of the pared edges, which never took more than a minute or two. When dentist William Thomas Green Morton introduced general anesthesia in 1846 at the Massachusetts General Hospital, Boston, it was soon used for these quick lip closures. John Snow reported giving ether for a lip repair in 1847 and by the time of his death in 1858 had administered chloroform 147 times for this operation, mainly for Mr. Fergusson of King's College Hospital. Most of the patients were infants between 3 and 6 weeks old. The infants had rested in Mr. Fergusson's lap, and according to anesthetist Snow:

In a few cases of strong children, in whom the bleeding is rather free, the breathing gets embarrassed, and Mr. Fergusson turns the face of the child downwards for a moment to let the blood run out of its mouth. . . . The effects of chloroform pass off very quickly in infants, and it is not often that they last till the operation of harelip is finished, short as that operation is.

Within a year of its presentation in Boston, ether anesthesia was used by Dieffenbach, in the fall of 1847. His stamp of approval had been awaited by the continent of Europe. Over the next 100 years anesthesia for cleft lip surgery progressed fantastically. The highlights were the change to ether, Magill's intubation, Ayre's T tube and Dott's mouth gag. Since all these devel-
opments had become well established by 1950, it was a shock to me at that time, returning from England, to find Claire Straith of Detroit operating without endotracheal anesthesia and thus resorting to mild sedation and local anesthesia. Such conditions were not conducive to careful design and meticulous execution of a lip closure. Yet there were times in Korea when my experience with Straith was put to good use in clefts. Fortunately this course is no longer necessary.

ANESTHESIA IN CLEFT PALATE

The cleft in the palate being less accessible, its surgery came later. Even then, the early palate surgeons—Monnier, von Graefe, Roux and Dieffenbach—did not have the advantages of general anesthesia. Von Langenbeck used ice to produce local numbing of the palate.

Gordon Jones of the University Hospital of South Manchester, England, in his excellent 1971 history of anesthesia for harelip and cleft palate, noted early isolated attempts with general anesthesia. The first report of a case that he could find appeared in the *Lancet* of 1850, stating that Mr. Gay, of the Royal Free Hospital, used chloroform in closing a bilateral cleft lip and hard palate in a 7-year-old boy.

The majority of surgeons, however, were against using general anesthesia for palate cases. In 1862 Fergusson of King’s College Hospital, who was quite happy with general anesthesia for lip clefts, declared that repair of the soft palate was one of the few operations in which chloroform could not be used. Sansom, an associate of Fergusson, wrote in 1866:

In cases of operation for cleft palate and such manipulations as require co-operation on the part of the patient, chloroform should be dispensed with or else given very sparingly. A few whiffs may be permitted so that the local sensibility may be benumbed. A gargle of iced water is the best local anaesthetic during the intervals of the operation.

John Snow was meeting the same opposition from other surgeons:
I assisted the late Mr. Avery by giving chloroform in two operations for cleft palate. The surgeon, however, much prefers to have the patient awake during this operation, when he can get his assent.

Even in America and at the Massachusetts General Hospital, J. Mason Warren, a friend of Morton's, affirmed that repair of cleft palate is one of the very few operations in which the use of anaesthetics is inadmissible. Under very peculiar circumstances, I suppose, ether might be administered, but not without some risk to the patient, and much embarrassment to the surgeon, from the constant flow of blood down the throat. It is necessary to wait until the patient is old enough to fully appreciate the importance of the operation and to submit patiently to pain and inconvenience.

This fearful attitude was changed during the 1860's through the work of M. H. Collis at the Meath Hospital, Dublin, and Sir Thomas Smith of St. Bartholomew's Hospital and the Hospital for Sick Children in London. Their heated controversy in competition for priority probably speeded the acceptance of anesthesia.

In 1868 Smith presented a paper to the Royal Medical and Surgical Society which was reported by a correspondent of the *British Medical Journal*:

The author's object in presenting this paper was to communicate to the Society a plan of operating on clefts of the palate, applicable to all who suffer from the deformity, but especially to children. The chief novelty in this proceeding was that chloroform could be employed. A painless and speedy operation could therefore be performed, and that with more precision and a greater prospect of success... while from the painless nature of the operation, the cure of cleft palate could be effected in children, to whom formerly the benefits of staphylorrhaphy were virtually denied.

In the same month a letter was published in the *British Medical Journal* from Collis:

Sir,—let me call the attention of Mr Thomas Smith to the *Dublin Quarterly Journal*, vol. XLIV, p. 345, by which he will see he is anticipated in this improvement... I have used it (chloroform) in all my palate operations for two years and a half... I believe, I was the first to operate with success on young children, and the first to use chloroform.
This suggested that Collis first used chloroform in cleft palate surgery in 1865 and, in support of his claim of having preceded Smith, the *Dublin Quarterly Journal of Medical Science* editor wrote in 1867:

> It is now fully established that chloroform can be given in these cases. Mr Collis gives it habitually and has been thus able to operate with success on very young children. The danger from chloroform is no greater than in any other operation and the relief from pain and from subsequent shock and depression is of the greatest importance. Of the advantages of early operation as regards the patient’s education, it is needless to speak.

