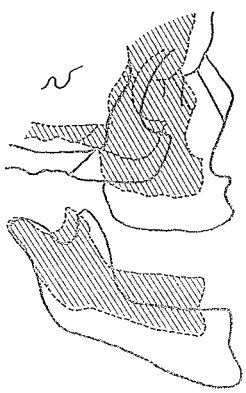
3. *Growth in the Normal and the Cleft Palate Patient and the Effect of Surgery on Growth*

LEST 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 phylogenetic 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

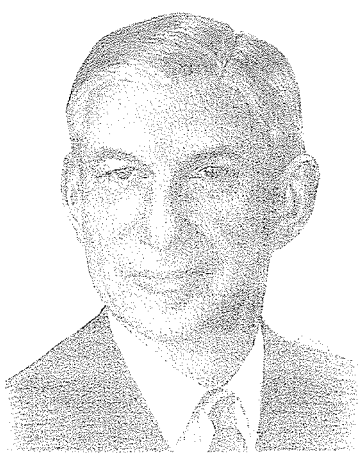
As pointed out by David O. Maisels of Liverpool in his Kay-Kilner prize-winning essay in 1966, a complete alveolar cleft will be present by the end of the eighth week of gestation. According to Scott, growth of the interorbital cartilaginous system is responsible for much of the early forward and downward 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 uncleft side, taking the nasal tip with it, so that the septum is bent and the alar arching 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 Sarnat

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

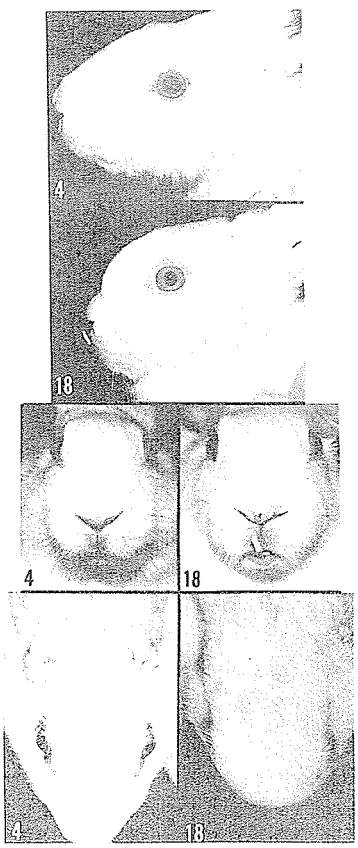
Primary endochondral centers . . . in the skull are the sphenoethmoidal and spheno-occipital synchondroses, and the septoethmoidal and septo-presphenoid joints, and the mandibular condyle. These centers contribute to the downward and forward growth of the face. . . . The loss of anatomical continuity 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 import. . . .

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.

← Important

!!

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 Thallwitz near Leipzig, Germany. Here 130 primary cleft lip, 150 primary cleft palate and

Oskar Herfert

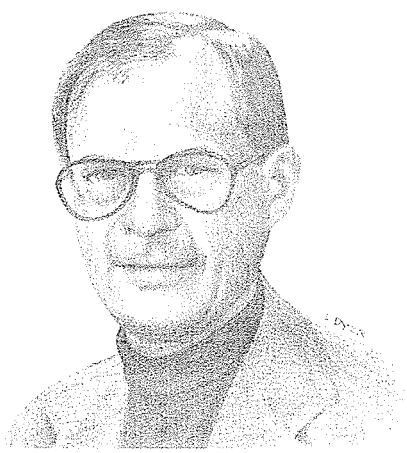
350 secondary operations were carried out annually. Oskar Herfert, with both dental and medical degrees, joined Rosenthal and was stimulated by him to examine 350 postoperative cleft lip and palate cases. He discovered that patients in whom the palate was operated on between 2 and 5 years of age showed 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.



Charles Kremenak

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.

Josh Jurkiewicz

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.

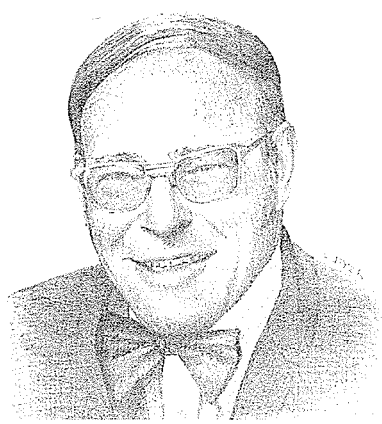
Similar difficulties have been encountered in the cleft palate of the horse.

