V. Hodgepodge
CONGENITAL velopharyngeal incompetence has as its most important manifestation hypernasal speech without a cleft of the velum. On examination, often to the surprise of the surgeon, there is at first sight a normal-looking palate with good mobility of the velum. More careful scrutiny reveals that in action the velum does not reach to the posterior pharyngeal wall, either because of a short palate or because of an abnormal backward position of the posterior pharyngeal wall. Of the many possible causes of velopharyngeal incompetence, one can be submucous cleft palate; however, not all submucous clefts have velopharyngeal incompetence.

Surgeons of the nineteenth century generally considered the submucous cleft of the hard palate the cardinal cause of open nasal speech. Some surgeons of that era, however, including Billroth, Passavant, von Langenbeck and Wolff, were convinced that late, spontaneous closure of palate clefts was frequent, without or after incomplete or unsuccessful surgical intervention. Robert Ivy himself reported such a case.

SUBMUCOUS CLEFT PALATE

In 1825 P. J. Roux of Paris first called attention to submucous cleft palate. In his Mémoire he recorded that, in 1823, he had been consulted by a young girl who nasalized so badly that her
speech was unintelligible. She had a cleft of the posterior portion of the velum, and there was a faulty union of the osseous tissue of the hard palate under an intact mucosa.

In 1846 Demarquay exhibited a dissection in which there was a cleft of the velum and a cleft of the bony palate which was filled with fibrous tissue and covered with intact mucosa.

Henri P. J. Winters of Utrecht University considers Gustav Passavant the rightful claimant of priority for describing congenital velopharyngeal incompetence. In 1862 Passavant reported a young female patient with a cleft lip and a spontaneously healed small cleft in the soft palate who three years later revealed the velum closed but a broad, deep submucous cleft of the hard palate and open nasal speech:

She visibly could not bring the velum into contact with the posterior pharyngeal wall.

In 1865 Passavant reported a man with a submucous cleft of the hard palate, an intact velum, a small bifid uvula and heavy open nasal speech. Further evidence suggesting Passavant’s genuine insight into the true physiology is given by his 1865 use of “insufficienz” in characterizing open nasality in speech after surgical closure of clefts of the soft palate.

In 1864 von Langenbeck described three types of bony clefts with the mucosa intact: (1) cleft of the velum with bony palate almost entirely absent, (2) cleft of the velum and hard palate in which the fissure in the bony palate was more extensive than in the soft tissues and (3) cleft of the velum associated with a fine split in the midline or on either side of the vomer, again with mucosa intact.

In 1869 Notta reported an 11-year-old girl with cleft uvula and bony cleft to incisive foramen with mucosa intact, who spoke with a distinct nasal twang. In 1870 Ulysse Trélat recognized the association of anteroposterior brevity of the bony palate and notching of the palate bones but attributed the nasal intonation to the anteroposterior shortening and lateral narrowing of the hard palate.

Lermoyez in 1892 defined “l’insuffisance vélo-palatine,” or congenital velopharyngeal insufficiency, as a developmental disturbance with a healthy, mobile, normal-looking short velum
which was always associated with a submucous cleft of the posterior hard palate and a bifid uvula. Gutzmann in 1899 expressed doubts as to this invariable association. In 1893 Mears of Philadelphia was the first American to note this condition.

In 1907 Ricardo Botey of Barcelona devised an operation for correction of the deformity. It involved elliptical excision of one or two vertical sections of posterior pharyngeal wall and closure with a continuous mattress suture, as diagramed.

Adam Brown Kelly, chief of the Throat and Nose Clinic at Victoria Infirmary in Glasgow, Scotland, was described as being a combination of modesty, geniality and humanity who "always remained an unrepentant laryngologist." He reveled in research and was the first, in 1910, to coin the phrase *submucous cleft palate*. He made a very complete study of eight cases and 11 cases of what he described as muscular insufficiency of the palate, in which all 19 presented rhinolalia aperta. He also observed a further 18 submucous cleft patients with normal speech. Measuring and comparing the hard and soft palate and the nasopharyngeal opening of these and normal subjects, he decided that both the hard and soft palate were short in submucous clefts. Kelly published sketches to demonstrate the variety of missing portions of the posterior hard palate. He was also the first to acknowledge that there could be hypernasality in the absence of the stigmata of submucous cleft palate.
In 1922 and 1923 Miloslav Seeman of Prague contributed several papers on submucous clefts. In 1927 Alexander Limberg of Leningrad introduced the terms *fissura ossea occulta* for submucous cleft of the hard palate and *fissura muscularis occulta* for submucous cleft of the soft palate.

In 1933 George Dorrance of Philadelphia, in his description of submucous cleft palate, remarked that the bony deficiency in the posterior part of the bony palate could vary from a large V-shaped defect to a mere notch to no loss of bone at all. Even with no bony defect, Dorrance pointed to the submucous cleft in the muscular tissue and in the palatal aponeurosis. He observed that the palatal mucosa was always intact but the velum was pulled forward with marked shortening in the anteroposterior diameter of the palate. He presented a sketch from Karl Peter showing a split uvula and he noted that the insertions of the levator palatini muscles were displaced forward and thus were unable to raise the velum upward and backward to the desired point of contact with Passavant’s cushion for velopharyngeal closure.

In 1954 James Calnan, while at Oxford, wrote a learned treatise on submucous cleft palate. He noted the varying deficiency in the bone of the posterior edge of the hard palate, the mobile but markedly shortened velum and the bifid uvula. The absence of muscle union and of a median raphe down the midline of the velum presented a translucent zone seen in the mouth when a beam of light was flashed from above. During phonation, this area broadened owing to the pull of the tensor and levator muscles.

Calnan excised the submucous cleft in eight cases and reported lack of muscle union across the cleft or poorly developed muscle fibres which lack orientation lying in a matrix of fibrous tissue. Mucous glands may present between the muscle fibres.

By cineradiography, as described by Ardran and Tuckey in 1951, Calnan studied the submucous cleft palate during speech and found that the velum failed to occlude the nasopharyngeal isthmus when it should, but its mobility and degree of elevation were not markedly impaired. The failure seemed to depend on the shortness of the velum, but when the posterior pharyngeal wall came forward, the velum moved upward and occluded the aper-
ture at a much lower level than normal.

Calnan was quite dogmatic about treatment:

In our experience there is only one treatment for submucous clefts with rhinolalia and that is surgical excision of the submucous portion of the cleft and V-Y retroposition of the soft palate on the lines described by Kilner.

If normal speech does not develop in three months, speech therapy is begun. Out of 17 cases, 15 achieved normal speech. Of the two remaining cases, one was successfully treated with a Hynes pharyngoplasty and in the other a Hynes procedure was planned. Calnan’s stand on submucous cleft in infancy is of interest:

The correct time at which to treat patients with submucous cleft (as with obvious cleft palate) is at or about one year of age, before the development of speech. As has been mentioned, patients are not sent for treatment or diagnosis until much older. This is due to a failure of diagnosis which could be improved: all patients with cleft lip should receive a thorough examination of the palate. If a submucous cleft can be diagnosed with confidence, then the rational treatment is to excise the fibrous cleft and retropose the palate.

Six patients with this condition have been seen and treated in infancy in this Department. Four now have normal speech without the need for speech-training: the remaining two seem to be developing normal baby-talk.

In 1965 H. William Porterfield and John C. Trabue of Columbus, Ohio, endorsed Calnan’s classic triad of (1) bifid uvula, (2) midline soft palate muscle separation with intact mucosa and (3) midline notching in the posterior edge of the bony palate as prerequisites for diagnosis of submucous cleft palate. They reported 18 submucous cleft palate cases out 505 cleft palate patients and made recommendations as to what to do:

In view of our experience of a delay in seeing these cases and inferior speech results, we advocate the following: (1) Recognition of this defect in early infancy by the initial examining physician. (2) Surgical repair at 16 to 18 months by the pushback technique.

**Diagnostic Aids**

In 1966 several publications dealt with submucous cleft palate, paying special attention to diagnosis and its guide to surgery.
Previous techniques used for diagnosis of submucous cleft palate had ranged from Olin's palpation of the bony defect to Van Riper's use of radiography. Raymond Massengill, dedicated speech pathologist at Duke University, who raises roses and Tennessee walking horses, was stimulated in 1966 to devise a simple light instrument with an extension to be passed into the nostril and over the nasal side of the palate. The light penetration of the submucous cleft is studied by oral photometry with a photocell placed under the palate. Readings establish the extent of the submucous cleft. Variations of this overt diagnostic technique, presented in *Plastic and Reconstructive Surgery*, are used in many clinics today.

In 1967 Thomas Rees, D. Wood-Smith, C. Swinyard and J. Converse of New York University studied 12 submucous cleft palate cases with electromyography and diagramed the typical zone (dotted region) of absent to diminished electrical activity. They reported in *Plastic and Reconstructive Surgery*:

1. Electromyography is a useful diagnostic adjunct in the submucous cleft palate.
2. Electromyographic "mapping" of the muscle deficiency in the submucous cleft palate can serve as a guide to the surgeon in selecting the appropriate width of nonfunctioning soft palate to be excised.
3. The muscle deficiency present in the submucous cleft palate would seem to warrant the addition of muscle tissue as a component part of the repair. A pharyngeal flap helps to achieve this objective.

Indeed, their best speech results, they found, occurred in the two Veau-Wardill pushback operations with a superior pharyngeal flap added.

In 1979 Randall G. Michel, G. J. Baylin, A. S. Hall, I. H. Pipkin and W. R. Hudson of Duke University reported palatal tomography to be of benefit in diagnosis of occult hard palate defects. Although not advocating wide clinical use of this technique, they noted:

Nine of the 12 patients were found by tomography to have palatal defects that had not been detected either by cineradiography or by clinical investigation, including physical examination of the palate.
Defining SMCP

In 1970 George F. Crikelair, P. Striker and B. Cosman of the College of Physicians and Surgeons, Columbia University, New York, presented a provocative stand against limiting the diagnosis of submucous cleft palate to Calnan's triad. They stated:

Actually, there is no such single grouping of findings that can be defined as a submucous cleft palate.

As recognized by the classical authors, from von Langenbeck through Dorrance, there is a variety of submucous defects in which a submucous zone (either large or small) is present together with (or rarely, without) an actual cleft—which, in turn (when present), may be large or small. The submucous area may be in the hard palate, the soft palate or both; the palatal bony defect may vary from near total absence to apparent normality (without even a notching of the posterior margin). This wide spectrum of anatomical appearances is borne out in our experience; the only constant feature of the "submucous cleft palate" is the presence of a submucous zone.

They presented their stand diagramatically in *Plastic and Reconstructive Surgery*, with the dotted area being the submucous
defect, the dashed line the posterior edge of the hard palate, and
the dashed and dotted line the normal outline of the alveolar
ridge or posterior edge of the hard palate or the posterior edge of
the velum.

Crikelair’s team, with a surgical experience of 20 significant
submucous cleft palate cases, opposed the accepted dogma of
excision of the submucous zone and palate pushback.

When the submucous area is large its excision may leave little tissue for
palate closure of any kind—and predispose to breakdown and fistula forma­
tion. On the other hand, it is clear that significant submucous defects are
not always associated with short soft palates and/or short hard palates.
Consequently the insistence on a push-back procedure in all cases is hard to
justify.

Our experience demonstrates that good speech results may be achieved
without excision of the submucous section of the defect and/or without a
push-back procedure. Where the palate does appear to be short, a primary
pharyngeal flap may be used (without submucous zone excision). Where the
submucous portion is smaller, narrower, and in the soft palate, and where
the palate segments are ample, excision and a simple von Langenbeck
closure can give good results. . . . It is thus possible to approach therapy in
each instance of submucous defect freely.

In 1969, 1970 and 1974 John E. Hoopes and others of Johns
Hopkins University confirmed by cineradiography the early find­
ing of Dorrance that the anatomical defect in submucous cleft
palate was the insertion of the levator palatini muscles too far
forward to elevate the soft palate effectively. He defined the
factors responsible for velopharyngeal incompetence in submu­
cous cleft palate as (1) a short soft palate and (2) an anteriorly
displaced levator insertion. The more anterior the levator inser­
tion, the greater the velopharyngeal incompetence.

Asymptomatic SMCP

Another surgeon interested in submucous cleft palate is R. C. A.
Weatherley-White of Denver, who seems to spend most of his free
time in the air. Born in India into a third-generation Indian Medi­cal
Service family, he served as a paratrooper in the 82nd Air­
borne Division, was once United States National Inter-Collegiate
Sky Diving Champion and is now learning to fly aerobatically.
His first contact with plastic surgery was through Sir Archibald
McIndoe’s visit to the R.A.F. Hospital at Holton, and he later trained with Richard Stark in New York.

In 1972 Chris Weatherley-White, C. Sakura, L. Brenner, J. Stewart and J. Ott of the University of Colorado found nine submucous cleft palate cases in a study of 10,836 Colorado school children in the Denver area. This presented an incidence of 1:1,200, but only one child had mildly abnormal speech and it was corrected by therapy alone. In these and 52 other submucous cleft palate patients referred, the combination of a relatively short palate with lessened mobility and demonstrable easy fatigue with effort was noted. Speech proficiency was not related to the degree of muscular clefting. Only four patients had surgery, and this involved the complete excision of the diastasis (“zona pellucida”) combined with pharyngeal flaps in all, plus Veau-Wardill push-back in two. Hypernasality was corrected in three and improved in one.

Weatherley-White wrote in 1976:

In our epidemiological studies on submucous cleft palate, I am constantly reinforced in our initial premise by the fact that I see so many asymptomatic submucous cleft palates. My aphorism concerning this lesion would be to follow all children with submucous cleft palate very carefully with frequent routine speech evaluations. Surgery should be done if there are signs of velopharyngeal incompetence; this should obviously be done as soon as the diagnosis is made, to prevent neurological pathways becoming stratified and causing a persistence of the speech problem.

In 1973 Raymond Massengill, K. Pickrell and M. Robinson of Duke University reported on a comparison of 12 submucous cleft palate patients with a random group of 12 postoperative cleft palate patients. The groups were matched according to age and sex, and all had some type of pushback procedure with what was presumed to be adequate length—either a Veau, a Dorrance, a Dorrance with an island flap, a Wardill or a Wardill with an island flap. In their small series, the submucous cleft palate patients had a higher percentage of velopharyngeal incompetence than the controls. This was partially explained by the later diagnosis of the patients, after they presented speech problems, and resultant greater palatal height in the control than in the submucous cleft palate groups.
Methods of Treating SMCP

In 1970 John Hoopes with A. Dellon, J. Fabrikant and A. Soliman of Johns Hopkins University suggested:

The procedure of an island flap pushback alone has proved disappointing in the management of submucous cleft palate, for which reason the combined procedure [island flap and pharyngeal flap] is utilised. The difference in the type of closure obtained with an island flap pushback versus a pharyngeal flap is essentially the difference between active and passive closure, respectively.

They reasoned that, in addition to the increase in active closure achieved with the posterior displacement of the levator muscles by the island, extra passive aid was given by the tethering of the pharyngeal flap.

In 1974 N. Culf, J. Chong and L. Cramer of Temple University stated that one of the ideal candidates for the double sandwich island flaps was the submucous cleft palate patient and reported 10 cases so treated. They wrote:

In cases of submucous cleft with significant separation of the levator muscles, the muscle bundles are easily approximated through the transverse incision at the junction of the hard and soft palate.

F. L. F. Innes of Norwich, England, trained by Kilner and Peet and influenced by the work of Calnan, is convinced that early cleft palate closure is ideal and is concerned that submucous clefts escape detection. He wrote in 1976:

Most of the patients do not reach me until a year or two after they have gone to school. This condition is still too often diagnosed much too late with the result that the unfortunate child is greatly handicapped.