In 1912 J. Berry and T. P. Legg recorded the difficulties being experienced at that time:

> The anaesthesia should be deep enough to abolish sensation, but not to do away with cough reflex. . . . The most suitable anaesthetic is undoubtedly chloroform. . . . We have sometimes employed ether for induction, but the tendency to the secretion of mucus and saliva, as well as the increased venous congestion caused by the ether . . . is apt to be troublesome. . . . [Chloroform] should be given through a Junker’s tube inserted either into one nostril or preferably at one corner of the mouth. . . . An experienced anaesthetist will often be able to lend a hand with the sponging, and . . . if the bleeding be unusually free, . . . or if vomiting occur, . . . it may be advisable to suspend the operation for a short time while the child is turned on its side.

Berry and Legg concluded, and with feeling:

> The difference to the surgeon, between doing a cleft palate operation with a thoroughly experienced anaesthetist and an inexperienced one, is the difference between pleasure and pain!

**PATIENT’S POSITION**

By 1874 Edmund Rose of Zurich was presenting a new position for the patient during the administration of general anesthesia while operating in the mouth. Rose placed the patient on the back on the operating table with the head hanging over one end while the foot end of the table was raised about 12 inches above the horizontal position. Thus blood collected in the nasopharynx could be sponged and, in later times, suctioned out instead of allowed to run down into the larynx and esophagus. This position became popular and was still being used and taught when I
interned with Ladd, Gross, and MacCollum at Harvard in 1944. In fact, Dr. Ladd sat down and operated with the baby’s head in his lap, as did Blair, Veau, Kilner and Wardill, among others.

**EXPOSURE**

Meanwhile, mouth gags were being designed to improve exposure and later were to assist the anesthetist. Gags by Lane, Rose, French and Doyen-Jensen simply held the mouth widely open, requiring a tongue stitch to maintain an airway and a view for the surgeon.

Thomas Smith’s 1868 gag incorporated a tongue depressor, as did gags designed by Whitehead, Collin, Geffer and Mahu. Berry and Legg warned of the dangers of obstructing the airway with the tongue depressor.
Some of the gags began to get more complicated and, incidentally, to look more like the gags of today. Trélat’s, Lexer’s modification of the Whitehead gag and the fantastic appliance of Edoardo Bassini are shown:

Then, to assist the anesthetist, both Mason and Doyen equipped their gags with thin metal tubes fixed to the gag blades through which the anesthetic vapors could be insufflated into the oropharynx.

**Dott**

Norman W. Dott of Edinburgh started out as an engineer but, convalescing from a motorcycle accident in which he suffered bilateral tibial fractures, became intrigued with medicine. He took his medical degree and for a time was involved in pediatric surgery before he became the first professor of neurosurgery at the University of Edinburgh. During his pediatric experience he did cleft palate surgery and turned his knowledge of engineering toward the construction of a mouth gag that forms the basis of all gags popular today. It was C shaped, with one side open, and incorporated a tongue depressor.

**Kilner**

One version of the Dott gag incorporated an anesthetic tube in the tongue depressor. Kilner added a spring coil around the top to hold sutures in perfect order prior to tying, in keeping with his tidy surgery.
Dingman

In 1962 Dingman and Grabb reported on a modification of the Dott-Kilner gag as a closed rectangular frame with bilateral alveolar retractors and a tongue blade sliding through a ratchet. The tongue blade supported the gag inferiorly and held an endotracheal tube over the tongue. To this had been added bilateral side retractors which hooked the lips near the commissures to pull the cheeks laterally out of the way. At about the time of this gag’s introduction, Reed Dingman, accompanying me on a Caribbean work trip, asked:

What is happiness?

and when none of us came up with the appropriate answer, he continued:

This mouth gag for a surgeon operating on a cleft palate!

He then presented us with one of his brand-new gags, and it has been standard equipment for cleft palate cases at the University of Miami School of Medicine ever since.

Over the years I have experienced a couple of difficulties with this gag, the major problem being the lack of adaptability of the rigid rectangle to fit the irregular alveolae.