STUDY OF THE EFFECTS OF SURGERY ON HUMAN GROWTH

Daniel Subtelny

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.



Thomas Graber

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:

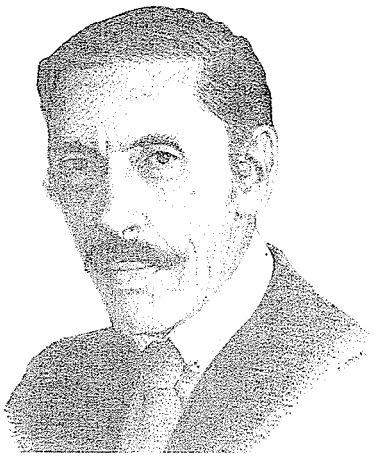
It is a well-known fact that the premaxilla unites with the maxilla to establish the maxillary arch between the age of four and five years. As I mentioned, we have run two parallel series using exactly the same technique. (1) An early age group before two years of age, and (2) a group at the age of four. 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

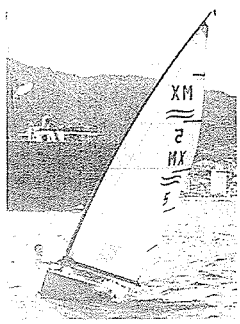
began scrambling around in search of these cases to help settle the argument of *just when the patient is really ready for surgery.* Yet finding unoperated adult cases was not so easy in the more advanced areas of the world for, as Claire Straith said over 25 years ago,

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



Fernando Ortiz-Monasterio

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.

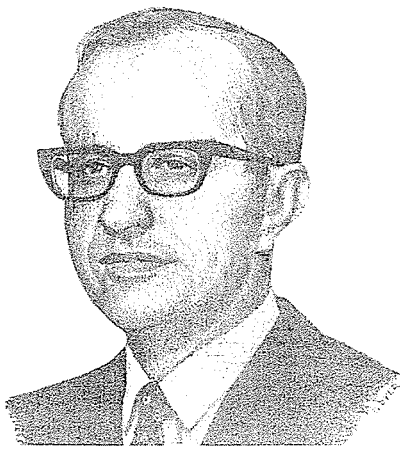
These findings of improvement in time were also observed by C. O. Innis in unoperated adult clefts of the Dusan tribes of North Borneo. He concluded that most deformities seen in the Western world in postoperative cleft patients were iatrogenic. I had noticed effects in adult unoperated clefts in Korea 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 Blocksma 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:



Sam Pruzansky

Some of the significant findings include a relative maxillary skeletal protrusion 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:

Egil Harvold

> 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.

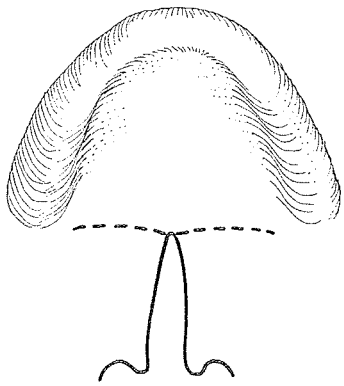
4. Buckling inward; or hourglass deformity, of the lateral portions of the alveolar arch.

HOURGLASS MAXILLARY COLLAPSE

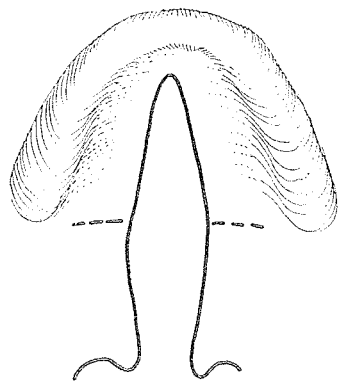
The "buckling collapse" deformity was first noted by T. D. Foster of Stoke Mandeville, England, in 1962. It appeared postoperatively in 11 of 102 complete unilateral cleft lip and palate cases, 4 of 47 bilateral complete clefts, and 7 of 19 postalveolar cleft palates without cleft lip. Foster made another 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.



Small cleft



Large cleft

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. Leck, 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.

7LKs

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:

1. Initial State ⟶ 2. Manoeuvre ⟶ 3. Subsequent State.

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:

1. The nasal septum varies in its cant and thickness (Aduss and Pruzansky, *Arch. Otolaryngol.,* 1967).
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 adenoidectomy, 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.