The educational and psychological problems of late diagnosis are by themselves bad enough but there are in addition physical problems. The soft palate elements in such patients are very poor and underdeveloped. The standard Kilner-Wardill operation is probably adequate for children with submucous clefts who are presented for operation at about the age of one year, but if there is any doubt about the result of this operation by itself, an island flap . . . can be easily introduced. For older patients, the Kilner-Wardill operation is not adequate. Something more is required because in such patients the pharynx will have become excessively large. The pharynx seems to grow too wide and too deep without the influence of proper palatal muscle action in the front of the palatopharyngeal isthmus.
In these cases Innes advocated the combination of a pushback procedure aided by an island flap and complemented with reduction of the pharynx by the Hynes pharyngoplasty.

In 1975 in the *Cleft Palate Journal* E. N. Kaplan of Stanford University noted that the classic submucous cleft palate with Calnan's triad represents the obvious overt physical signs of an underlying anatomical abnormality—the insertion of the levator and other palate muscles onto the hard palate instead of forming a sling across the midline. He stated:

As a result of this muscle malposition, velar function may be abnormal and velopharyngeal incompetence may result. We now recognize that muscle malposition can occur in the absence of the triad of overt signs. This condition is designated "occult" submucous cleft palate.

We believe that isolated cleft of the secondary palate, submucous cleft palate, and occult submucous cleft palate are variations in expression of the same embryologic disorder. . . . However, we would exclude cleft palate associated with craniosynostosis . . . branchial arch syndromes . . . mandibular micrognathia . . . and cleft palate with cleft lip . . . because they are probably embryologically distinct conditions.

Kaplan reviewed 250 cases of velopharyngeal incompetence without cleft lip or cleft palate and identified 41 cases of classic submucous cleft and 23 cases of occult submucous cleft. Here are his diagnostic aids:

1. Facial features suggestive of occult or classic SMCP:
   a) maxillary hypoplasia—"dish face" (75%)
   b) lip contour deformity at vermilion border—"gull wing" (75%)
   c) drooping of oral commissure (25%)
   d) dynamic facial muscle abnormality (25%)
      paranasal bulge—horizontal
      lateral lip bulge—vertical
      hypoanimation—a "dull" face or expression
   e) external ear abnormality—flat arc of superior helix (10%)
   f) alveolar arch abnormalities (5%).
2. Cephalometric studies revealed that in 90% of patients, the hard palate length was less than average but within one standard deviation of normal; in 75% nasopharyngeal depth was greater than average and only 10% fell more than one
standard deviation away from normal, but in 90% the soft palate was short by one standard deviation and 75% were short by two standard deviations.

3. Cinefluorographic studies were recommended to confirm velopharyngeal incompetence and to help determine need for therapy or surgery.

Kaplan noted:

Ultimately, however, the definitive diagnosis is dependent upon the intra-operative exploration of the soft palate muscles.

He then elaborated:

1. A midline incision extends from the distal centimeter of the hard palate to the proximal centimeter of the soft palate.

2. The oral mucoperiosteum is lifted laterally with a periosteal elevator and the oral mucosa of the soft palate is dissected sharply with scissors. Extreme care must be taken to avoid cutting into the muscle or damaging the mucosa.

He admitted to great variation of levator muscle insertion but noted that 75 to 90 percent of the levator muscles inserted on the bone with some muscle meeting in the midline.

Kaplan outlined his surgical approach:

1) If the diagnosis of occult submucous cleft palate is not confirmed (i.e., muscle anatomy is normal), the following plan is generally followed:
   a) Patients with normal palate mobility—retropharyngeal implant.
   b) Patients with palate paresis—obturating pharyngeal flap.
2) If there is an occult submucous cleft . . . we have reconstructed the palate by levator muscle reconstruction [A], palate pushback, and high superiorly based pharyngeal flap inserted into the raw surface of the nasal side of the palate [B].

This is the method described in 1965 by Dibbell, Laub, Jobe and Chase, applied to occult submucous clefts by Kaplan with the addition of reconstruction of the levator muscle sling.

In 1975 Minami, Kaplan, Wu and Jobe of Stanford University reported finding that 44 percent of submucous cleft palate patients with velopharyngeal incompetence had decreased palatal mobility on lateral cineradiography. They also pointed to the tethering effect of the abnormal levator insertions into the posterior hard palate as the key defect in SMCP. As they wrote:

The levators contract isometrically against an immobile insertion; thus, they may appear to be paretic.

They also reported four patients with none of the classic findings of SMCP who, after palatal exploration, revealed in each case a very small bony notch, unmistakable abnormal levator palatini insertions into the posterior border of the hard palate and diminished palatal mobility.

Treatment: As pointed out by Weatherley-White, normal speech will develop without any treatment in about 90 percent of SMCP patients, making early treatment inadvisable. For cases that do come to surgery, the Stanford group advocates, as standard procedure, pushback to correct the short palate and release the abnormal levator attachments and superiorly based pharyngeal flap to line the raw area and hold the backward displacement with a partial obturation of the velopharyngeal gap. To this they have added reconstruction of the levator sling.

In 1976 Frank E. Abyholm of Oslo, Norway, reported 47 submucous cleft palate patients, operated on at the average age of 10.8 years during 1965 to 1974. Eleven had had tonsillectomy and/or adenoidectomy prior to diagnosis. He noted better results when the patient was operated on under 7 years of age and from his experience advocated von Langenbeck or pushback palate closure with levator muscle sling construction and a superiorly based pharyngeal flap.

Meanwhile, Porterfield had continued his interest in the palate
and submucous clefts. In 1976 he recalled the problems faced 20 years ago and felt encouraged by the improvements that have followed:

My venerable old Chief, Harold Trusler of Indianapolis, had a reputation for being a gruff, tough old fellow, but who beneath the surface, was a very gentle and kind man. He would look at a previously operated palate that demonstrated much scar and immobility, turn away and say to the gathered audience, "that surgeon just had a bad pair of hands." To me, this merely points out what has been accomplished in these years.

**The Calnan Controversy**

Eleven years after his first paper, Porterfield, with Mohler and Sandel in 1976, still held rigidly to Calnan’s triad as requirements for a case to be admitted to the inner sanctum of "submucous cleft palate.” In a direct attack:

We would take issue with the statements of Crikelair et al in which they described submucous defects in association with overt cleft palate deformities and designated that deformity also as a SMCP. We feel the strict criteria of Calnan are valid ones for delineating the submucous cleft palate problem.

Meanwhile, Porterfield became aware that a pushback at 16 to 18 months was not sufficient treatment of SMCP. He began backtracking with an "ink had hardly dried" revision, suggesting in 1976 that a primary pharyngeal flap, or a superiorly based pharyngeal flap combined with a von Langenbeck palatoplasty if the palate seemed short, would be a more efficacious procedure.

This, of course, excited a 1977 Letter to the Editor from Crikelair and Cosman to Porterfield in reference to submucous cleft palate diagnosis and treatment. Excerpts from the letter follow:

It is a simple fact that submucous defects can occur in the hard palate without any involvement of the soft palate. It has also been observed that extensive submucous defects in the hard and soft palate may coexist with clefts of the soft palate considerably larger than a mere bifid uvula. Such cases have been amply documented [Crikelair et al., Roux, Demarquay, Trélat, and Veau]. What name could one give to these defects, if not "submucous cleft palate?"

The restrictive criteria of Calnan serve to perpetuate the view that submucous cleft palate is a wholly different entity from cleft palate, rather than one of its manifestations. This concept has led Calnan, Porterfield, and
others to the too severe stricture concerning the necessity for excision of the submucous portion of the defect, a view which Dr. Porterfield and his colleagues now agree is in error.

It would have been generous of Dr. Porterfield et al to have pointed this out—and to have indicated, as they now also show, good speech may be achieved by the simple addition of a pharyngeal flap to the submucous cleft palate, without any other manipulation.

Porterfield’s 1977 reply to the Crikelair-Cosman letter ended:

My principal reason for disagreeing with them . . . is, however, simply that of being a “purist.” I think that the original description of Calnan should be retained, that it should not be confused by extraneous modifications.

To throw another dimension into submucous cleft palate, M. Fára of Prague stated in 1977:

All our 105 patients with submucous cleft manifested medial cleft palate with vomer in the central line . . . The wider the cleft in the palatal plates, the greater the parting of the velar muscles and the substituting attachments of these muscles to the palatal plates are functionally less valuable. Every fourth patient with submucous cleft was afflicted with the syndrome of developmental shortening of the palate: special physiognomy with striking hypomimia and decreased intellect.

A SIMPLER APPROACH

Dennis Walker of Johannesburg, who devised a different approach to the various submucous cleft palate problems, remembered a visit to the theater at 149 Harley Street when Gillies, in a mischievous mood, called through to Sir Archibald McIndoe in the next room, to ask whether Walker might watch him work:

McIndoe said, “yes, of course,” and then the wicked old maestro engaged my attention so that I was forced to stay with him, until McIndoe looked at me sideways when I eventually went to watch him.

With a touch of Gillies’ flexibility, Walker designed his attack on what he considered the specific problems of the submucous cleft, leaving the uvula, when not bifid, intact.

CONGENITAL HARD PALATE HOLES

Defects affecting only the hard palate are among the rarest found in clefts. They appear as oval holes in the midline, not usually...
extending the whole length of the palate plates. Their occurrence is usually thought to be connected with a submucous cleft. Evidently there are exceptions.

In 1966 J. B. Lynch, S. R. Lewis and T. G. Blocker of the University of Texas, Galveston, reported a case of a Caucasian male with a moderately wide cleft of the hard palate extending from the incisive foramen to the junction of the hard and soft palates and an unattached, underdeveloped vomer. The alveolus was intact and the soft palate normal with microscopic sections of its midline revealing

muscle fibers extending to the midline, which indicated that the muscular portion of the soft palate was, in fact, normally developed and did not represent a submucous cleft.

This defect, they felt, was not explained by any modern concepts of embryology.

It is more common, however, for this hard palate defect to accompany a submucous cleft palate. Both Trélat and Veau suggested that the phenomenon was a prenatal rupture of a submucous cleft, either spontaneous or artificial, and Fára endorsed their stand, estimating the occurrence of the opening at the time when the intrauterine growth of the head was reaching completion. In 1954 Calnan presented one of these cases with a hard palate hole accompanying a submucous cleft palate.

In 1971 in *Plastic and Reconstructive Surgery* Miroslav Fára of Charles University, Prague, reported five cases of congenital defects in the hard palate associated with typical complete submucous clefts and, in addition, slightly underdeveloped maxillae and marked hypoplasia of the palate plates.

**TREATMENT**

One of the cases reported by Fára was treated by F. Burian in 1935 with a mucoperiosteal rotation flap and inferiorly based pharyngeal flap.

Fára himself used a more refined approach. Excision of the midline submucous cleft area revealed the following:
On the sides of the excision, numerous cross-sectioned muscle fibers are seen; toward the midline they become rare, with an oblique or longitudinal change in direction. In the midline, however, the muscle fibers are completely absent and one sees only dense connective tissue.

Fára emphasized the importance of proper retropositioning of the palate, after detaching the muscle insertions from the posterior margins of the hypoplastic palatal plates, and suturing the muscles in the midline with a primary pharyngofixation [using a superiorly based flap.]

**PATHOGENESIS OF SUBMUCOUS CLEFT**

Dynamic David Poswillo of the Royal College of Surgeons of England Research Establishment in Downe, Kent, in 1974 reported an intriguing study of exogenous factors in the etiology and pathogenesis of complete and submucous cleft palate. A series of pregnant mice were given phenytoin in pediatric suspension at the rate of 150 mg per kilogram by gastric intubation from day 12 to day 16 of pregnancy. Examination of 100 consecutive fetuses at day 18.5 revealed 16 with complete cleft palate, 15 with submucous cleft palate and 69 normal. Serial study of the mouse fetuses in which SMCP had been induced supported the hypothesis of interference with mesodermal differentiation. A centripetal gradient of differentiation in the palatal shelf was described, commencing at the nasal foramen and extending to the uvula. When this gradient was disturbed by teratogens, after fusion of the palate, either SMCP plus bifid uvula, or bifid uvula alone resulted, the anomaly being determined by the stage of onset in relation to the anteroposterior gradient risk.

Study of the animal defect helped identify the causal mechanism in man. The findings supported the proposal that the teratogensensitive period of palatogenesis in man should be regarded as extending from early embryogenesis to about the twelfth week of development. Until that time, agents can act to interfere with the developing palatal plates and the velar mesoderm in such a way that SMCP and bifid uvula, microforms of cleft palate, could result.
OTHER CAUSES OF VELOPHARYNGEAL INCOMPETENCE

In 1933, in his remarkable book *The Operative Story of Cleft Palate*, George Dorrance of Philadelphia discussed congenital insufficiency of the palate and categorized six varieties:

1. Normal appearance but inability of velum to approximate the pharyngeal wall because of anteroposterior shortening of the hard palate and velum.
2. Velum normal but hard palate short.
3. Hard palate normal but velum short.
4. Hard palate normal but submucous cleft of the velum.
5. Velum normal in appearance but with submucous cleft extending into the hard palate.
6. Palate insufficiency after successful cleft closure with the velum too short to reach the posterior pharyngeal wall.

Dorrance noted that children with congenital shortening of the palate usually learn to speak later than normal children, or speak indistinctly. Speech deficiency improves with time but is often associated with compensatory mechanisms such as development of compressor naris muscles and hypertrophy of the faucial and pharyngeal tonsils. His dissertation on diagnosis by symptoms was impressive.

1. Rhinolalia aperta or open nasalizing where vowels take a nasal tone ("ah" becomes "an"), consonants become altered with exception of "M" and "N" and sigmatismus, or the inability to produce the letter sound "S."
2. Shortness of breath in speaking due to air loss through the nose.
3. Inability to whistle.
4. Inability to hiss.
5. Inability to blow out a candle flame.
6. Mouth breathing becomes a habit presenting a vacant expression.
7. Pronounced facial movements with such muscles as the nasal compressor and corrigitors to turn the patient into a "face-talker."
8. Diminished hearing due to improper ventilation of the middle ear.
10. Fast talk avoiding difficult words.
11. Intra-oral examination reveals velopharyngeal insufficiency due to overall shortness of the palate and possible weakness in the superior pharyngeal constrictors.
12. Intranasal examination reveals insufficient velopharyngeal closure.
13. Bifid uvula in some cases.
14. Palpation through normal mucosa reveals various examples of submucous cleft in the muscles of the soft palate and the bone of the hard palate.
15. Irregularity and crowding of teeth, congenital absence of maxillary incisor tooth or the association with cleft lip.

In reference to nasal escape Dorrance stated:

The most reliable test is to hold a piece of cotton in front of the anterior nares while the patient makes efforts to pronounce non-nasal letter sounds. The escaping air makes the cotton move when this mechanism is insufficient.

**DIFFERENTIAL DIAGNOSIS**

Dorrance warned that congenital insufficiency of the palate should not be confused with palsy of the velum, "stomatolalia" and speech defects due to loss of teeth or faulty lingual articulation. Palsy of the palate is recognizable by the absence of the palatal reflex, inability of the velum to move and its lack of response to faradic stimulation. Stomatolalia or rhinolalia clausa is a condition of speech in which the letter sounds lack their nasal resonance. Dorrance added that speech defects due to loss of teeth are correctable by dentures and, if due to faulty tongue habits, are benefited by methodical speech training. He recommended his "push-back" operation of the palate as the best treatment of congenital insufficiency.

In 1954, while still at Oxford University James Calnan outlined, in addition to submucous cleft palate, other causes of velopharyngeal insufficiency:

2. Cerebral agenesis of the supranucular bulbar origin.
3. Paralysis of the palate (infectious or viral, which usually clears after recovery from the infection).
4. "Tonsillectomy palate" in which the surgeon has taken a portion of the soft palate along with the tonsils.
5. Functional rhinolalia where the patient "talks down the nose" for no apparent reason.
6. Rhinolalia following adenoidectomy.
RHINOLALIA AFTER T & A

Great interest has been shown in the condition of velopharyngeal incompetence following adenoidectomy.