Miami modification

The majority of severe cleft palate patients have abnormal spacing between the maxillary alveolar arches. When there is maxillary retrusion, collapse or a protruding premaxilla, it is difficult to fit both alveolar hook retractors on the irregular alveolae when these retractors are attached to a rigid rectangular frame. Once fitted, the purchase is often unstable because only one retractor can get good contact with an alveolus. The gag may slip midway through the operation. Then too, the frame does not allow the retractors to maneuver into an effective position in the presence of a severely projecting premaxilla. If it is forced on, it traumatizes the premaxilla, or at least the prolabium. Resident David Slepyan was challenged to try to improve the gag, and he in turn found Jack Nestor, an ingenious machinist and sculptor, who used the Dott-Dingman tongue depressor and ratchet arrangement with a
posterior bar to hold a pair of telescoping 360-degree rotating barrels. The anterior bar was divided to admit any projecting premaxilla. For more mobility the anterior segments were constructed to slide sideways through the lateral barrels, and these sliding anterior arms were capped with swivel-hook retractors which could be set at any angle to clasp the alveolae. The swivel-hook retractors were made interchangeable—one for infants, one for adults. The adult hook has a beveled radius to present a wedge which fits between the teeth to prevent slippage. A lock-stop screw controls outward rotation of the swivel-hook retractor. This completed the total adaptability the gag to near-ecstatic proportions for any irregularity of the alveolar arch in patients of any age.

The Dingman side cheek retractors can be installed, yet in palate surgery it has been my experience that the amount of vertical mouth opening needed for velopharyngeal surgery will sometimes burn the commissures. Thus one fears that the additional lateral pull for transverse opening, which for palate surgery is unnecessary, may cause even greater stretch burns.

The Miami gag modification, published in Plastic and Reconstructive Surgery in April 1977, can be obtained from Jack Nestor, 148 N.E. 29th Street, Miami, Florida 33137, upon request or from Padgett Instruments, Kansas City, Missouri.

ENDOTRACHEAL ANESTHESIA

The most monumental advance in cleft lip and palate anesthesia came with the introduction of endotracheal anesthesia. This was accomplished by a thin insufflation catheter passed by direct vision into the trachea. Ivan Magill first used the method on infants in 1921 and first intubated a cleft palate case for Harold Gillies at Great Ormond Street in 1924.
Sir Ivan Magill’s story is fascinating. He was graduated from Queen’s University Belfast, Ireland, and near the end of World War I arrived at the Plastic and Jaw Unit at Queen’s Hospital, Sidcup. One look down the wards of bandaged broken jaws and wounded faces was warning enough that anesthesia here would be no picnic. At that time ether vapor was administered through a gum elastic catheter with the aid of an electrically driven pump to create a positive pressure to prevent blood from trickling into the trachea. The surgeon got the blast of the patient’s ether-laden expirations and was often enveloped in a spray of blood. Constantly, Harold Gillies, half asleep from Magill’s ether, would growl:

Maggie! You seem to get this ether in here jolly well. Why can’t you take it out again?!

Magill tried two tubes, then one wide-bore single rubber tube which he found could be passed blindly into the trachea through the nasal route when the head was in the position affording the freest nasal airway, as in “drinking a pint.” In 1925 in Paris, at the first truly International Congress of Plastic Surgeons, Magill demonstrated his nasal intubation on a cadaver. Later, at the request of a French surgeon, he intubated a patient. The surgeon, accustomed to a porter with a bottle of chloroform crouched over a cyanotic, coughing, struggling patient, took one look at his patient lying quietly with Magill’s tube in place and cried out that his patient was not breathing, then that he was dead! Whereupon Magill assured him all was well.

Evidently, French surgeons were not totally convinced. Visiting Paris in 1948 I watched Jacques Récamier do a Veau cleft palate procedure at l’ Hôpital St. Michel. With some difficulty from behind a modified gas mask worn to prevent the chloroform from anesthetizing himself, Récamier was forced to pause constantly to allow the patient’s cyanosis to pass.

Meanwhile, Magill continued to improve the technique of endotracheal anesthesia. He placed a battery in the handle of the laryngoscope to provide a light without wires and wall plugs, a slot in the laryngoscope barrel to accommodate tubes, mineral-
ized rubber tubes smoothly beveled at one end and in 12 sizes. He also designed wide-angled metal tube connectors for oral intubation and acute-angled connectors for nasal intubation, in order to keep the anesthetic apparatus fitted snugly to the profile and out of the way of the surgeon.

Magill found that in cleft lip and palate cases laryngeal edema was not necessarily a danger. The real difficulty was in developing a tube thin enough for an infant that would not collapse. Finally, in 1932 he turned to a metal coil covered with thin rubber cemented to a $\frac{3}{4}$ inch piece of standard rubber endotracheal tube beveled at one end. The other end was attached to a Y metal connector to reduce rebreathing. This principle preceded Ayre’s T tube. Such a flexible, uninkable, armored tube could withstand the pressure of the tongue depressor in the Dott mouth gag without collapse and facilitated oral intubation with the tube out of the surgeon’s field. The anesthetist could also relax as his tube was safely stabilized by the tongue depressor. A pharyngeal pack or a Magill inflatable cuff was used to prevent blood and secretions from slipping by the tube down the trachea. Once Magill had his intratracheal tube in position and the patient sleeping peacefully, he would get out some string, cork, tubing and a pocketknife to experiment in constructing gadgets for his anesthetic apparatus with much the same concentration he tied flies while trout fishing the Test River.

By 1936 Digby-Leigh and Fitzgerald of Montreal were using Magill’s tubes, as was Ayre for Wardill in 1937 and Gillespie for Kilner at Elizabeth of York Hospital for Children at Shadwell in 1939.

Ayre

Red-haired Philip Ayre, anesthetist to Babies’ Hospital, Newcastle upon Tyne, was a postoperative unilateral complete cleft of the lip and palate individual with only a reasonable repair himself. He found on cleft lip and palate cases that the Magill wide-bore rubber catheter through the mouth into the trachea worked satisfactorily except for the suboxygenation when nitrous oxide was being used in babies and the difficulty in adjusting the amount of rebreathing. No matter how small the rebreathing
bag, there was always too much "dead space". Because of anoxemia and excessive rebreathing, the condition of cleft babies showed marked deterioration within a short time. Ayre was in a state of desperation as he explained in 1937:

In an endeavour to remedy this distressing state of affairs (and spurred on by the caustic criticisms of a candid surgeon!), the writer sought to devise a method by which the endotracheal technique could still be utilized without the drawbacks associated with nitrous oxide and excessive rebreathing. . . .

Briefly, the apparatus consists of a T-piece which is connected by a short piece of rubber tubing and a Magill angle-piece to a wide-bore rubber catheter previously inserted into the trachea. Through one limb of the T-piece oxygen and ether vapour is delivered from a Boyle or other continuous-flow apparatus. The other limb remains open to the outside air; for convenience, a short piece of tubing may be attached and allowed to hang down beneath the operating towels. A strand of fine gauze, fixed with adhesive strapping close to the open end of the latter tubing, will wave to and fro with the patient’s respirations, thus serving as a useful indicator to the anaesthetist. . . . The excellent colour and quiet, natural breathing of the babies have convinced us that oxygen and ether vapour, administered by the T-piece method, is the anaesthetic of choice for all hare-lip and cleft palate operations on babies and young children. . . . The post-operative convalescence is remarkably smooth and free from anxiety. Last, but by no means least, comparative peace now reigns in an operating theatre formerly the scene of many sanguinary battles!

A final point of finesse came when in 1954 R. M. Davis modified the Dott tongue depressor with a slot to accommodate the Magill tube.

As Gillies was doing very few primary cleft lip and palate operations, I took the opportunity to visit Wardill in Newcastle and there observed Ayre using his happy T tube. Once a fortnight, at Lord Mayor Treloar Children’s Hospital, Alton, I had a chance to observe Kilner and Peet operating on lips and palates. Their anaesthetist was John Hunter, who at 240 pounds was the Friar Tuck of anaesthesia. It was fascinating to see huge and jovial John turn an infant fuzzy with N₂O, taking it down with Vinethene followed by ether drip prior to intubation under direct vision. He carried the babies through the operation on pure gas-oxygen-ether with the Ayre open T tube. His anaesthesia was smooth, the baby almost never turning blue or coughing blood.
in the surgeon’s face. The operation by Kilner or Peet was so organized that Hunter could intubate, leave the room, return in one hour by the clock and, without even looking or asking, it seemed, pull his tube. By 1949 he had anesthetized over 2,000 infants without a mortality.

During the last 20 years the use of controlled intermittent positive pressure respiration, with or without the use of muscle relaxants, has become popular for pediatric cases. Anesthetic agents in cleft surgery have changed from chloroform to ether and now to halothane. Wallbank has shown that in infants and children up to 2 years of age the doses of epinephrine required to aid hemostasis may be safely used in the presence of small concentrations of halothane.

**FINDING THE VEIN FOR I.V.**

In 1977 Robert Woolf of Salt Lake City suggested an aid to anesthetists attempting to place a needle in infants for administration of intravenous fluids during surgery. If a fiberoptic light is placed under the hand, the arcade of vessels, not easily visualized through the skin in fat babies, will be vividly presented.

**MIAMI ANESTHETISTS**

It has been my good fortune to have two excellent anesthetists for the cleft babies. Their thoughts and tricks are important.

*MacMahon*

F. Stan MacMahon received his medical degree at University College, Dublin, Ireland, trained at the Anesthesiology Center, Copenhagen, and then became a journeyman anesthetist around the world, ending up in the Bahamas. For many years he gave superb anesthesia for the cleft babies I operated on at Princess Margaret Hospital, Nassau. Imagine my elation when he decided to come to Miami and became Clinical Director of Anesthesia at the University of Miami School of Medicine, Jackson Memorial Hospital. Here is his contribution:
Ralph Millard and I have been operating together on patients with these problems for over sixteen years. Early in the series I learned that the virtuosity involved in the functional and cosmetic repair of these babies requires the anaesthetist to apply the art as well as the science of his profession. The surgeon demands a clear, uninterrupted operating field with normal, uncontorted anatomy; the anaesthetist must have control of the airway at all times. For both parties on either side of the ether screen to perform smoothly, such a mutual understanding is essential. Compromise is the essence of diplomacy, and, after a few initial skirmishes, Ralph and I signed a "Mutual Aid Pact" and have since worked in perfect harmony.