In 1958 A. G. Gibb published his findings on a series of 19 patients seen by him in Scotland with permanent nasal escape following removal of tonsils and adenoids. He found that there had been 27,734 operations for removal of tonsils and adenoids in a population of 62,000 from 1950 to 1957. The incidence of permanent speech defect was 1 in every 1,459. Yet Calnan reviewed the Scottish series and indicated the probability that five were sub mucous cleft palate patients and four were mentally retarded with possible congenitally large pharynx. Gibb, he noted, felt that the soft palate was short and considered this factor the cause of nasality after adenoidectomy.

In 1971 James Calnan of the Royal Postgraduate Medical School, London, reported that between 1951 and 1968 he had a series of 19 patients with permanent nasal escape during speech following removal of tonsils and adenoids. All had had normal speech prior to the T & A and were above average intelligence. Radiological studies demonstrated a fully mobile soft palate with a gap between it and the posterior pharyngeal wall. When the gap was occluded by a cartilage implant behind the posterior wall of the pharynx, speech returned to normal. Cephalometric measurements suggested that the essential defect was a pharynx deeper than normal. He stated:

A good analogy would be a size 7 foot in a size 8 shoe: both are “normal” but of little use together.

Calnan acknowledged that the disproportion between palate and pharynx was probably not possible to diagnose before adenoidectomy.

In 1975 Roland Minami, Ernest Kaplan, George Wu and Richard Jobe of Stanford University reported on 23 patients with hypernasality following removal of adenoids and tonsils. In most cases, they noted, velopharyngeal incompetence after a T & A is transient and disappears in a few weeks after compensation for the missing adenoid prominence. A marginally adequate mechanism may not be able to cope and will “unmask” the presence of
a submucous cleft or other pathology. Surgical experience with the standard Stanford pushback with superiorly based pharyngeal flap achieved improvement of severe to moderate hypernasality to minimal with the best prognosis in patients with normal amplitude and quickness of palatal motion. Nine patients had no surgery and, out of six of these followed three years, two showed improvement.

Noting the extensive reports of good results in such cases with retropharyngeal augmentation, Minami and his colleagues admitted favoring this approach “with a velopharyngeal gap of 5 mm. or less.” When there was preexisting hypernasality worsened by T & A, surgery, they felt, should be directed toward correction of the underlying cause.

PROPHYLAXIS

It is important that children with congenital palatal incompetence who have only minimal hypernasality not be subjected to a standard adenoidectomy. Subtelny and Koepp-Baker advised that when adenoidectomy was absolutely indicated because the adenoid tissue covered the orifices of the Eustachian tubes, a lateral band adenoidectomy could be done, leaving the bulk of adenoid tissue in the midportion of the pharynx undisturbed.

CONGENITAL LARGE PHARYNX

By 1971 James Calnan of London had added congenital large pharynx to his list of six groups besides submucous cleft palate which can result in velopharyngeal incompetence. He reported a personal series of 41 patients seen over an 18-year period presenting nasal escape with apparently normal palatopharyngeal mechanisms. Extensive clinical and cephalometric studies revealed the pharynx to be larger than normal, justifying the term congenital large pharynx.

Calnan found no improvement with the Wardill-Kilner V-Y procedure, lack of nasal resonance with the Hynes procedure, extrusion of Teflon implants and the best results with autogenous costal cartilage implanted in the retropharyngeal area above the
arch of the atlas. His overall result was 60 percent normal speech obtained after the various types of surgery.

In 1975 Minami, Kaplan, Wu and Jobe of Stanford University divided velopharyngeal incompetence without overt cleft palate into the "Big Four":

1. Acquired palatopharyngeal disproportion after removal of adenoids and tonsils.
2. Abnormal anatomy of levator palati muscles—submucous cleft palate.
3. Palatal paresis.
4. Other causes—mental retardation, congenitally short palate, congenitally large pharynx and possible "occult" SMCP.

In 1979 W. S. Hagstron, R. W. Parsons, S. J. F. Landa and M. C. Robson of the University of Chicago, reported two cases of familial velopharyngeal incompetence caused by myasthenia gravis. They suggested consideration of: "myasthenia gravis (electromyography or testing with edrophonium) when the cause of neuromuscular dysfunction cannot be definitely established."

**PALATAL PARESIS**

The discussion of the treatment of palatal paresis by Minami et al. is of special interest. They noted that surgery has included pharyngeal flaps, citing Randall, Bakes and Kennedy (1960), J. C. Hardy et al. (1961), and Crikeland, Kastein and Cosman (1970); unilateral pharyngeal flap limited to affected side in unilateral palatal paresis, suggested by Broadbent and Swinyard (1959); and temporalis muscle and fascial sling reported by Kiehn et al. (1965). The obvious objective in a paralyzed palate is the construction of an almost complete obturator. If this can be accomplished by Hogan’s wide, lined pharyngeal flap closing off the lateral ports to 3 mm., then that is the method of choice. Palatal lifts and obturating types of prosthesis have been used successfully and may be the treatment of choice, at least in patients with cerebral palsy, as noted by Gibbons and Bloomer (1958), Lang and Kipfmuller (1969), Gonzales and Aronson (1970) and Hardy et al. (1969).

In all types and degrees of palatal paralysis resulting in velopharyngeal incompetence, the pharyngeal flap, by reduction,
constriction and obturation of the velopharyngeal aperture, has earned an important place in the surgical treatment. Its contribution is great even to such rare conditions as congenital suprabulbar paresis described in 1950 by Worster-Drought, in which the motor outflow to the tongue, lips, palate, pharynx and larynx is affected. In the mildest form of this syndrome, the soft palate suffers most with a marked rhinolalia. In 1958 Wynn Williams of Nottingham, using the Rosenthal pharyngeal flap, reported good results on 49 percent of the patients.

At the 1973 International Congress on Cleft Palate in Copenhagen, C. H. Waar of the University Hospital Dijkzigt, Rotterdam, advocated surgical fixation of the uvula to the lower adenoid region for speech improvement in paralysis of the soft palate. The chance of improvement is far better if, during phonation, a constriction of the pharyngeal musculature is noted. Waar cited two successful cases, a 5-year-old girl with bilateral paralysis of the glossopharyngeal nerve but no other neurological symptoms, and a 30-year-old male with dysarthria, hemiplegia and open nasality as a result of a traffic accident. After long, unsuccessful speech therapy, a uvula fixation was followed shortly by a definite reduction in nasality.

In 1977 at the Third International Cleft Palate Congress in Toronto, Donnell F. Johns, Ph.D., and Kenneth E. Salyer of the University of Texas Southwestern Medical School, Dallas, determined the width of their obliterating superiorly based pharyngeal flap by observing and marking the most medial excursion of the lateral pharyngeal walls. Using this width flap, they were able to report 14 successes out of 15 cases of neurogenic velopharyngeal incompetence.

In 1975 alert Jack C. Fisher of the University Hospital, San Diego, and M. Edgerton of the University of Virginia Hospital, Charlottesville, reported the combined use of the levator retrodisplacement and pharyngeal flap for congenital palate insufficiency. They presented their argument:

The retrodisplacement principle has been combined with a pharyngeal flap in a manner which provides muscular union between the LVPM [levator veli palatini muscle] in the midline of the soft palate and the fibers of the superior constrictor in the posterior pharyngeal wall. Continuity of muscular tissue at the borders of each new lateral velopharyngeal portal thus provides
an anatomic configuration with the potential for functional sphincter action.

Acceptable indications for this procedure include non-cleft palate insufficiency, clefts of the soft palate with short levator insertions, and previously repaired clefts with persistent nasality.

They use this approach in primary palate surgery, combining it with an extensive pushback. Their outline of the procedure in the *Cleft Palate Journal* began with elevation of mucoperiosteal flaps, freeing of vascular bundles and division of the nasal floor in the midline. This was followed by division of levator muscle bundles from the hard palate insertion as well as from the mucoperiosteum and nasal floor in order to permit retrodisplacement. Then a superiorly based pharyngeal flap was turned into the nasal defect and the levator bundles joined in the midline at the base of the flap. After completion of the palate pushback, catheters were left in each portal and fixed to the columella.

Here are Fisher's remarks in 1978 in reference to the paper:

First of all, it was the first honest admission that levator retrodisplacement alone in children with non-cleft velopharyngeal incompetence isn't very good very often. Second, it suggests a theoretic means for establishing a dynamic sphincter around the newly formed velopharyngeal portals. As of today, that is still speculative and unproven. I'm not sure my convictions are still strong that one can produce a dynamic sphincter with inherent muscular function. I was reminded of this when I listened carefully to Otto Kriens in Toronto last year. Surely, he has applied the levator repositioning principle most avidly, but he feels it is of advantage only within the first few months of life. I wonder if we have any chance at all of redirecting the function of those muscles if we wait until the child is 8 or 9 or 13? Thus, Levator Retrodisplacement with Pharyngeal Flap asks more questions than it answers.

**PUTTING IT ALL IN PERSPECTIVE**

After 150 years, finally, at the 1977 Third International Congress on Cleft Palate in Toronto, Samuel Pruzansky, with S. Peterson-Falzone, J. Laffer and P. Parris of the University of Illinois Center for Craniofacial Anomalies, imposed order on the chaos of the loosely grouped and ill-defined cases of hypernasality in the
absence of an overt cleft:

Hypernasality may be due to 1. disorder of the nervous system, 2. end organ defects, or 3. a combination of both.

All these cases of hypernasality were grouped under the heading of “Congenital Velopharyngeal Incompetence” (CPI) because the causative factors are present at birth. The incompetence (hypernasality) cases were divided into two groups, CPI(1) and CPI(2). In CPI(1), one or more of the triad of associated structural defects are present in a frequency of: bifid uvula, 85 percent; dehiscence of velar muscles, 65 percent; submucous cleft of the hard palate, 73 percent. Pruzansky noted that these associated defects are not causes of hypernasality, and one, two, or all three can be present without hypernasality. In CPI(2), no visible or palpable stigmata are present.

Radiographic examination of both types revealed one or more of the following: short or thin velum, platybasia, craniovertebral anomalies contributing to a deep pharynx, paucity of adenoid and early involution of adenoid.

CPI referral over the past 25 years has shown an increase in absolute number and in proportion to overt clefts, with an incidence of 49 percent CPI(1) and 40 percent CPI(2).

A study of 20 families of each group revealed the incidence of inheritance. CPI(1) showed a family history in 16.8 percent and CPI(2) in 22.2 percent, with an overlapping. Family history of clefts was seen in CPI(1) in 18 percent and in CPI(2) in 9.1 percent. It not only runs in families; it runs in syndromes!

As noted by S. Peterson-Falzone, S. Pruzansky, J. Laffer and P. Parris:

Both [CPI] types may occur in conjunction with a number of known craniofacial malformation syndromes, including mandibulofacial dysostosis, Klippel-Feil, von Recklinghausen, hemifacial microsomia, familial craniovertebral malformations, and others. Patients with Apert syndrome and Crouzon disease frequently exhibit the stigmata associated with CPI type 1 but do not show hypernasality due to decreased depth of the pharynx and abnormal size of the soft palate. In the presence of an overwhelming constellation of malformations, the hypernasality may be overlooked.
44. **Timing Palate Surgery**

The optimum timing of palate surgery has been, and still is, a much debated subject. Before the advent of anesthesia, palate closure was postponed until adulthood. Today, with endotracheal anesthesia, palate closure is possible at any age. It is logical that the sooner the palate cleft is closed the easier it will be for the patient to develop normal speech patterns, avoiding bad habits gained during compensation for an inadequate velopharyngeal sphincter. Thus the most popular time for closure of a palate cleft has been set at about 18 months of age, or the time when speech begins to develop.

All reports on the effect of the age at the time of palate closure indicate that the earlier the operation, the more normal the speech. Holdsworth reported that his youngest group, 6 to 9 months, had 77 percent apparently normal speech as opposed to the next age, 10 to 12 months, with 54 percent normal speech. Peet’s earliest group, 12 to 15 months and 170 V-Y “pushbacks,” showed 82 percent without nasal escape as compared to the next age, 16 to 30 months, with 77 percent. Axhausen found that patients with palates closed at 3 years had 80 percent normal speech, while those with palates closed at 4 to 6 years had only 71 percent. Lindsay found 70 percent acceptable speech when the palate was closed before 3 years and only 60 percent after 3 years. Jolley also found that the cutoff time for good speech was before 3 years. Calnan reported normal speech in 75 percent when operation took place between 1 and 2 years of age but only 40 percent at 4 years.

In 1964 Michael Lewin of New York published the answers to a questionnaire sent to all American and Canadian plastic sur-
geons in 1962. Of the 284 surgeons responding, 80.6 percent close the palate cleft between 1 and 2 years of age, with 18 months being the age of choice; 11.9 percent delay operation until the patient is between 2 and 4 years old. Only 15 surgeons operate on the palate before 12 months of age.

In 1972 S. O’Riain and B. N. Hammond of Odstock Hospital, England, noted that the majority of British surgeons, Battle (1954) and Braithwaite (1966) included, advocate operation at 1 year old or before the age of 18 months.

In 1976 Innes stated:

I believe, with Kilner, that the ideal time for the primary repair of all types of cleft palate is as early as possible. . . . In almost all cases conditions are right for successful surgery at the age of one year.

EARLY FOR SPEECH

Most speech pathologists favor early cleft palate closure because they are convinced that the speech results will be better with less effort. By 1964 Madame Borel-Maisonny had been P. Petit’s speech analyst long enough to follow 100 cleft cases and report the percentage of patients who recovered “normal” function of the soft palate during phonation—60 cases of 100 = 60 percent. Then she presented her statistics to probe the necessity of performing cleft palate operation before the age of 2 with the purpose of obtaining normal speech. In her study, she found that speech was normal in 63 patients, or 66 percent, operated on before 2 years of age but in only 37, or 33.4 percent, operated on after age 2.

Richard Cole, once speech therapist of the Lancaster Cleft Palate Clinic and chairman of the 1969 Symposium on the Early Treatment of Cleft Lip and Palate, and now at the University of Detroit Dental School, was quite definite about his feelings in reference to early palate closure and speech:

The fact that seems rather clear is that articulatory proficiency is related to the age at which palatal adequacy is established, but that there is a very definitely greater likelihood of encountering what we refer to as “cleft palate
speech” including glottal stops, pharyngeal fricatives, the later palatal adequacy is established. Integrity need not necessarily be by surgery as prosthetic separation may serve the purpose.

I think that most speech therapists would agree that glottal stops and pharyngeal fricatives are the most difficult sound substitutions to alter in the speech of cleft palate persons, and we know that the older the patient when he begins speech therapy, the less chance we have of completely eliminating this habit.

In 1974 D. Evans and C. Renfrew of Churchill Hospital, Oxford University, made a speech assessment of 229 cleft palate patients treated by Eric Peet using a Wardill-Kilner V-Y palatoplasty. It is interesting that their study showed a slight advantage conferred by operation within the first eight months of life, and strong theoretical arguments concerning early speech development and the effect of maternal separation after this age were presented in support of early operation.

Muriel Morley of Newcastle, speech pathologist for Wardill and later Braithwaite, recalled in 1977:

Regular observation of the developing speech from preoperative times until normal speech was established indicated that the majority of these children, submitted to surgery before three years of age, eventually developed normal speech without any specialised assistance.

Hughlett Morris of the University of Iowa stated in 1977:

We’re caught between the fear, set off by Graber in the 1950s, that early surgery leads to midfacial growth deformities and the suspicion labeled by Morley, also in the 1950s, that late surgery leads to a higher incidence of velopharyngeal dysfunction and patterns of misarticulation than otherwise expected. There are more data to support the former theory than the latter though in neither case is the picture entirely clear. Naturally, as a speech pathologist, I urge that early palatal surgery be seriously considered.