In attempting to describe the anaesthetic technique which we have evolved over the years, I am reminded of the Ninth Century Irish monk who, in a moment of anguish, wrote on the margin of his scholarly text:

Meisse oclus Pangur Ban,
cechtar nathar fria shaindán;
bith a menma-sam fri seilgg
mo menma cén im shaincheird.*

At least Gallarus had his cat for company and inspiration. . . .

Simplicity usually means safety. For this reason, a modified Jackson Rees system, delivering nitrous oxide, oxygen and halothane on spontaneous respiration and incorporating an artificial sigh, is utilised. During the mask induction it is important to recognise that airway management may prove difficult. The most common cause of obstruction (which, at times, may be complete) is what I refer to as "corking" of the palate by the tongue. When the cause is known, however, the remedy is indeed simple.

When the patient has reached a surgical plane of anaesthesia, laryngoscopy is performed. To avoid even mild trauma to the epiglottis, the author prefers to use an infant Mackintosh laryngoscope blade at first and, in 90% of cases, can obtain a clear view of the larynx. If a clear view cannot be obtained with this blade, then one progresses through the Roberts-Shaw, the Sheila Anderson and the Miller blades until a clear view is obtained. Personally, I never attempt to intubate these children unless I can see the larynx clearly. My reason for this is that I look on these cases as elective, and at no time should the child be jeopardized by injudicious attempts at intubation. Cole tubes cut to the appropriate length, with a Magill type catheter connector, are used. It is essential that the Magill connector lie flush with the chin.

*For the non-Gaelic speaking:

1 and Pangur Bán my cat,
'Tis a like task we are at:
Hunting mice is his delight,
Hunting words I sit all night.
thus providing an uninterrupted operating field. To achieve this, tubes of varying sizes and lengths should be at hand.

The introduction of the corrugated tubing between the Inglis valve and the Magill connector is a compromise reached between the anaesthetist and the surgeon. From the anaesthetic point of view, a measured "dead space" of 7 cc's is added to the system, but the flexibility achieved is of great value to the surgeon.

For cleft lip adhesion or closure, a solitary piece of plastic tape placed low down on the chin, combined with a pharyngeal pack, suffices to keep the endotracheal tube in situ. In cleft palate closure, the blade of the mouth gag serves the same purpose admirably.

Pulse, respiration, blood pressure, temperature and EKG are monitored at all times. Maintenance I.V. fluids are given. Hypothermia is avoided by the use of a warming blanket. Accurate measurements of blood loss are made by weight and calibrated catchment trap in the suction line. It is seldom that blood loss exceeds 10% of the estimated circulating volume; therefore, replacement transfusion is rarely indicated.

Positioning the child is of utmost importance to the surgeon, who sits at the head of the table. The extension must be maximal for cleft palate surgery, less extreme for lip repair. I prefer to obtain head extension by raising the whole body on a folded blanket, rather than by placing a folded towel under the shoulders. The work of breathing is decreased by this simple manoeuvre.

At the conclusion of the surgical repair, and following a careful pharyngeal toilet under direct vision, extubation is carried out in the surgical plane of anaesthesia. To avoid trauma to the surgical field, an oral airway is not used. Instead, a tongue stitch, combined with a lateral position, is utilised to maintain a clear airway until normal reflexes return. The insertion of the tongue stitch always seems to cause extreme anguish to the surgeon, but having signed our "Mutual Aid Pact," it is now performed with a minimal show of revulsion.

If it becomes necessary to administer oxygen to the patient before protective reflexes have returned, a simple trick incorporating the tongue stitch, mask and mask connector can be used. The tongue is pulled out by the stitch. The stitch then is passed through the mask aperture, and the mask connected to an oxygen supply. This is quite effective and technically easy.

When normal protective reflexes have returned, the baby is transported from the Operating Room to the Post-Aneesthetic Recovery Room. Here arm splints are applied to prevent damage to the operation site by thumb sucking. Some of the cleft lip babies will have been converted temporarily to complete mouth breathers by the surgical repair. Gentle traction on the tongue stitch may be required in the immediate post-operative period to
remind our little patients to open their mouths and yell a challenge to the world.

Many anaesthetists will ask why we go to so much trouble for these relatively short but challenging procedures. The answer is obvious some years later, when the eye can barely detect a scar on the lip, and the ear hardly detects a nasal tone. That, indeed, is sufficient reward.

Freeman

It has also been my good fortune to have another expert anaesthesiologist, Alfred Freeman, M.D., trained at Temple University, available at Variety Children’s Hospital to help in the care of these children. He is an extremely smart, dexterous and gentle man and has provided here the details of his approach:

Children’s hospitals, because they are relatively small and because their staffs work closely together, provide what I believe to be the ideal environment in which to treat cleft lip and palate patients. It is important to have personnel who are specifically fond of infants and children and who can provide the emotional support that these patients require. Needless to say, teamwork is the keystone to the overall successful outcome in the treatment of these children.

Infants under two weeks of age are usually intubated under deep general anesthesia to allow for an atraumatic intubation of the larynx. Anatomic problems imposed by the cleft palate lead to easy impaction of the tongue against the palate with subsequent airway obstruction. Soft tissue obstruction of the airway will occur earlier and be more pronounced if the anesthesiologist inadvertently presses his fingers under the mandible and causes increased pressure of the tongue against the palate.

Halothane with 50% nitrous oxide/oxygen is usually used for the induction and maintenance. As soon as the patient is unconscious, a plastic cannula is inserted percutaneously into a peripheral vein and an infusion of appropriate fluids started. Monitoring of the patient’s vital signs is started at the same time. A water mattress, warmed to 37°C, helps maintain the patient’s body temperature at a normal level. When an adequately deep plane of anesthesia is reached, direct laryngoscopy with oral intubation of the trachea is performed. It is advisable not to use muscle relaxants in infants unless the anesthesiologist is experienced and has ruled out the possibility of anomalies which make direct visualization of the larynx difficult. When difficulty in exposure of the larynx is anticipated, it is wise to have the patient deeply anesthetized, saturated and breathing spontaneously at the time of laryngoscopy. During laryngoscopy, it is possible to
insufflate the anesthetic into the oropharynx by means of a mouth hook to lengthen the duration of deep anesthesia and allow exposure of the larynx in a less hurried atmosphere.

The choice of laryngoscope blade varies from one anesthetist to the next. My personal preference has been the #1 Miller Blade up to six months of age, the #1 Flagg Blade from six to 18 months of age, and the #2 Miller or Flagg Blade for over two years of age. The #2 and #3 Mackintosh Blades also are useful on occasion.

The correct size endotracheal tube is that which will allow a slight leak of air around the tube when an airway pressure of 15 to 20 cm. of water is exerted, but no leak at pressures lower than that. It has been my experience that there have been no cases of post-endotracheal croup when an air leak at these pressures has been assured. Children who have a history of having had croup in the past (whether viral or post-anesthesia) and those with known subglottic stenosis, can be expected to accept a much smaller endotracheal tube than other children of the same age. The proper length to cut the tube will have the upper end of the tube at the level of the teeth or gums, while the bevel of the tube is in the distal third of the trachea, about 1–2 cm. above the carina. A right angle Magill adaptor inserted into the tube and directed caudad can be fixed to the lower lip without distortion of the corners of the mouth.

Recently, pre-formed polyvinyl chloride endotracheal tubes have been available commercially as shown. The pre-formed curves are designed to allow the endotracheal tube to lie flat against the patient and also eliminate the possibility of the endotracheal adapter becoming detached from the tube during the operation.

Where pressure of the mouth gag would threaten to kink (occlude) the endotracheal tube, a latex rubber metal-armored endotracheal tube of branchial length should be used. The ring of tape around the tube indicates the point of fixation to the lower lip to avoid endobronchial intubation. The extra length of the tube permits the anesthesiologist to position the remainder of the anesthetic apparatus at a greater distance from the surgical field.

After intubation an orogastric catheter should be passed to empty the stomach of gas and secretions and the catheter removed before preparation of the surgical field.

For cleft lip closure a small posterior pharyngeal pack is inserted deeply into the pharynx to help stabilize the endotracheal tube. For the cleft palate closure the use of the Millard-Dingman gag allows the surgeon easy unencumbered access to the field while stabilizing the endotracheal tube, without the need of a pharyngeal pack. It is important to properly position the patient to allow for the best possible exposure. For cleft palate repair, the entire body is raised by means of a foam rubber mat, 8 cm. thick, allowing
the head to drop back and be fully hyper-extended into a soft, round head support. This position provides good exposure while allowing blood to run downhill away from the larynx towards the nasopharynx from which it can be removed by suction.

To prevent injury to the eyes, the lids are taped closed by 1” cellophane tape, or ocular lubricant preparation is instilled into the eyes. The use of a 4 X 8 sponge held firmly over the proximal edge of the eye tape during the surgical prep will prevent any of the prep solution from running under the edge of the tape and into the eyes.

During maintenance of anesthesia the patient is allowed to breathe spontaneously. Gentle augmentation of respiration by the anesthesiologist’s hand on the reservoir bag is used since moderate depths of anesthesia are required to prevent coughing on the endotracheal tube should it be moved.

In recent years, I have found that one of the newer lightweight disposable carbon dioxide absorption circle systems offers the best anesthetic delivery. If a small extension of latex rubber is used on the Magill adaptor, this system provides minimal deadspace, low resistance, easy assistance of respirations and adequately humidified gases without the use of additional equipment. Scavenging of waste anesthetic gases is also more efficient with this type of circle system.