Kenneth R. Bzoch of the University of Florida, of Czechoslovakian descent and a co-editor of the extensive 1971 Cleft Lip and Palate, wrote in 1977:

I noticed most authority figures in Speech Pathology were either stutterers or had aphasia or some other claim to the Communicative Disorders field and all I had was a cleft palate in utero (before fusion) so perhaps that’s why I focused on this field.
After over a quarter of a century of experience the state-of-the-art in cleft palate research has progressed to the point where a challenging clinical team goal can be set. The goal of achieving speech, language and hearing function indistinguishable from that of their peers by three years of age for cleft palate infants has three critical steps too often missing in treatment programs today. In order of priority these appear to be: (1) early complete reconstructive surgery of both the hard and soft palate clefts between 12 and 18 months of age, (2) establishment of a regular early effective home speech and language stimulation program in years 1 and 2, and (3) early critical evaluation of the efficacy of primary surgical closure followed directly by secondary reconstructive surgery before 3 years of age when velopharyngeal insufficiency is indicated by clinical and diagnostic therapy techniques.

On the Efficacy of Early Complete Palate Closure. My experience clearly indicates the achievement of early functional palatal reconstruction does make a marked difference in the life of children with congenital cleft palates. Research to date is inconclusive mainly because the timing of primary closure rather than the date of achievement of velopharyngeal sufficiency to support speech has been used in the past studies addressing this question. Empirically it appears that the abnormal speech stigmata requiring later prolonged speech therapy are usually avoided when functional palatal reconstruction is accomplished before 18 months of age. When closure is routinely postponed for any reason or when primary closure is not successful in achieving velopharyngeal adequacy, compensatory articulation, breathing, and phonation habits almost regularly emerge between two and four years of age. This is because the pneumatic requirements of speech physiology do require a complete and rapid coupling and uncoupling of the velopharyngeal mechanism. The use of expressive oral language for obtaining wants and needs is regularly evidenced in all infants from years one to three. The adequacy or inadequacy of primary closure can be determined from clinical tests during diagnostic therapy for any child between one and three.

Later for Maxillary Growth

In 1954 bone growth expert Wilton Krogman, while at the University of Pennsylvania, noted:

The palate grows relatively little after the age of about six years . . . [thus], an optimum time for surgical procedure which would be, both in theory and in practice, in accordance with growth dimension and growth potential, is somewhere between four and six years, with the earlier age acceptable in the great majority of cases. However, if the growth tempo in the individual child be such that there is evidence of advanced growth . . . it is possible that an earlier chronological age (as early as 2 to 3 years) may be permissible.
Bill Grabb called my attention to the 1968 Mosher Award winning work of Leslie Bernstein on the effect of timing of cleft palate operations on subsequent growth of the maxilla, published in *Laryngoscope*. I wrote Bernstein at the University of California, Sacramento, for recent information and was rewarded with a reprint and his permission to use any part for publication. The accompanying letter ended with

I am sure that your comments will be as fair and gracious as before.

Only those familiar with Chapter 33-1/3 in Volume I will appreciate the scalpel edge of this line, and I would take my hat off to him for it, if I had one.

Bernstein, with both dental and medical degrees, while in the Otolaryngology Department of the University of Iowa, made a study of 325 subjects with surgically corrected maxillofacial clefts and 49 without surgery of the palate defect. A von Langenbeck soft tissue closure had been done in 278 while 32 had a vomer flap several months prior to soft tissue closure. In 222 subjects the preoperative occlusion of the posterior teeth had been recorded in dental casts. All retained the preoperative occlusal relationships of the posterior teeth when examined postoperatively. Under the premise that “surgical assault on the palate interferes with its lateral growth,” he emphasized

the need to delay surgery on the cleft palate until the deciduous molars are in occlusion. It also serves as a very favorable argument in support of instituting pre-operative orthodontic expansion of the palate in those patients who have already developed a cross-bite malocclusion, because after surgery the cross-bite usually becomes worse.

The main thrust of his paper was shown with two diagrams of the coronal section through the palate and mandible to show cusp relationships in the normal and in bilateral crossbite. Bernstein stated:

The mandibular buccal cusps, articulating on the inside of the maxillary buccal cusps, exert a wedging effect which prevents medial contraction after cleft palate repair. This effect is cancelled out in cross-bite cases.
He concluded:

The results of this study indicate that growth and development of the maxilla, and the appearance of the mid-third of the face, are materially altered if the palatal operation is performed before all of the deciduous molars are in proper occlusion—namely, before the age of 24–30 months. . . . It is concluded that the optimal time for cleft palate repair is between 30 and 36 months of age.

In 1977, in Toronto, N. Robertson and A. Jolleys of the Welsh National School of Medicine, Cardiff, presented a seven-year follow-up on 40 newborn infants with unilateral complete clefts of the lip and palate. They had been divided into two groups handled skillfully and identically, except in group 1 the hard palate was closed at 12 months while in group 2 the hard palate was closed at 4½ years. Serial records had been kept from birth onward, and no real differences in maxillary growth had been noted at 3 and 5 years of age. At 7 years there was still no marked difference, but the delayed group showed slightly better maxillary development, especially in the upper arch by cephalometric measurements, slight disadvantage in speech and no difference in ear problems. Robertson and Jolleys concluded that the type and degree of the original cleft condition was more important!

The orthodontists facing dental and maxillary deformities following early traumatic surgery have been effective in pointing out the direct relationship between the time and type of surgery and the severity of the deformities. They have promoted both reduction of traumatic surgery and delay of surgery. At present there is some concern about the effect of elevation of mucoperiosteal flaps and maxillary growth retardation. Early premaxillary setbacks have been discouraged. Yet both of these surgical procedures, at the right time, are acceptable. As most maxillary growth has been completed by 3 to 5 years, this is the optimum time if extensive elevation of mucoperiosteum is required.

Some surgeons, sympathizing with the ideal early palate closure for the best speech results, follow early surgery with expansion plates and other devices to maintain arch alignment against the postoperative scar contracture. This is not a luxury I enjoy.
TIMING AND TYPE OF SURGERY

In 1973 Wolfgang Koberg of Düsseldorf reported on electronically analyzed data on 1,033 cleft patients in Rehrmann's clinic. For the previous five years, speech had been evaluated by a speech therapist and maxillary growth investigated by the "Dysgnathia Index." Koberg summarized:

A direct linear relationship is existent between the age of operation and speech result. . . . The measure of iatrogenic disturbance in maxillary growth depends on the technique applied in palatoplasty. . . . Without any vital damage to maxillary growth, a relatively early but "atraumatic" palatoplasty at the age of 2-3 years can be achieved.

He concluded with suggestions for type and timing of surgery:

1. The isolated median incomplete or complete cleft palate should be closed in one sitting after the second year of life, using the pediculated [Veau] flap technique. Prognosis for normal colloquial language rates 65-70%.

2. The uni- or bilateral total cleft palate cases, we . . . advocate closure in 2 sittings: at the age of 6 months, primary closure of anterior part of cleft palate according to Pichler, with simultaneous closure of cleft lip and alveolus. And at the age of 2 years, the plastic closure of residual cleft using the stalked or pediculated flap technique [Veau]. Where a primary Pichler-plasty is not usable, Andrä (1966) still thinks that Schweckendiek's procedure should be attempted at the age of 12 months. The prognosis for acquiring a flawless colloquial language verges on 70% for unilateral complete clefts, and about 65% for bilateral cases.

3. Isolated clefts of the velum should be operated at two years of age, using the pedicle flap technique. To elongate the nasal mucosa, incisions [Z] should be made as suggested by Schuchardt (1966). This operation should be performed in spite of the expected unfavourable anatomical results. A favourable prognosis for very or fairly good articulation verges on 85%.

4. Submucous cleft palate should, without exception, be repaired with the bridge flap technique [von Langenbeck or Axhausen]. With nearly 90% incontestable functional speech result, these clefts have the most favourable prognosis.

EVEN EARLIER SURGERY

There is a new push on for very early palate surgery, including mucoperiosteal dissections.
In 1977, at the American Cleft Palate Educational Foundation Symposium on the Refinements in Cleft Lip and Palate Surgery held at Northwestern University, Ernest Kaplan of Stanford University made a strong argument for early total closure of the palate cleft. He set 4 to 6 months as the optimum time for palate closure to avoid bad speech habits, stating that speech results are better if the palate is closed at six months.

He cited the surgeons who have spoken out for earlier palate surgery and its linear relation to better speech results.

Kaplan further backed his argument for very early total palate surgery by his study of Central American Indians. As found by others, he had noted normal facial growth in the adult unoperated cleft case. Where the lip cleft had been closed surgically but the palate left unoperated, he found maxillary retrusion. In two specific examples in which the palate had been closed but the lip left open, there was normal maxillary growth.

Kaplan also observed that clefts of the secondary palate, including submucous clefts (not including Pierre Robin and Treacher Collins syndromes), often show maxillary retrusion. He suggested that early surgery of the palate in these cases should not be blamed except for possible slight crossbite deformities.

The palate operation he proposed was the standard V-Y Wardill-Kilner palatoplasty, vomer flap, freeing, uniting into a loop and retroposing the levator muscle fibers, and, in about 50 percent of cases, dividing the nasal mucosa from the hard palate edge and sliding into this defect a buccal mucosal flap.

At the same meeting, Desmond Kernahan, who has migrated from Oxford to Liverpool to Winnipeg to Chicago’s Children’s Memorial Hospital, also rose to the rostrum to champion early palate closure.

LOGICAL TIMING

Franz Härle of the University of Freiburg escapes in the summer to the near-deserted fishing village of Pelion in the Aegean Sea to hunt with the fishermen. Yet the majority of his energy is spent in orthodontic surgery, rat research on osteoplasty effects on
mid-face growth and the logical design of treatment timing of palate clefts. It is his conception that with each period of independence in the developing child there comes a greater effort to express himself, and thus orthodontia and surgery should be coordinated in time to facilitate these spurts of vocal effort. As he wrote in 1977:

In 1967 I formed a team with pediatricians, phonodiologists, otorhinologists and psychologists to care for about 1000 cleft children. Our main concern was to work out the most appropriate time for the treatment of the cleft child, taking into consideration the psyche, the speech development and the facial growth. The result of this study is described in German as "Freiburger Pfeffermühle" or "pepper-mill of Freiburg." The pepper-mill symbolises the phases of speech differentiation and undifferentiation. Operations in the soft palate and pharynx are only valuable when done in the phase of speech indifferentiation, so that the children can make use of the new anatomical situation when entering the next phase of speech differentiation. At the age of 20 years, and after complete dental rehabilitation, normal speech, satisfactory aesthetics and corrected occlusion, the cleft patient leaves our regular control.

**FREIBURG'S PEPPER-GRINDER**

**PHASE-SPECIFIC DEVELOPMENT**

**OF THE CLEFT-CHILD**
GENERAL PERSONAL TIMING

In Miami at present I have no presurgical orthopedic aid available and thus have been forced into specialized staged surgical action.

Cleft of the velum
At 6 to 9 months retropositioning of the levator muscle into a sling and closure of the soft palate cleft in three layers are done. If the velum is short with good mobility, at 5 years a pushback with an island flap is used. If it is severely short, both an island and a posterior pharyngeal flap will be used in the pushback.

Submucous cleft
At 6 to 9 months, excision of the cleft area and retropositioning of the levator muscle with construction of a sling is followed by three-layer closure. If at 3 to 5 years there is velopharyngeal incompetence with good mobility but shortness, a pushback is used with a bipedicle island flap to the nasal defect. If the mobility is poor, a wide pharyngeal flap will be preferred.

Severe horseshoe cleft of the secondary palate
At 6 to 9 months, the soft palate is closed as much as possible. If molding and growth reduce the hard palate cleft enough, a modified von Langenbeck operation may close the hard palate at 18 months to 2 years. If the defect is still too large, there are two possibilities. The defect can be filled with an obturator or covered with a plate until 4 to 5 years, when an island flap and a pharyngeal flap can be used to aid pushback and closure. Another possibility at 18 months is to fill the nasal side of the cleft with a superiorly based pharyngeal flap and then close the oral side with the aid of von Langenbeck flaps.

Complete unilateral cleft
At 3 weeks of age, ear tubes are inserted, a lip adhesion is constructed and the soft palate is closed as much as possible. It is a temptation to turn a vomer flap at this time to close the anterior cleft but this is usually avoided. At 6 to 8 months, the definitive rotation-advancement lip closure is achieved. At 18
months, the vomer flap and a modified von Langenbeck (minimal incisions and undermining) can be used to close the hard palate defect. If the hard palate defect is too large, requiring extensive surgery, then a plate or obturator is utilized until the age of 3 to 5 years, when more radical surgery can be tolerated. Depending on the shortness of the palate and the amount of soft palate tissue, a pushback with an island flap and/or a pharyngeal flap will be employed.

**Complete bilateral cleft**

Rubber band traction to a headcap is started against the projecting premaxilla as soon after birth as possible. When the premaxilla is back enough for lip closure and the infant is ready physically for surgery (1 to 2 months), ear tubes are inserted, the soft palate is closed as much as possible and the lip is closed in one stage, bringing the lateral muscles together and banking the fork.

Closure of the lip and soft palate will help mold the premaxilla, and with growth the size of the hard palate cleft should be reduced. At 18 months to 2 years, if the hard palate cleft is reasonable, a modified von Langenbeck will succeed; and if the premaxilla is in good position, the alveolar clefts can be closed. In the event of velopharyngeal incompetence, a pharyngeal flap is first choice at 5 years since in bilateral clefts the amount of mucoperiosteum is limited, reducing the donor area for an island flap.

**LOGIC OF VARIATION**

In Miami the basis of the general plan is first that the sooner the soft palate is approximated the sooner coordination begins between the velum and the pharyngeal musculature. Early adhesion or closure of the lip cleft molds the premaxilla and maxilla into alignment. This action, with the aid of growth, will decrease the hard palate defect, making possible a modified and less radical closure of the hard palate at optimum speech age. There is then a good chance that 75 percent of the patients will develop speech within normal limits without further surgery. The amount of
subsequent maxillary deformities is consequently reduced. At 5 years, when the major portion of the maxillary growth has been completed, more extensive and imaginative surgery is justified to correct velopharyngeal incompetence in the other 25 percent, so that by school age all should be well.

P. S.

While we are racing toward earlier surgery, it is well to ponder that there is an occasional patient who has had no closure of his palate cleft but has adjusted his pharyngeal musculature, tongue and palatal halves so that he can speak within normal limits. A 24-year-old black woman with an unoperated cleft of the soft palate and perfectly normal speech was seen when she brought her daughter with a cleft of the secondary palate into clinic. Timing of the surgery (in fact, surgery itself) was not important in this individual, but her child’s cleft will be closed as soon as it is physically feasible—I suppose.

The patients with palate clefts who can adapt without surgery are fascinating when you consider the problems operated palates experience. Then, to add further to the surgeon’s ego deflation, a discussion during the 1969 Chicago Cleft Palate Symposium is recalled, when Jan Dreyer of the University of the Witwatersrand, Johannesburg, announced:

Our cleft palate dogs are able to bring the two halves of the soft palate together after only two months and without treatment at all. Also they are able to lap food and they have no nasal bark.

Richard Cole of Lancaster countered:

In other words, they don’t say "Mark, Mark?"

Dreyer elaborated:

If these dogs get excited they do have a nasal bark but if not excited, they are able to control and approximate the two edges of their soft palates.
I N developing countries of the world, expediency in surgery, as in other aspects of life, must often take precedence over the ideal. When patients are forced to travel great distances to be treated by a plastic surgeon at all, as much as possible must be done for them at the one, and probably only, time. Seldom will they have a chance to return. If the lip cleft is closed, there will be less motivation to return as the palate cleft is not so easily visible. In these circumstances the surgeon is under pressure to try to close both the lip and the palate cleft in one operation. In the light of modern knowledge it may not be ideal for maxillary growth in the young child to carry out traumatic mucoperiosteal elevating procedures, but logic dictates that it is better to do “all” and take the chance of some secondary deformity than to settle for none or only part, thus retaining a portion of the residual cleft forever.