The goal of the anesthesiologist should be to maintain (1) clear unobstructed airway devoid of secretions, (2) adequately deep respirations, (3) a stable satisfactory level of anesthesia, (4) normal body temperature, (5) adequate fluid balance and (6) proper blood volume. Careful monitoring by the anesthesiologist will indicate proper management to achieve these goals.

There are differing opinions as to the management of the anesthesia upon termination of the operation. Many of these patients will be converted to mouth breathers as the result of the cleft palate repair and must therefore re-learn how to breathe to avoid soft tissue obstruction. It has been my experience that the lowest incidence of post-operative problems is encountered if the patient is extubated after awakening from anesthesia.

At the conclusion of the operation, the oropharynx and anterior nares may be carefully and gently suctioned and a small bite block (made from short lengths of tongue depressors wrapped with 1” adhesive tape) is inserted a short distance between the teeth and gums and taped securely alongside the endotracheal tube. The patient is then turned to the lateral position, taken to the post-anesthesia recovery room lightly anesthetized and allowed to breathe humidified oxygen by T-piece apparatus until awake. When the patient is able to open his eyes, the oropharynx (and trachea if necessary) is gently suctioned and the endotracheal tube removed. This technique does not require the use of a tongue stitch for traction. The use of sedative drugs in the post-operative period is discouraged until the patient can avoid soft tissue obstruction when asleep.
PREPARATION FOR SURGERY

As I noted in the *Nursing Clinics of North America* in 1967,

The patient should be relatively free of upper respiratory infection and possess a hemoglobin level of at least 10 Gm. He should be prepared for a postoperative routine by being fed with a spoon, cup or the Asepto syringe by various people using a good variety of liquids. Two weeks prior to the palate surgery, the mother can use a white cap and gown to prepare the child for hospital nurses [MacCollum].

In the older children and adults, in whom bleeding can be more extensive, a type and crossmatching for 500 ml. of blood is advisable, but seldom used.

POSTOPERATIVE CARE

Again as remarked in 1967,

It is important to remember that this baby has been used to breathing with a hole in the roof of his mouth. After surgery, this hole is closed and a new pattern of breathing is now necessary. The adjustment may not come easily. In addition, there may be some oozing of blood from the operative site. The baby should lie face down and suction should be available at bedside with alert nursing attendance for several days, particularly in the first ten hours.

Here again elbow restraints are applied to prevent the patient from inadvertently disrupting the healing palate. These restraints are maintained three weeks at home with short periods of controlled freedom. Because of
unavoidable and continuous contamination, a systemic antibiotic may be used for three to five days postoperatively.

With the aid of the Asepto syringe or cup, the baby is fed liquids the first two weeks following surgery. Each feeding is followed by a drink of water which serves as a mouthwash. During the next two weeks the baby receives a soupy diet. At one month, a regular diet excluding hard food such as toast and rock candy is begun. No straws are allowed for a couple of months from the time of surgery.

MORE DETAILS ON FEEDING

As an intern at Boston Children’s Medical Center, I learned from Donald MacCollum the trick of using a special feeding technique for cleft patients. As described in 1967,

The baby is held in the nurse’s arm with his head held upright in her left hand. Formula is fed by bulb compression through a 10cc Asepto syringe with a 1\(\frac{1}{2}\) inch rubber catheter extension. The catheter is slipped over the baby’s tongue and the formula is fed as the baby is able to take it. As soon as the baby has adjusted to this routine the mother is instructed in the technique. Once mother and baby are prepared by the nurse, they are allowed to go home.

THE BIFID NIPPLE

Kenneth Adisman of New York University has a 1957 patent on a Dow-Corning Company Silastic bifid cleft palate nipple designed for the infant whose sucking reflex is absent or minimal. As noted in *Cleft Lip and Palate*:

The liquid flows from the bifid nipple in a stream through openings directed laterally against the cheeks. The flared teat head, which serves to partially close the cleft opening, is compressed by tongue action against the maxillary process, expelling the liquid from the nipple. This bifid type of nipple construction enables these infants to suckle while in the usual feeding position without fluid entering the nasal cavity through the cleft opening. . . . The nipple is made in two sizes to conform to different shaped maxillary processes. The nipples may be provided with suitable venting means which will allow air to trickle into the bottle as liquid is withdrawn . . . thereby reduc[ing] the suction requirements for a satisfactory flow of liquid.
A NEW SQUIRT

Here are Betty Jane McWilliams’ thoughts on cleft palate feeding:

It is perfectly true that the babies usually do not gain as well as would be hoped if the mothers are left completely alone to develop a feeding plan by a trial-and-error method. Unfortunately, we reluctantly conclude that very little assistance is provided in the average new-born nursery and that the nurses are often quite uncomfortable about feeding the infants and about providing instructions to new mothers.