Of course, except for the time required, it is easier to do “all in one.” Closure of the alveolar and hard palate cleft is greatly facilitated by the exposure afforded by the open lip. For many years, and even today in many clinics in England, the alveolus and anterior hard palate were and are closed at the time of lip closure. The crossbites created are being studied and treated by orthodontists.

F A R I N A

Brazil is a vast country of contrasts encompassing such extremes as the tropical jungles of the Amazon infested with the deadly
piranha to the great industrial city of São Paulo with skyscrapers and innovative plastic surgeons like Roberto Farina. Since much of Brazil is undeveloped, many clefts must travel great distances for treatment; hence the motivation for a one-stage closure of the entire cleft. Farina, who had used the two-stage Veau successfully, wrote in 1958:

Today, we have come to think that one can easily do everything at once in certain cases.

He set the age at 1 year or older and reported 23 such cases with the average operating time of two hours and normal recuperation. His one-stage cheilognathouranostaphyoplasty included a Veau-type closure of the alveolus and hard palate extended to close the soft palate followed by a LeMesurier lip closure. He cited the advantages:

1. This is hardly any risk to the patient.
2. One avoids two operations and thus two anesthetics—a doubled risk.
3. One no longer has the difficulties which one had at the junction of the hard and soft palate in palatoplasties done in two stages as recommended by Veau.
4. The closure at the level of the alveolar arch becomes easier with a perfect reconstitution of the nostril floor, without having to fear the appearance of bucco-nasal or vestibular fistulae.
5. It is more economical for the patient.
6. The results, in general, are "marvelous" from an anatomical and morphological point of view.

The 1971 Melbourne International Congress was the site for simultaneous reporting by Manchester, Kaplan and Davies of simultaneous lip and palate closure in one operation.

M A N C H E S T E R

William Manchester of Auckland, New Zealand, is rather proud of his extensive closure of the alveolar and hard palate cleft along with his lip closure. This radical action is with the aid of presurgical orthodontics by Peat in the form of an expanding plate to spread the maxillae and simple elastic traction to restrain the premaxilla, so that by 5 months the segments are prepared. As he
wrote in the 1971 *Transactions* of the Melbourne International Congress:

Everything is now ready for the first stage of the programme which aims to repair not only the lip on both sides but the whole length of the hard palate as well. Thus, when the second stage is reached at the age of nine months, only a cleft in the soft palate remains.

Manchester's second stage involves a palatal pushback using the Cronin posterior advancement of the nasal mucoperiosteum and a V-Y retropositioning of the oral mucoperiosteum. This totally denudes the hard palate at 9 months of age; which outcome should give some interesting data after 20 years.

Manchester also has a third stage for rhinoplasty and columella lengthening at 15 years. Thus the number of surgeries is not remarkably reduced.

**KAPLAN**

Isaac Kaplan, born in the Orange Free State, served as an intelligence officer in the South African division of the 8th Army during World War II and received his degree after the war at the University of the Witwatersrand Medical School in Johannesburg. Trained by Sir Harold Gillies in England, he established a plastic surgery department in Beilinson Hospital, University of Tel Aviv, Israel, where, during the Yom Kippur War, he turned over an entire floor for care of the wounded. Kaplan, also a pioneer in the development and use of a continuous-wave carbon dioxide laser knife, in 1974 with Dresner, Gorodischer and Radin, recalled his work at Barsky's unit in Vietnam:

In 1 year (1968–1969) over 400 untreated cases of cleft lip and palate were seen at the Children's Medical Relief International Plastic Surgery Unit in Saigon. Their ages ranged from infancy well into adulthood. Because of the workload and the difficulties of multiple-staged operations, simultaneous repair of the lip and palate was carried out on some of the older patients (Kaplan and Wesser, 1971). The operation was found to be so technically feasible that it soon became routine for infants too.

An experiment was set up in Israel between 1970 and 1972 in which 13 cleft infants had the combined lip and palate closure at
3 months and a control group of 13 cleft infants had the lip closed at 3 months and the palate at between 10 and 12 months of age. The lip was closed by the LeMesurier or Millard operation, the palate by the von Langenbeck with no attempt at closure of the alveolus, primary bone grafting or premaxillary setback. Naturally, the psychological effect of early complete cleft closure lessened family tensions at this stage. No maxillary growth retardation had been noted after two to four years and none would be expected to be noticeable at that time. The incidence of ear infections was much less in the experimental one-stage group. Study of speech development was of interest:

Commencement of “babbling” in the experimental group was comparable with normal children whereas in the control group it was delayed.

Uttering of first sounds such as nasal sounds and sibilants commenced at the time expected for normal children and was delayed in the control group.

“One-word response” appeared in all these cases at the time expected for normal children, whereas in the control group this was delayed.

Two- to three-word response was delayed as compared to normal children but was manifest earlier than in the control group.

The initial nasal sounds on which intelligibility of phonation depends were judged to be adequate in 40 per cent whereas the remaining 60 per cent were comparable with the control group.

Word intelligibility was adequate in 50 per cent of the cases whereas the remaining 50 per cent were comparable with the control group.

Three of these children have now reached the age of fluent speech and speak normally with regard to length and sequence of the sentences although nasality is variable.
In 1977 Kaplan, enclosing these photographs, wrote:

We are at the moment preparing an article which will confirm our previous observations that the development of the maxilla in these cases of simultaneous repair of lip and palate is not significantly affected, provided the classical Langenbeck procedure is performed on the palate. The speech in these cases is highly satisfactory in a vast majority and our results compare very favorably with those of others.

**DAVIES' TRUE ALL-IN-ONE CLOSURE OF THE CLEFT**

David Davies of Capetown, South Africa, said at the International Congress in Melbourne, Australia, in 1971:

Our problem, which I am sure is common to all developing countries, is the difficulty of insuring adequate parental care and nutrition of these children until they are ready for operation. Farina approached this problem initially in adults by suggesting a one-stage repair. I was told many years ago that the late Eric Peet from Oxford repaired adult bilateral clefts in one stage while holidaying in India. However, most of these repairs were simple closures with no pushback and a high percentage of resultant fistulae. Unless one can produce a comparable or better result than a multistage procedure the operation should not be done. We started this operation at first tentatively in 1964, but the procedure has now become routine. . . . The lip is repaired with a Z-plasty, the alveolar defect grafted, and an extensive pushback done with the use of a Millard island flap. After the operation parental acceptance of the child is good and no further adjustments are necessary until the child is five or six years of age. . . .

A good speech result is the most important aim of the cleft palate repair. For this reason, the entire palate is closed before the child starts making any noises so that auditory feedback can be normal from the beginning; otherwise, it will result in habit patterns which are difficult to break. This is particularly important in the less intelligent patient. It has often been noted on cineradiography that an intelligent child will be able to use the pharyngeal muscles for a slightly short palate and still have good speech results.

In a series of 85 complete clefts of the lip and palate closed in one stage at 3½ months, with more than 60 percent of the children black, Davies reported 22 out of 27 at normal or near normal resonance (82 percent). His impression is that the longer, more mobile palate resulting from the island flap is giving much
better speech results, but would like to wait until he has 50 children over 6 years of age before making a final comparison with other series.

He does not agree with the orthodontists’ objections to an island flap on the ground that it leaves a large raw area and the resultant scarring causes collapse. The raw area epithelializes in two to three weeks. No decrease in vault space has been noted in any of the cases. Davies feels that in the one-stage closure there is less surgical insult than in the multiple-staged procedure since the operation is easier. This claim is borne out by the fact that he had only two fistulae in 85 cases, one of which closed spontaneously.

In 1972, in a paper prepared for the Journal of the South African Speech and Hearing Association, D. Davies, D. M. Whitting, B. H. Miller, B. J. Cremin and D. Morrison gave an extensive report on 95 cases of one-stage closure. They discussed all aspects, but the one of greatest concern was orthodontic assessment.

Results show that 47% of these patients have a Class III relationship of the incisor teeth, i.e., the lower incisors occluding in front of the upper. The remaining 53% have a Class I (or normal) occlusion. The maxillary minor segment showed varying degrees of collapse in 66.7% of cases. This was assessed by relating the teeth on the minor segment to the opposing mandibular teeth and noting the amount of collapse towards the midline.

Although the percentage of cases showing Class III incisor relationship and collapse of the minor segment may appear to be high, these figures compare quite favorably with those of other published series. Also, in many of these cases, simple orthodontic treatment is all that will be required to bring about correction.

In reference to speech assessment the senior speech therapist of this group, D. M. Whitting, wrote in 1972:

95% of the children did not have normal speech. They had normal or near normal nasal resonance. 60% had normal or near normal articulation. I think we will have to wait till we have assessments of 50 children over the age of 6 years before we can quote figures suitable for comparison with other series. Articulatory development is not stabilized in the normal child before 6-8 years.
LÖSKEN AND OTHERS

In 1973 H. Wolfgang Løsken, trained by David Davies in Capetown and as a Maytag Fellow in Miami, wrote after one year of practice in Pietermaritzburg, South Africa:

The most exciting repair of all was when I did my first all-in-one cleft repair on a five months old complete cleft of the lip and palate, with a gap in the alveolus of 1.7 cm.

Løsken’s next letter was written in 1974, and an excerpt from it is of interest:

I have now done three complete repairs of the lip and palate together with no fistulae. At our recent Congress in Johannesburg, Davies presented the results of his 110 complete one-stage repairs. He was certainly tremendously honest in his presentation and very critical of his own work and results. He is certainly a tremendous example to us younger surgeons who tend to be over-enthusiastic and possibly not sufficiently critical of our own work and results.

I got the impression from David that he felt it was still a little early to come to any conclusions about his results or felt one would have to wait a long time before being certain.

In 1977 Davies wrote:

In spite of criticisms from our orthodontic colleagues I feel that there is a definite place for a more radical approach to cleft lip and palate surgery. We have completed 155 cases repairing the lip and palatal clefts in one operation and the last fifteen cases have been neo-nates six days of age. Technically the operation is not a difficult one for the surgeon and providing that the anaesthesia is superb there does not appear to be any increased risk for the child. Eight of these cases needed no blood transfusion as their measured blood loss was less than 5% of their total blood volume. Neo-nates are very resilient as they are still protected by maternal antibodies and in fact from the physiological point of view the infant is more vulnerable at three months of age when most surgeons start their operative procedures.

Early radical surgery is ideally suited to the 75% of the world that is under supplied with medical care and has socio-economic problems unknown in the States. I was most interested to hear from Fernando Monasterio that his mobile units and the people that they have trained in outlying parts of Mexico have completed no fewer than 1000 radical repairs.
Brown and McDowell stated in 1945:

It is unfortunate that cleft lips so frequently coexist with cleft palate, causing many surgeons to be pre-occupied with closing part of the palate at the same time the lip is closed. Aside from the probability that early surgical treatment to the palate may result in unnecessary dental damage, it seems to us that good repair of the lip is difficult enough to require the surgeon’s individual attention in the process.

Of course, it depends on the circumstances involved in the specific case. If it is a question of “all at once” or “only a part ever,” then the “all in one” principle is the wiser choice. Under the usual modern conditions, speed or “all in one” is not the goal. The surgeon must design the number of stages, sequence of closures and optimal age for each, guided by his circumstances, his experience and that of the other specialty members of his team.
A cleft is indicated by stippling, a submucous cleft or submucous distortion by horizontal lines.
This chapter traces the evolution of my personal quest for a surgical solution to the cleft palate problem as exemplified by a sample of pertinent cases, many of which are the palatal follow-up of cleft lip cases presented in Volumes I and II. My actions were first influenced by training and later by experience. Observations of von Langenbeck procedures at Harvard and Vanderbilt in the mid-1940's revealed closure without lengthening and about a 70 percent good speech result. Kilner, Peet and Wardill in England in the late 1940's impressed me with the V-Y lengthening, and even without nasal lining release more than 75 percent obtained good speech but with some arch collapse. Brown, Byars and McDowell in St. Louis in the early 1950's demonstrated the Dorrance incision and radical nasal lining release during the pushback leaving large raw areas, but they obtained similar speech results and some arch collapse. My first cases combined V-Y lengthening with nasal lining release and left a raw area.

**Primary Surgery**

**V-Y Pushback**

CASE 1 was born with a unilateral incomplete cleft of the lip and the palate but with an intact alveolus. In 1957 rotation-advancement of the lip cleft was successful.

In 1958, at age 1 year, a Kilner-Wardill type of three-flap V-Y pushback procedure was carried out leaving a portion of the nasal lining raw (stippling).

In 1960 Berkowitz first noticed a crossbite developing, on which he commented in 1963:
Unilateral crossbite of the right buccal segment due to medial collapse of the alveolus. Suggest expansion.

When seen in the cleft palate clinic in 1967, the patient was found to have normal speech and hearing, and a full-banded appliance was in place on both upper and lower arches. When last seen in 1977, the patient was a corporal in the army, with good occlusion and normal speech and hearing.

CASE 2 was born with an incomplete cleft of the lip, severe nasal distortion and a complete cleft of the hard and soft palate.

On 4-15-59 the lip was closed by means of the rotation-advancement method. On 3-23-60, at about 12 months of age, a vomer flap was used to close the anterior hard palate and a four-flap Wardill V-Y procedure achieved some pushback, leaving a raw nasal area at the junction of the soft and hard palate. A small fistula formed.

In 1966 the maxillary teeth were behind the mandibular teeth. In 1967 collapse of the cleft segment with crossbite necessitated placement of an upper, fixed lingual arch wire for maxillary expansion. On 6-18-68, following maxillary expansion, at the age of 9 years rib bone grafts were used as struts and chips to fit between the alveolar segments, overlay the arch and underlay the alar bases. In 1970 a palatal space retainer was adequate. Speech was normal. On 7-1-76, at age 17 years, a cleft lip rhinoplasty was carried out.

**Bottom line:** Concerned about the limited lengthening, the nasal lining raw area, the inevitable contracture and possible fistulae, I conceived the island flap method of pushback in 1960.

**EARLY PUSHBACK WITH ISLAND FLAP UNILATERAL CLEFTS.** CASE 3 was born with a cleft of the soft palate which was treated at 19 months of age with a horseshoe shaped pushback using a unilateral island flap taken from the anterior mucoperiosteum and
crossing the midline 1 cm. Thomas Cronin of Houston observed the surgery and admitted with a slight wince that there might be a place in palate surgery for the island flap.

Healing was uneventful, and at his first cleft palate clinic evaluation the patient presented normal speech but a slight hearing loss, otitis media, enlarged tonsils and adenoids and allergies. Conservative treatment including anti-allergic therapy was instituted, and in 1967 Pigott pronounced the patient a perfect speaker, a judgment endorsed in cleft palate clinic in 1969, 1970, 1972, 1975 and 1976. Orthodontic expansion by R. Litowitz was started in 1971 for a mild "hourglass" deformity with crossbite of the upper left second bicuspid amenable to orthodontia. When last seen in 1976 the patient had a long, mobile palate with normal speech. At that time photographs were taken of his palate at rest and elevated into a competent sphincter while phonating "Aaahh."

CASE 4 was born with a cleft of the soft palate which was treated at 1 year of age with a horseshoe shaped pushback, a right island flap being used to fill the nasal defect after release of the soft from the hard palate. At age 12 years the patient began orthodontic treatment for crossbite and at age 16 years was reported by Berkowitz to have excellent palatal arch development and good occlusion.
CASE 5 was born with a cleft of the soft palate extending into the hard palate. At 10 months of age a V-Y pushback was performed; a 1.5 cm. wide right island flap was used to fill the nasal lining defect after release of the soft palate from the hard palate edge. The levator muscles were detached from the edge of the hard palate, dissected into two bundles, retropositioned and sutured together into an intact loop. The surgery took 43 minutes.

From the age of 3½ years the patient has had normal speech, but Berkowitz noted a narrowing of her upper dental arch.

At age 6 years (1975), this record of the contracted arch was taken by Berkowitz; subsequently, a palate expander was used to spread the arch.
CASE 6 was born with a severe complete unilateral cleft of the lip and palate which was treated at 3 weeks by a lip adhesion with no undermining of the lateral lip segment from the maxilla. At 3 months of age the anterior cleft was closed with mucoperiosteal flaps from the cleft edge for nasal closure and excess mucosa from the lip adhesion for oral closure. At the same time, the lip was approximated with the rotation-advancement method. In 1971, at 14 months of age, a V-Y pushback was accomplished with a 1.5 cm. left island flap inserted into the release of the nasal lining.
Levator muscle attachments were freed from the hard palate edge, dissected into bundles and sutured into an intact muscle loop.

In 1973 the patient revealed mild right buccal and anterior crossbite, which was treated successfully with expansion orthodontia. In 1977, at age 7 years, speech was normal except for a lisp. Correct occlusion is being maintained by a retainer. Nasal correction at 16 years is planned.

*Bottom line:* Here, an early pushback with island flap achieved normal speech with only moderate maxillary crossbite, corrected early with orthodontia. But maxillary deformity is usually greater than in this case.

CASE 7 was born with an incomplete cleft of the lip, notch of the alveolus and cleft of the soft and the distal third of the hard palate. At 2 months of age rotation-advancement achieved lip closure. At 1 year a V-Y pushback of the palate used a right mucoperiosteal island flap, 1.25 cm. wide, to fill the nasal defect. The hamulus was fractured bilaterally, and closure of the palate was achieved in three layers with excellent healing.

When last seen at age 10 years, after Berkowitz removed the maxillary expander, the patient revealed a good arch and good occlusion with a long, mobile palate producing normal speech.
CASE 8 was born with a short, cleft soft palate. At age 2 years she had a V-Y palate pushback preserving an anterior V-wedge of mucoperiosteum. After release of the soft from the hard palate, a 1.5 cm. wide left island flap was inserted into the nasal lining defect. The levator muscles were freed from the hard palate, dissected into bundles and sutured into a retroposed, intact muscle loop.

Short, cleft soft palate
Splitting edges
Horseshoe incision
Left mucoperiosteal flap being elevated
CASE 9, showing a moderate cleft of all of the soft and a portion of the hard palate with thin soft tissue elements, was treated with a pushback using a unilateral island flap at 14 months of age, with no fracture of the hamulus. Peter Randall assisted.
At age 6 years basal and lateral view videofluoroscopic evaluations were conducted during isolated and connected speech. The lateral view indicated light contact, but the basal view was somewhat obscure, suggesting no contact in connected speech. Because the patient’s speech was “incredibly” good, intensive speech therapy was instituted. At age 7 years speech seemed near normal and occlusion of the teeth was good, but an expansion wire was still in place.

CASE 10 was born with an incomplete unilateral cleft of the lip, an alveolar notch and a cleft of the soft and part of the hard palate. At age 3 months the lip was closed with the rotation-advancement method. At 11 months a V-Y pushback was done using a right mucoperiosteal island flap to fill the nasal lining defect. At 8 years of age the child has normal speech but requires maxillary expansion to improve occlusion.

CASE 11 was born with a complete unilateral cleft of the lip and palate. A lip adhesion was performed at 3 weeks of age to mold the maxillae and was followed with a rotation-advancement lip closure at 5 months. On 2-18-72, at age 10 months, the palatal arch molding and growth were good. On 9-1-72, at 16 months, the hard palate cleft was closed with a turnover vomer flap and the soft palate lengthened by a V-Y pushback using a right island flap to fill the nasal lining defect.
On 2-7-73, the palatal scar had brought the lesser segment medially into buccal crossbite. Palatal form had stabilized by 9-6-73.

At age 4 years and again at 7 years speech reported normal.

After orthodontia; excellent palatal development
Bottom line: The "early island" gave good speech in 80 percent of the patients, with minor orthodontic correction required in 70 percent. The maxillary distortion in some cases was so great that it not only troubled the orthodontist but may require a Le Fort I advancement in a rare case. This possibility is seen in a couple of the following four cases.

CASE 12 was born with an incomplete cleft of the lip but a wide cleft of the palate. At 2½ months rotation-advancement closed the lip and at 7½ months was followed by closure of the hard palate cleft with a vomer flap. At 18 months a V-Y pushback was performed with a right island flap inserted into the nasal lining defect. Normal speech was noted at 3 years, and speech is still normal at 13 years. Development of the maxilla was not so normal: This is one of the cases recorded by Berkowitz which caused me to stop the early island flaps!

Berkowitz noted:
The too-early island flap resulted in maxillary growth inhibition in all three dimensions. Both lateral segments were drawn medially, obliterating the palatal vault space. Anterior-posterior growth disturbance resulted in arch length deficiency with crowding of teeth and anterior cross-bite.

Orthodontia has begun to expand and advance the upper arch. Maxillary osteotomy may be required eventually if orthodontia cannot correct the deformity.
CASE 13 had a severe cleft of the lip, alveolus and palate with a deficiency of the maxilla and distortion of the nose. At 3 months of age rotation-advancement closure of the lip was carried out. The anterior palate cleft was closed at 18 months with a vomer flap. The patient had excellent arch form and palatal segmental relationship before the island flap, as seen in Berkwitz’ model.

At 21 months a V-Y pushback of the palate was achieved, leaving the V over the anterior closure untouched and taking a unilateral island flap for nasal lining. Nine months after palate surgery, models show the transverse palatal scar causing medial pull of the lesser segment and carrying the buccal teeth into crossbite.

Palatal expansion widened the arch, correcting the crossbite. On 11-10-70 an iliac bone graft was placed across the cleft. Removal of orthodontic retention permitted medial relapse of the lateral palatal segments, resulting in palatal narrowing and severe buccal crossbite (11-73).
After orthodontic advancement and expansion, occlusion was achieved in spite of the palatal scar, but fixed retention will be necessary to maintain arch form integrity (7-78). At age 10 years the patient had normal speech. Cephaloradiographs made in 1978 by Berkowitz show the velum contacting the pharyngeal wall with vocalization of u.

CASE 14 was born with a severe right complete cleft of the lip and palate with maxillary deficiency. At 4 months of age the anterior palate cleft was closed with a Burian flap and the lip with rotation-advancement. At 15 months maxillary expansion was started with a pinned screw plate, and at 20 months of age rib bone grafts were inserted across and into the cleft. At 2½ years, a V-Y palatal pushback was done using a left island flap to fill the nasal lining defect after release of the soft palate from the hard palate edge.
At age 12 years the patient has a long, mobile palate with good speech. The original deficiency of the maxilla, the bone graft at 20 months and the denudement of the hard palate by the island flap at 2½ years seem to have influenced maxillary development with resulting arch contracture and crossbite. Treatment by expansion springs has brought improvement in occlusion, but it is possible that the patient will require maxillary onlay grafts at 15 to 16 years.

CASE 15 was born with a complete unilateral cleft of the lip and palate with a severe nasal deformity. At 2½ months the anterior palate was closed with cleft edge mucoperiosteal flaps, covered on the nasal side by a mucosal flap of the inferior turbinate based inferiorly after removal of bone. At 3½ months the lip was closed by the rotation-advancement method, but without nasal tip correction. At 11 months the alveolar and anterior hard palate cleft was filled with a rib bone graft and covered with a Burian labial flap. At 18 months a V-Y pushback of the palate was performed using a unilateral right island flap.

When the patient was seen in cleft palate clinic in 1971 at age 5 years, the speech pathologist reported that speech was essentially normal. The orthodontist noted collapse of the maxillary segments and an anterior crossbite deserving orthodontic treatment.
Orthodontic expansion of the maxilla was started, with improvement in occlusion. This treatment will continue, and at age 16 years a corrective rhinoplasty will improve the boy’s appearance.

5-76 After buccal expansion and occlusal correction
At rest
“Aaahh”

Although a convenient, early anterior cleft closure and normal speech were obtained and intentions were the best, using the turbinate in the anterior closure possibly started maxillary collapse. Then the early bone graft may have retarded growth. Subsequent early denudation of the maxilla with mucoperiosteal flaps and an island further affected the maxilla so that its growth was abnormal. It is remarkable that orthodontia seems to be expanding the arch with correction of the occlusion.

**Bottom line:** The temptation of convenience of anterior cleft closure and early pushback with an island for good early speech must be resisted until maxillary growth has progressed to 5 years.

BILATERAL CLEFTS—INCOMPLETE. CASE 16 was born with an incomplete bilateral cleft of the lip and a cleft of the soft palate. At 4½ months of age a bilateral rotation-advancement closure of the lip, without columella lengthening, was done. At 28 months a V-Y pushback of the
palate, leaving a V anteriorly, was achieved using a 1.25 cm. wide right island flap to fill the nasal defect. The levator muscles were freed from the edge of the hard palate, dissected into bundles and sutured into an intact loop.

When the patient was seen in cleft palate clinic in 1972, at the age of 4 years, her speech was normal but a severe buccal crossbite of the molars was noted and orthodontic therapy advised. When seen in 1978 at the age of 10 years, she still had normal speech, but as no orthodontia had been used the crossbite was still present. The patient is now receiving dental correction.

BILATERAL CLEFTS—COMPLETE. CASE 17 was born with complete bilateral clefts of the lip and palate, a projecting premaxilla and almost no columella. Rubber band traction was started at 7 days, and at 3 months of age the lateral lip mucosa and muscles were joined in the midline behind the prolabium and a forked flap was banked in whisker position.

11-19-69 Before rubber band 11-26-69 During rubber band traction

8-7-70 After closure of lip and fork banking
Berkowitz described the condition of the maxillae after lip closure:

Excellent approximation of the premaxillary and lateral palatal processes with marked reduction in the cleft space.

Two and a half months later, on 4-23-70, the banked fork was advanced into the columella. 12-10-70, at age 13 months, a V-Y pushback was achieved using a right mucoperiosteal island flap to fill a 1 cm. nasal lining defect. Vomer flaps were turned laterally for nasal closure. The levator muscles were dissected free and sutured into an intact muscle loop. A postoperative suction test was positive.

In 1974 otological examination revealed fluid in the right ear and retraction of the left superior tympanic membrane. Bilateral myringotomy was followed by insertion of P.E. tubes. Cleft palate clinic evaluation of speech and hearing found no abnormalities in 1976 and again in 1978, but the orthodontist's report was not so happy. Berkowitz reported on the 1974 models:

After island flap and palatal cleft closure at 13 months, the resulting palatal scar tissue caused distortion with bilateral crossbite. Where the premaxilla was in extreme overjet at the newborn period, at the age of five years, the incisor teeth were in tip-to-tip relationship due to diminished anterior-posterior maxillary growth.

Maxillary expansion was advised.

**Bottom line:** In complete bilateral clefts, as in complete unilateral clefts, extensive elevation of mucoperiosteal flaps before 5 years of age is contraindicated because of scar retardation of maxillary growth. In these double clefts it is possible that there will never be enough expendable mucoperiosteum for an island flap. Once the premaxilla and maxillae have been positioned normally and stabilized through the growth period (Latham-Georgiade approach), an island flap is sometimes available. At the
moment, despite the good speech with the primary island flap, I
do not use it in bilateral clefts until the teenage years.

LATER PRIMARY ISLAND FLAPS (AFTER
AGE 5 YEARS)
In certain cases the diagnosis or opportunity for operation may be
delayed.

CASE 18 was born with a wide cleft of the soft and half of the hard
palate. On 1-18-61, at about the age of 5 years, a V-Y pushback was
achieved, taking a small atypical island flap for transverse lengthening of the
nasal lining. A superiorly based pharyngeal flap was used primarily to close
the nasal side of the soft palate cleft.
In 1967 cleft palate clinic evaluation reported class I bimaxillary protrusion with a lingual crossbite of the upper left first molar. Speech was normal. Last evaluation in 1977 found teeth in good occlusion and speech normal.

CASE 19 was born with an apparently normal palate. Following T & A the patient developed nasal escape in speech; cephalometric studies by Berkowitz revealed an incompetent velopharyngeal sphincter at age 10 years. Mobility seemed good, so on 5-9-73 a pushback of the palate was performed with a bipedicle island flap inserted into the nasal lining defect. Healing was uneventful, with the donor area of the island epithelialized in three weeks. Speech pathologist Bensen reported a long, mobile palate with normal speech in 1974, and again in 1976.

CASE 20 was born with a submucous cleft palate with a V defect in the hard palate, cleft in the muscle with mucosa intact and a bifid uvula. Following T & A at 5 years the patient began to have nasal escape. Cephalometric studies by Berkowitz revealed a 1 cm. gap between the velum and pharynx on vocalization of u.

On 10-11-73, at the age of 6 years, a horseshoe-shaped incision was used to dissect the mucoperiosteum. Bilateral neurovascular bundles were freed.
CASE 21 was born with a submucous cleft palate with a hard palate defect, cleft in the muscle, bifid uvula, short velum and nasal escape in speech. On 1-21-71, at age 6 years, a pushback was accomplished using an anterior bipedicle island flap transposed into the nasal lining release.

Cleft palate clinic evaluations in 1975 and 1977 found normal speech and hearing. The maxillary arch was narrowed and the vault space diminished. Buccal crossbite existed at both maxillary molars. In 1978 expansion wires to correct a maxillary hourglass deformity were in position. The palate was mobile and producing normal speech.
In 1975 orthodontic braces were used for maxillary expansion. In 1976 evaluation in cleft palate clinic found good occlusion with no crossbite and a long, mobile palate with normal speech. Photos taken in 1977 show the musculus uvulae contracting into an "inchworm."

CASE 22 was born with a submucous cleft palate which caused difficulty in speech. At age 10 years, the speech pathologist in cleft palate clinic noted excellent movement of the palate and lateral pharyngeal walls, a short palate, nasal escape and good articulation. On 10-20-76, at age 10 years, a portion of the translucent area of the velum was excised and the edges of the bifid uvula were pared prior to a three-layer closure of the soft palate. A horseshoe-shaped incision well away from the teeth allowed elevation of a mucoperiosteal flap. The anterior portion, taken as a bipedicle island flap, was used to fill the nasal lining defect after release of the soft palate from its attachments to the hard palate.
Two years after surgery the palate seemed slightly short; there was good speech with individual words and slight nasal escape in conversation. Cephalometric studies by Berkowitz revealed good velopharyngeal closure, but Dickson's nasendoscopy found a slit-like aperture during velopharyngeal closure for speech in the area of the right lateral port and a probable similar condition on the left. The posterior palate translucency indicated a thinness in this submucous cleft.
Assessment: Mild hypernasality; moderately good lateral wall and velar activity; short palate. This does not appear to be a problem which will be alleviated by speech therapy alone. The parents refuse further surgery (pharyngeal flap), but, as the hypernasality is of a mild degree and probably does not present too much difficulty, it can be postponed for now.

CASE 23 was born in Haiti with a cleft of the entire soft palate and two-thirds of the hard palate. The cleft was over 1 cm. wide at the greatest point and remained untreated until, at the age of 6 years, the patient was first seen presenting typical nasal cleft palate speech.

On 9-21-66 a V-Y pushback of the palate was carried out using a specially designed bipedicle island flap for nasal lining.

In 1967 Pigott studied this palate with his early method of endoscopy eight months postoperatively and reported only minor nasal emission. Speech therapy was advised.

In 1971, 1973 and 1974 the patient was evaluated in cleft palate clinic by all disciplines and reported to have normal speech, normal hearing and normal occlusion. He was last seen in 1978 with an excellent result.
CASE 24 had always had difficulty with speech. At the age of 30 years, evaluation in the cleft palate clinic reported:

Gross nasal emission is present on all consonants. . . . He uses the substitution of a glottal stop for many phonemes. He is now expressing a concern about the low degree of intelligibility. There is suspicion of congenital palatal insufficiency.