We were using a more complicated feeding system until one evening at a tiny neighborhood drugstore I picked up an Evenflo disposable nurser, plastic bags, and nipples, took them home and experimented. Next morning, Pediatrician Paradise was teaching a mother our old technique. I squirted him with the new bottle; he took it, tried it, and turned to the mother and said:

"Forget everything I've said. We'll use this," and we have ever since.

The system that we found successful for most babies with clefts is very simple indeed:

1. Hold the baby in a sitting position.
2. Use a plastic shell with openings in the sides and with no bottom.
3. Use disposable bags to hold the formula.
4. In the beginning, use a nipple for premature infants.
5. Be sure that the opening is a cross-cut. Never enlarge the hole. The cross-cut permits the milk to flow but also provides a good mechanism for preventing the milk from gushing into the baby's mouth and causing him distress.
6. The mother should learn to insert her fingers into the side slots and the bottom opening so that she can gently express the milk into the baby's mouth—being careful not to provide more help than the infant needs.
7. Burp the child frequently.
8. Attempt to complete the average feeding in no more than a half hour.
9. Follow a normal schedule for the introduction of solids including finger and table foods.

Evenflo disposable nursers (a) are not on the market now but may be ordered in quantities from Questor Juvenile Products, Ravenna, Ohio. In addition, Playtex nursers (b) may be used quite well by enlarging the slots of the sides so that a finger may be inserted. Both of these techniques permit the mother to use equipment that does not look "special" and that is much cheaper than are many of the special devices.
It has always been my feeling that well-planned, careful surgery and simple, practical postoperative precautions as noted are sufficient to achieve a well-healed palate. When in 1971 Sam Pruzansky wrote his approval of Cleft Craft, Volume I, he expressed disappointment in the chapter "Postoperative Care," mentioning that his wife, Donna, was an expert in this area. Although our babies were healing and gaining weight, long ago I learned to heed Sam's cry!

For 10 years Donna Pruzansky at the Abraham Lincoln School of Medicine, Chicago, had been involved in a nursing outreach program which arranged for

the visiting nurse, trained in the comprehensive needs of the child with an oral-facial cleft, to play an important role in crisis intervention by preventing potential feeding problems and assuring adequate nutritional intake during the critical neonatal period. This entails instruction of the parents, the hospital staff, and follow-up contact with the mother, and including home visits where required.

These were some of her suggestions:

Only minor modifications of the usual techniques used in bottle feeding are required to feed most CLP babies. To begin with, either a premie nipple or a lamb's nipple (DAVOL, Inc., Providence, R.I.) is preferred. The lamb's nipple is necessary in the case of a wide unilateral cleft lip and palate since the broad nipple will not slip into the cleft, thus allowing the infant to bite down against the alveolus and use the tongue to strip the milk from the nipple. This process obviates the need for feeding obturators.

The opening should be enlarged to facilitate flow. To do this, the nipple is turned inside out and a cross-cut is made with a scalpel or razor blade. Enlarging the opening in this manner, rather than merely enlarging the diameter of the hole in the nipple, allows the infant to control the flow of milk. This permits coordination of sucking, swallowing, and breathing and establishment of a normal feeding pattern. Adequate flow is critical to avoid unduly prolonged feeding periods which will exhaust the infant before he can complete sufficient intake. In contrast, nipple openings enlarged by poking holes with a hot needle result in flooding the infant with milk and necessitate a continuous insertion and withdrawal of the nipple to allow the baby to breathe.

There is no need to hold the infant upright, aim the milk at the cheek, or burp more frequently, as is so often advised in manuals for parents.
Helpful Hints

Since the lamb’s nipple does not fit onto conventional bottles with screw tops, it is necessary to improvise. A most effective arrangement is to cut the base from a conventional nipple and use it as an adapter.

The anxiety engendered by the malformation often discourages the mother from breast feeding. In our experience, successful breast feeding is contingent upon a number of variables that include the type and severity of the cleft and the dedication of the mother. Previous experience helps. Even then, it requires a longer period of time for each feeding.

The problem of feeding an infant with a cleft is complicated by the Robin syndrome where the maintenance of an adequate airway is compromised by the micrognathia and glossoptosis. The introduction of the nipple and milk further aggravates the problem.

Depending upon the severity of the condition, bottle feeding may be possible with certain modifications in posturing to maximize the airway. This can be achieved in some instances by holding the infant in an upright position with manual assistance to guide the mandible forward.

Feeding can be accomplished with the infant in the prone position on the nurse’s lap with head extended. For the severely compromised Robin syndrome, nasogastric feedings may be required.

Although the foregoing is limited to the neonatal period, parents can be assured that baby foods and table foods can be introduced to the CLP baby at the same age as for the non-cleft child.

It is an obvious but often overlooked fact that the baby with a cleft is first of all a baby. Like all babies, they may spit up, have allergies and a myriad of other problems unrelated to the cleft. Therefore, when problems do arise, it is important to examine the whole child and not focus only on the cleft.