Cephalometric studies by Berkowitz revealed an A-P gap of at least 1 cm. On 5-30-73 a V-Y pushback was performed, the nasal lining defect being filled with a 1.25 cm. wide right island flap. The details of the surgical steps were recorded photographically.

Anterior island donor area marked
Horseshoe-shaped incision being made
Mucoperiosteal flap dissected off bone

Freening left neurovascular bundle for pushback
Releasing nasal lining from hard palate (No. 11 BP blade)
Freening island neurovascular bundle (No. 15 BP blade)

Cutting the mucoperiosteal island free
Island poised to go into nasal defect
Key sutures in the distal ends of island for placement in defect
Island sutured into nasal defect

Pushback with island flap completed

Postoperatively: At rest (above) and positive suction test (below)

Even when a potentially normal palate is constructed at age 30 years, speech therapy is indicated to eradicate bad habits.

**Bottom line:** The later primary island flap achieved good speech results in a high percentage of cases but with much less arch and dental distortion. The fortunate delay in surgery in these cases was due in great part to the late diagnosis of submucous cleft palate.

**PRIMARY PHARYNGEAL FLAP**

There are certain cases with palatal paralysis, large cleft defect, huge velopharyngeal gap or unavailable island flap in which a primary posterior pharyngeal flap may be of benefit.

CASE 25 revealed speech difficulty to the parents at 2 years of age. At cleft palate clinic the diagnosis of paralysis of the velum was treated by a speech aid ineffectively. On 6-4-69, at age 7 years, a 1 cm. superiorly based pharyngeal flap was attached to the upper surface of the velum and the raw flap partially covered with a turnback mucosal flap from the uvula.

On 9-13-70 our speech pathologist noted some improvement but recorded nasal escape on all fricatives and on some plosives in conversation. On 4-20-71, at age 9 years, a second, wider (2 cm.) superiorly based pharyngeal flap was attached along the free border of the velum and again partially covered with a turnback mucosal flap from the uvula. This maneuver was carried out just beneath the previous pharyngeal flap and without disturbing it. On 11-26-73 the patient stated that her speech had improved so much that speech therapist Bensen saw her only once a year.

**Bottom line:** A paralyzed velum usually requires a wide pharyngeal flap.
CASE 26 had a congenitally short palate with good levator action but incompetent velopharyngeal closure. The patient was treated at age 12 years with a 1.5 cm. wide superiorly based pharyngeal flap attached to the nasal side of the velum. One year postoperatively, speech was normal except for a slight articulation problem of w/r substitution.

CASE 27 was born with a normal-looking palate but a large nasopharynx and velopharyngeal incompetence. The problem was first treated with speech therapy, which only frustrated the patient. At age 13 years a 2 cm. wide pharyngeal flap was inserted into a fishtail incision along the posterior border of the velum and on the upper surface of the uvula; a mucosal turnback flap was used to cover the raw undersurface of the pharyngeal flap. In 1969 cleft palate clinic, the speech pathologist reported normal word production without nasal escape and with good prognosis for speech therapy. A parental report in 1978 stated that the patient can speak as well as he wishes, has a job and is getting married.

CASE 28 was born with a wide cleft of the hard and soft palate. On 3-9-61, at the age of 3½ years, a primary superiorly based pharyngeal flap was used for nasal lining of this large defect. Von Langenbeck incisions allowed release of the mucoperiosteal flaps for oral closure.
Two months after surgery the pharyngeal flap seemed to tether the velum and restrict motion. Division of the flap was contemplated but postponed. In 1963 evaluation of speech in cleft palate clinic revealed a large number of minor sound substitutions, notably f for s and w for l. It was thought that maturational improvement would improve speech without therapy, and, in fact, speech was within normal limits in 1973.

CASE 29 was born with a wide, horseshoe-shaped cleft of the soft and hard palate and a deficiency of tissue. On 4-3-74, at 3 years of age, a primary 1.25 cm. wide superiorly based pharyngeal flap was turned into the entire nasal defect. A V-Y mucoperiosteal flap pushback was carried out. The levator muscle attachments were freed from the edge of the hard palate and sutured into an intact loop, and the oral side was closed with mattress sutures.

Examination of the patient in 1978 revealed a mobile palate with good speech having a slight hypernasal quality but no difficulty with s's.

CASE 30 was born with a wide cleft of all the soft and part of the hard palate. This person received no treatment until, at 28 years of age, he joined the police force. An island flap pushback was planned, but during surgery no discrete bundles were found so the nasal defect was filled with a T-shaped superiorly based pharyngeal flap.
Ten years later the patient’s speech is nearly normal, with minimal nasality causing him no difficulty in his work. He has advanced to the rank of police lieutenant.

CASE 31 had a rather wide cleft of the soft palate, which was short. Treatment included a variation of the T pharyngeal flap. At age 2½ years closure of the cleft and lengthening of the palate was achieved without dissecting mucoperiosteal flaps and endangering maxillary growth. A superiorly based pharyngeal flap was split at its distal end. As the flap was sutured into the nasal side of the cleft, the split ends fitted into the lateral releasing incisions in the nasal mucosa along the posterior edge of the hard palate.

_Bottom line:_ The pharyngeal flap may be _the flap of last resort_, but in severely deficient cases like these it is _a flap of great import._

**CONSERVATIVE PRIMARY APPROACH**

In view of the effect on maxillary growth and development and dental occlusion caused by early denudation of bone, elevation of mucoperiosteal flaps was reduced to a minimum until the age of 4 to 5 years.
CASE 32 was born with a cleft of the soft palate, which was also short. At 9 months of age simple splitting of the cleft edges allowed closure of the velum in three layers. When seen in cleft palate clinic at age 7 years, the patient revealed a short, mobile palate. The speech pathologist noted excellent mobility, normal resonance balance and articulation, absence of hypernasality and mild hoarseness. Speech therapy was advised. Nine months later the patient had a T & A. At cleft palate clinic 10 months postoperatively her speech was borderline normal, indicating that she will probably succeed with speech therapy.

CASE 33 was born with a soft palate cleft which was closed at 1 year of age by a procedure simply splitting the cleft edges and closing in three layers. This included freeing the levator attachments from the edges of the hard palate and, after dissecting them into discrete bundles, suturing them into a retropositioned (1 cm.) intact loop.

Bottom line: This approach handles the muscles in a more sophisticated manner than was used in the previous case and should give minimal distortion with a good chance of normal speech.
CASE 34 was a complete unilateral cleft of the lip, alveolus and palate treated at 1 month of age with P.E. tubes, soft palate closure and a lip adhesion. Six months later rotation-advancement of the lip was completed. At age 2 years a vomer flap and von Langenbeck incisions allowed closure of the hard palate. Evaluation at 4 years revealed that speech was progressing well.

CASE 35 was born with an incomplete unilateral cleft of the lip, an abutting cleft of the alveolus and a cleft of the hard and soft palate. At age 3 months P.E. tubes were inserted, the soft palate edges were split and approximated and the lip was closed by rotation-advancement. At 7 months a vomer flap closed the anterior hard palate. At 3½ years von Langenbeck incisions and freeing of mucoperiosteal flaps allowed division of the levator muscle attachments to the hard palate edge so the muscles could be dissected free, retroposed about a centimeter and sutured into an intact muscle loop. The cleft in the soft and hard palate was then closed in three layers.
The patient returned from Alaska for a checkup in 1978 at age 5 years to reveal good appearance, good speech and good occlusion.

CASE 36 was born with a severe complete cleft of the lip and palate, a deficient lateral lip element and extreme nasal distortion.

On 12-11-74, at 2 months of age, a lip adhesion was accomplished using an I flap to release the lateral alar base and the short vestibular lining. P.E. tubes were inserted after myringotomy, but the soft palate cleft was too wide for closure. The adhesion was effective for six months, until 6-4-75, when rotation-advancement of the lip was achieved along with soft palate closure.
On 7-14-76, at about 21 months, a vomer flap was turned for hard palate nasal lining. Von Langenbeck mucoperiosteal flaps were dissected and the aponeurosis and levator muscle attachments divided from the hard palate edge, allowing the soft palate a slight posterior lengthening when the muscles were sutured.

When the patient was seen in 1978 at the age of 4 years both lip and palate seemed to be functioning well. It was noted that the child would return from Colorado at age 5 years, at which time minor nasal correction would be important for school. If speech is not found adequate at this time because of palatal shortness, an island flap pushback will be considered.

CASE 37 was born with a complete unilateral cleft of the lip and palate. At 6 weeks myringotomy with insertion of P.E. tubes and a lip adhesion procedure were done, but the cleft in the palate was too wide for closure. Rotation-advancement closure of the lip was performed at 9 months and followed with soft palate closure at 18 months. The cleft edges were split and the levator muscles dissected, divided from the anterior attachments to the posterior edge of the bone and sutured into an intact loop, achieving a 1 cm. retropositioning. At 2½ years the cleft in the hard palate had narrowed enough so that nasal mucosa and oral mucoperiosteum could be dissected and closed in two layers without elevation of large flaps.
At age 5 years the child needed orthodontia to spread the maxillary arch. Speech was good despite a cephalometric study showing a slight velopharyngeal gap. He may need an island flap later.

CASE 38 was an Ecuadorian patient born with a complete cleft of the lip and palate. At 3 months P.E. tubes were inserted, the soft palate was closed and a lip adhesion was done. At 6 months the definitive lip closure was achieved by means of the rotation-advancement method. At 2 years of age the hard palate was closed with a vomer flap turned over and tucked under the opposite mucoperiosteal edge of the cleft.

Cleft palate clinic evaluation in 1978 at the age of 4 years found excellent velar and lateral wall motion, good resonance balance and an anterior lisp with every indication of normal speech to come.
CASE 39 was born with a complete unilateral cleft of the lip and palate. At 2 months P.E. tubes were inserted, the posterior soft palate was closed and the lip was approximated with an adhesion. At 6½ months definitive lip closure was achieved with the rotation-advancement method. At 16 months the hard palate was closed with a vomer flap and the remaining soft palate approximated, leaving a fistula at the junction. At 3½ years modified von Langenbeck incisions around the maxillary tuberosities allowed dissection of mucoperiosteal flaps and closure of the remaining opening.

Evaluation in 1977 cleft palate clinic at age 4½ years revealed a short palate functioning well, with closure against the adenoid pad and good progress in speech therapy. The patient’s occlusion was excellent except for a "slight crossbite of upper left deciduous cuspids" with no need for orthodontia. In 1978, again in cleft palate clinic, no changes were noted in dental condition and speech was reported normal.
CASE 40 was born with a left complete unilateral cleft of the lip and palate. At 5 weeks a lip adhesion was performed, mobilizing only the medial element. At age 6 months the soft palate edges were split and sutured, and the lip was closed with rotation-advancement. At age 1 year a vomer flap was turned over for nasal closure of the hard palate cleft. Von Langenbeck incisions allowed total oral closure of mucoperiosteal flaps.

At age 5 years, cleft palate clinic evaluation revealed excellent maxillary development with upper left deciduous cuspids in crossbite but posing no problem. When the patient was last seen in 1978 at age 7 years, a maxillary expander was in position with good arch form. The palate was short but moving well, with intermittent nasal escape. The patient is able to close the sphincter now, but when the adenoids shrink at about 11 years he may need a pushback with an island flap.
CASE 41 was born with a severe unilateral complete cleft of the lip and palate. At 2 weeks of age a lip adhesion molded the maxilla and at 4½ months the lip was closed by the rotation-advancement method. At 1 year of age the soft palate was closed in three layers. At 17 months the hard palate cleft was closed with a turnover vomer flap tucked under the opposite cleft edge. On 2-23-77 basal and lateral view videofluoroscopic evaluations were made during isolated speech sounds and connected speech. Both lateral and basal views indicated very good velopharyngeal motion and somewhat discoordinated velopharyngeal closure. Intensive speech therapy for a period of one year was advised. In 1978, at age 6½ years, clinic evaluation repeated this recommendation. The orthodontist reported excellent buccal occlusion but found the anterior teeth developing in anterior crossbite or tip-to-tip relationship, meriting close observation and eventual orthodontia.

**Bottom line:** This regimen of early lip adhesion, soft palate closure at 1 year and hard palate closure at 17 months with no mucoperiosteal flap elevation and no palate lengthening has resulted in minimal, easily correctable orthodontic problems and borderline speech, often with enough velopharyngeal contact to make normal speech possible with the aid of intensive speech therapy.

CASE 42 was born with a severe complete bilateral cleft of the lip and palate and a projecting premaxilla. At 7 months of age the soft palate was
closed by splitting the cleft edges and suturing in three layers. At the same
time the bilateral cleft lip was closed with union of the muscles across the
cleft and banking of the forked flap. At 18 months of age bilateral vomer
flaps were turned over for nasal closure and bilateral von Langenbeck
incisions facilitated the dissection of bipedicle mucoperiosteal flaps, which
made closure on the oral side possible.

At 5½ years the banked forked flaps were used to lengthen the columella
and free the nasal tip. The father reports from Europe that at age 6 years the
child is doing well in school and that "her speech is perfect." She is now
starting orthodontic treatment.

CASE 43 was born with a complete unilateral cleft of the lip and palate.
At 7 weeks a lip adhesion was achieved, but the palate proved too deficient
for closure. At 8 months the edges of the soft palate cleft were split and
sutured and the lip was closed by the rotation-advancement method. The
maxillary arch, molded by the lip adhesion and definitive closure, facilitated
hard palate closure employing a vomer flap and unilateral von Langenbeck
incision at 16 months. Excellent arch form is present at 18 months.
**Bottom line:** This conservative approach achieves early coordina-
tion of soft palate and velum, as well as hard palate closure
before 18 months without wide mucoperiosteal dissections.

In the following case there was such a wide cleft that a
conservative velar adhesion had been used primarily and the
residual gap threatened to require the aid of a pharyngeal flap.

CASE 44, first operated on in central Florida, had had a wide cleft of the
soft and hard palate partially closed with a velar adhesion at age 2 years. At
age 5 years the residual defect was still huge. Freeing of the nasal mucosa
from the bony edge of the cleft allowed closure of this layer except for a 0.5
cm. hole at the anterior end. The levator muscles were dissected from the
posterior bony edge, retropositioned and sutured into an intact muscle loop.
Then a variation was used to achieve oral closure without tension, especially
at the anterior end. A von Langenbeck incision on the right and a V-Y type
of flap on the left facilitated shifting of a mucoperiosteal flap over the
anterior hole with sound closure, bypassing the need for a pharyngeal flap.
SECONDARY SURGERY

SECONDARY ISLAND FLAP PUSHBACK
It was estimated in the conservative approach that the arch collapse and malocclusion would be greatly reduced and easily amended with orthodontia, but that only 70 percent of patients would get normal speech. Thirty percent (or more) would require an island flap pushback at 5 years or older. This regimen thus seemed acceptable.

CASE 45 was born with a cleft palate, closed in Boston at 14 months of age. At age 5 years the patient revealed nasal escape during speech.

On 8-14-62, at 5 years, an anterior bipedicle island flap was used in a pushback of the palate for a 1.5 cm. lengthening. The palate was well healed two months postoperatively. Final evaluation in 1976 at 19 years revealed an active, mobile palate producing normal speech and good occlusion.

CASE 46 was an Ecuadorian girl with a failed cleft palate closure, who revealed nasal escape when seen at age 8 years. An island flap was taken from the anterior portion of each side of the mucoperiosteum, sutured to the other and inserted into the nasal lining defect after a pushback of 1.7 cm. A 1.25 cm. wide superiorly based pharyngeal flap was let into the bifid uvula. A suction test was positive.
CASE 47 was born with a complete unilateral cleft of the lip and palate, treated in infancy in Tennessee. The palate was short, allowing nasal escape in speech. In 1968, at age 12 years, a secondary V-Y pushback was performed using 2 cm. wide left island flap to fill the nasal lining defect after release of the soft palate from the hard palate edge. Within two months after the pushback surgery the patient was speaking normally. In 1977, at age 21 years, the transverse palatal scar had caused some palatal migration of the bicuspsids. Cephaloradiographs by Berkowitz soon after the island flap pushback surgery and nine years later show good elevation and contact with the posterior pharyngeal wall, consistent with the action that can be seen directly during speech.
CASE 48 was born with a cleft of the soft palate which was closed at 4 months of age following splitting of the cleft edges and suturing in three layers. Cleft palate clinic evaluation in 1977 found good velopharyngeal activity with speech attempts characterized by mild to moderate hypernasality. In April 1978, nasopharyngoscopy by Pullen and the Dicksons found good velar and lateral wall motion with a rectangular velopharyngeal gap approximately 5 mm. in anteroposterior direction and 2 mm. in lateral direction. The lateral walls were observed to nearly meet with effort on the production of /silks/. Total closure was not achieved during any speech samples elicited.

Assessment: Velopharyngeal activity is inadequate for balanced resonance, presenting a persistent $5 \times 2$ mm. aperture during the production of fricative-vowel-consonant-fricative speech samples which prognosticates poorly for sustained and adequate closure during connected speech.

On 5-19-78, at age 6½ years, a palatal pushback was done with an anterior 1.5 cm. bipedicle island flap inserted into the nasal lining defect. A small (Stellmich) superiorly based pharyngeal flap was inserted into the nasal side of the uvula for an adhesion. Postoperative suction test was positive.
Bottom line: Early simple closure was not quite enough, so a pushback with an island enabled the velum to function in closure. A small pharyngeal flap adhesion offered a “hand-up” to the velum, reducing the lift excursion for better efficiency at little cost.

CASE 49 was born with a left complete cleft of the lip and palate treated at 1 month with a lip adhesion. At age 4 months rotation-advancement closed the lip, using 1 flap for nasal closure over turnback mucoperiosteal flaps from the alveolar cleft edges for nasal floor reconstruction. At 10 months the edges of the soft palate were split and sutured, and at 2 years a vomer flap closed the hard palate defect.

At age 5 years evaluation revealed a short, mobile velum and active lateral walls but hypernasal speech. The upper left cuspid was going into crossbite. In 1977, at age 6 years, a V-Y pushback leaving an anterior V over the previous cleft area and placing a right island flap into the nasal defect achieved a 1.5 cm. lengthening.
In 1978 the patient had a slight upper left cuspid crossbite, to be treated by Berkowitz. Both isolated and connected speech were normal.

CASE 50 was born with a complete unilateral cleft of the lip and palate. A lip adhesion at age 1 month separated and was resutured five days later. At 6 months rotation-advancement closed the lip. At 16 months the soft palate was closed, and at 2 years a vomer flap was turned for hard palate closure. At age 4 years, because of nasal escape in speech, a V-Y pushback was performed, an island flap being inserted into the nasal lining defect. After one year of speech therapy the patient’s speech was good enough to discontinue therapy. Orthodontia was in progress at age 7 years to give moderate maxillary arch expansion.
CASE 51 was born with an oblique unilateral incomplete cleft of the lip and complete cleft of the alveolus, hard palate and soft palate. On 5-8-73, at 5 months of age, lip closure by rotation-advancement and soft palate closure by splitting and suturing the edges were accomplished.

On 5-8-74 at nearly 18 months, a vomer flap was turned over and tucked under the opposite mucoperiosteal edge of the cleft to close the hard palate in one layer. A small fistula remained at the junction of the hard and soft palates.

In 1975 the patient revealed good occlusion except for a crossbite of the left cuspid. Speech was unintelligible. In 1976 cephalometric evaluation by Berkowitz revealed velopharyngeal closure and good motion but a small soft palate and a thin, contracting adenoidal mass. The speech pathologist recorded nasal escape.

On 6-14-77, at about 4½ years, mucoperiosteal flaps were elevated on each side of the cleft and an island flap was developed on the right. The neurovascular bundle was freed on the left and the nasal lining divided, as were the levator muscle attachments from the posterior edge of the hard palate, allowing the soft palate to move posteriorly 1.5 cm. The island flap filled this defect, and the mucoperiosteal flaps were advanced posteriorly in V-Y manner to close the fistula and cover the island flap. The suction test was positive at the end of the operation.

When seen in 1978 the patient had perfectly normal speech without nasal emission, and only the left deciduous cuspid needed to be brought into normal labial position.
The reduction in scar contracture of the maxilla and the amount of required orthodontia seems to justify a delay in normal speech development in some cases. At 5 years an island flap can lengthen the palate and obtain normal speech with maxillary impunity.

**A TERTIARY PPF STEP**

CASE 52 was born with a cleft of the palate which was evidently treated at 8 months and again at 18 months by surgery elsewhere. When seen at age 6 years the child had a short palate and marked nasal emission during speech. In 1969, at age 6, a V-Y pushback was performed with a 1.5 cm. right island flap inserted into the nasal lining defect after release from the hard palate. Improvement in speech was noted, but nasal emission continued. At 8 years of age, therefore, a small anterior residual fistula was closed in two layers and a 1.4 cm. wide superiorly based pharyngeal flap inserted on the superior aspect of the soft palate and into a split along the posterior edge of the velum. In 1977 cleft palate clinic at the age of 14 years, the speech pathologist reported, “Speech is normal.”

CASE 53 was born with Pierre Robin syndrome, including a cleft of the soft palate. Glossoptosis with respiratory distress was treated effectively with a Kirschner wire through both mandible angles and the tongue. At 14 months of age a V-Y pushback was achieved, inserting a right island flap into the nasal lining defect.

In April 1978, at age 10 years, nasopharyngoscopy by Pullen and the Dicksons revealed lateral wall motion to be minimal and velar motion good, with occasional touch contact. Connected speech samples showed near but not complete closure, with an opening narrow in the anteroposterior direction and broad in lateral dimension, as shown in the diagram, producing hypernasality in both isolated and connected speech samples.

On 6-28-78 a 2 cm. wide superiorly based pharyngeal flap was inserted on the superior surface and into fishmouth incisions of the lateral posterior
edges of the velum. Velar mucosal turnback flaps were used to cover the raw underbelly of the pharyngeal flap. Speech improved postoperatively.

Note the normal development of the mandible and minimal need for orthodontia of the maxillary teeth.

CASE 54 had a submucous cleft palate which developed speech difficulty after a T & A. At 5 years of age a bipedicile island flap pushback achieved a 1.25 cm. lengthening but with negative suction test results. Cephalometric evaluation revealed a 0.75 inch gap in velopharyngeal closure and hypernasal speech.

At age 7 years a 1.5 cm. wide pharyngeal flap based superiorly was attached to the velum with reduction in nasal escape except in connected speech.

At 9 years lateral recess flaps were transposed transversely across the posterior pharyngeal wall (Hynes). At the same time a V-Y upward advancement of the pharyngeal flap base was done (Hirshowitz). The suction test was positive, and near normal speech followed.

In 1979, David and Wilma Dickson gave a possible explanation for the prolonged difficulty of this case:

Our best guess is a supranuclear lesion somewhere within the extrapyramidal system, perhaps in the corticobulbar pathways, perhaps involving the cerebellar system. Our reasons for this are: (1) no observable peripheral pathology; (2) no paresis or other signs of pyramidal tract involvement; (3) normal function when operating "cortically"; and (4) reduced or absent velopharyngeal function during rapid or spontaneous speech and whenever his attention is distracted. In a sense this behavior is exactly the opposite of that found in dyspraxia or apraxia. Other possible explanations include a very subtle lower motor neuron pathology or involvement of the proprioceptive feedback system. The patient is being referred to pediatric neurology for further testing of both motor and sensory integrity.

**Bottom line:** Some cases need help from both ends, so never give up.
SECONDARY PPF

Some secondary cases with only a pharyngeal flap are presented.

CASE 55 was born with a complete unilateral cleft of the lip and palate, receiving primary surgery in Virginia. Secondary deformities included a constricted nostril, a lip without landmarks, a retropositioned maxilla and a short palate with nasal escape during speech. At age 13 years the mobile but short and scarred palate was benefited by a 1.5 cm. wide superiorly based pharyngeal flap which was let into the nasal side and posterior edges of the velum. The raw underbelly of the flap was covered by a mucosal turnback flap from the upper surface of the velum and uvula. Subsequently iliac bone grafts to the maxilla, orthodontia, a midline Abbe flap and cleft lip rhinoplasty achieved a pleasing result. At age 17 years the patient had good occlusion, good speech and a good appearance.

CASE 56 was a 9-year-old boy who had received speech therapy since kindergarten. He revealed a submucous cleft palate with a bifid uvula, a midline cleft in the muscles and hypernasal voice quality during connected and isolated speech. On 1-4-78 Pullen and the Dicksons, using a Machida nasendoscope, visualized the velopharyngeal aperture, noting good velar and lateral wall motion during speech. Yet closure was incomplete at the midline for all samples of speech tested, particularly for high vowels and fricatives. There was a marked curvilinear depression at the velar midline, precisely where the uvular bulge is expected to produce closure. This was their assessment:
The midline velar depression results in incomplete velopharyngeal closure at midline; air escape is observable through this "hole" during isolated and connected speech samples, but not during swallowing. Since velar and lateral pharyngeal wall motion were clearly adequate except for midline closure, it is recommended that surgery be concentrated on this specific area—a narrow pharyngeal flap should suffice.

On 1-23-78 the edges of the bifid uvula were pared and the soft palate was divided in the midline. Levator muscle fibers were freed from the hard palate edge, dissected into bundles and sutured into an intact loop. A 1.3 cm. wide superiorly based pharyngeal flap was let into the longitudinal muscle defect on the nasal side to bolster the area of weakness. The pharyngeal wall donor area was closed with 3-0 Vicryl mattress sutures.

Five months later, evaluation in cleft palate clinic by the Dicksons revealed competent closure with good speech in isolated words, including s, but speech therapy is required to complete the correction in connected speech.

**Bottom line:** This is the kind of coordination of diagnostic and surgical teams that will eventually solve most palatal problems.

CASE 57 was born with a severe unilateral complete cleft of the lip and palate and mucous pits of the lower lip. At 3 months rotation-advancement closed the lip, and at 8 months alveolar mucoperiosteum was elevated so that split rib grafts could be inserted into and across the cleft. At 10 months the soft palate was closed, reducing the hard palate cleft so that at 2 years it could be closed with a vomer flap. The mucous pits in the lower lip were excised at this time. Speech was reasonable until the patient had a T & A which resulted in nasal escape during conversation. At 7 years a wide superiorly based pharyngeal flap was attached to the upper surface of the velum and along the posterior edge, facilitating normal speech. Orthodontic correction by Silver was being maintained with a retainer at age 15 years. A corrective rhinoplasty at age 16 years completed reconstruction.
CASE 58 was born with isolated cleft palate, treated elsewhere with a Wardill pushback at age 2 years and another pushback with a pharyngeal flap at 8 years. When first seen in our cleft palate clinic at age 12 years, this girl still had incompetent velopharyngeal closure with nasal escape.

On 4-15-70 there was no evidence of the first pharyngeal flap. A second, 2 x 3 cm. superiorly based pharyngeal flap was attached nasally to a split in the soft palate.

Speech pathologists’ 1974 evaluation at age 16 years reported essentially normal speech.

**Bottom line:** The velar-pharyngeal synechia may not be physiological but it often works.

Here is a modification in technique for attaching a wide superiorly based pharyngeal flap to the superior surface of the velum and laterally along the posterior pillars. It does not require splitting the entire velum, and there is a cosmetic advantage as the pharyngeal flap is hidden by the intact uvula.

**Both Island and Pharyngeal Flap**

Finally, when the tissues are deficient or the velopharyngeal gap is large, the island flap pushback and a complementary posterior
pharyngeal flap can be used simultaneously to lengthen the palate (belt) and reduce the velopharyngeal gap (suspenders). An additional advantage of the pharyngeal flap synchia is its "hitching post" function, which keeps the velum up and close to the posterior pharyngeal wall at all times and thus reduces the length of excursion and the amount of velar lift required by the levator muscle for more efficient sphincter control.

CASE 59 had an incomplete cleft of the soft palate treated in infancy. After adenoidectomy at age 5 years, the patient began to have difficulty with speech and was treated with a pharyngeal flap. The attachment, however, became disrupted and left a large central defect in the velum which was responsible for nasal emission. Contraction of the levator muscles presented limited lift of the velum because of their attachments to the hard palate edge.

At age 29 years the mucoperiosteum was elevated from the hard palate through a horseshoe-shaped incision. Both neurovascular bundles were dissected free to create an anterior bipedicile island flap 2 cm. wide. The soft palate was divided from the edge of the hard palate with a pushback of 2.1 cm., and the island was sutured into this defect. The levator muscles were dissected into bundles and sutured to each other to form an intact loop. A 1.4 cm. superiorly based pharyngeal flap was inserted into the nasal side of the split velum and uvula. Healing was uneventful.

Sixteen months postoperatively, referring surgeon Gary Russolillo of Hartford, Connecticut, wrote:

*Your patient has progressed marvelously and during an evaluation at the University of Connecticut Medical Center Speech Therapy Department was informed that she had such excellent speech that further therapy would be of no benefit to her. She indeed has a marked change in speech, noted both in personal contact and over the phone. Prior to surgery it was difficult to understand her on the phone and in person.*

CASE 60 was born with a soft palate cleft, treated elsewhere at age 12 years with a pushback using a horseshoe-shaped incision. At age 19 years the patient revealed a short, mobile palate with a large velopharyngeal gap. The speech pathologist in cleft palate clinic reported a short palate with extreme
hypernasality but excellent articulation. On 1-16-78 a horseshoe-shaped incision at the line of the old scar allowed elevation of a mucoperiosteal flap. Bilateral dissection of neurovascular bundles produced a bipedicle flap of anterior mucoperiosteum which was turned over to fill the nasal lining defect after release of the soft palate from the hard palate edge. The old scar of the soft palate was opened, and a 1.4 cm. wide superiorly based pharyngeal flap was inserted on the nasal side.

Six months after surgery there was a long, mobile palate which already made for much improvement in speech and good production of s's. Speech therapy is in progress.

CASE 61 had an isolated cleft of the palate which was closed elsewhere with the von Langenbeck method. The result at 17 years of age was a mobile but short velum with marked nasal escape.

On 2-7-78, by means of a Dorrance incision, an anterior bipedicle island flap was used to fill the release in nasal lining. A small superiorly based pharyngeal flap was attached to the split in the uvula. The suction test was positive.
On 6-16-78 the palate appeared long and mobile. Nasal escape in speech was still present but diminishing. The patient is to have speech therapy.

CASE 62 was born with a complete unilateral cleft of the lip and palate. The lip was closed at 6 months and the palate at 18 months in Boston. At age 10 years this boy revealed buccal crossbite which was under orthodontic expansion. His palate was mobile but short and scarred, producing hoarse, hypernasal speech.

On 3-24-78 at age 12 years a 1 × 2 cm. superiorly based pharyngeal flap was sutured into a split on the superior surface of the distal velum and uvula as an adhesion. Bilateral mucoperiosteal flaps were elevated on either side of the previous cleft and the neurovascular pedicles dissected. A 1.25 cm. island flap was cut free on the right and turned over into the nasal lining defect after release of the soft from the hard palate. The remaining mucoperiosteal flaps were advanced posteriorly in V-Y fashion and sutured over the island flap. Uneventful healing was followed by speech therapy and normal speech.

Bottom line: When the palate is mobile and short, with an anteroposterior gap of no more than 1 cm., a pushback with an island flap is considered more physiological. If the gap is greater than 1 cm., then a pharyngeal flap can be used in conjunction with the island flap. If the gap is extremely large, a wide pharyngeal flap may be necessary. Remember that too much reduction in nasal air flow causes destructive changes of the nasal mucosa, reduction in lung function and hyponasal speech.

Of course, there are other ways of achieving good results. This chapter is but a study of the successes and failures of one surgeon over 21 years.