Converse: Chapter 45

Cleft Palate

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An infant born with a cleft palate inherits many handicaps. Unable to develop intraoral negative pressure, he cannot suckle and hence cannot feed in a normal fashion. Furthermore, on swallowing, he expels his feedings through the nose. The open cleft, because of failure to warm and moisten the inspired air, produces a continuing sore throat. The middle ears become secondarily involved with serous otitis media; mastoiditis can be a secondary complication. If the cleft involves the alveolus and hard palate, the cleft segments of the jaw may collapse, producing malocclusion. Speech is often unintelligible, occasioning cruel mimicry and taunts from peers. It is no surprise that these handicaps may produce serious psychological sequelae.

The distraught parents (and often the grandparents) are usually overcome with fear, guilt, and dark forebodings. What has caused their child to be so cruelly deformed? What have they done to deserve such misadventure? Will other children they might have be similarly affected? These and similar doubts obsess them.

Counseling

It is mandatory that a person knowledgeable about facial clefts see the parents as early as possible. The guilty feeling that something the parents have done in the past is to blame for the malformation must first be dispelled. They must be told that an educated guess as to the etiology can be made in only 20 per cent of cases. Indeed, in the vast majority of cases, the physician simply cannot assign a cause to a specific abnormality despite recent developments in the field of teratology.

As background material, it is known that a facial cleft occurs in about one in 700 births in the US and less frequently in the black than in the white population (see Chapter 38). Furthermore, if a sibling has a cleft palate, the likelihood of another child being born with a cleft is 12 per cent. If one parent has a cleft, the likelihood is about 7 per cent. If a parent and one child are so afflicted, the expectancy that another child will be so malformed rises to 17 per cent. The incidence is somewhat less if one is dealing with cleft lip and cleft palate.

Environmental factors in utero may play an etiologic role either alone or multifactorially in combination with an hereditary diathesis. Such factors must be operational during the seventh to twelfth weeks of gestation when the palatal shelves are elevating to the horizontal position and finally fusing together (see Chapter 39). Several environmental factors which seem to have etiologic potential in the development of the cleft palate are hypoxia, rubella, and diabetes. In addition, a period of relative infertility, abortion, or stillbirth may be followed by the birth of an anomalous infant.

Cleft lip alone (cleft of the primary palate) occurs more frequently in the male, cleft palate alone (cleft of the secondary palate) more frequently in the female. The combined anomaly of cleft lip and palate is equally prevalent in males and females. When the double
An anomaly occurs, it is present on the left side three times more frequently than on the right. An exceptionally broad head with anteroposterior shortening (oxycephaly) is associated with palatal clefts and is partially responsible for their occurrence (see also Chapter 46). The palatal gap during organogenesis is too wide for the normal palatal shelves to bridge. Similarly, an exceptionally large tongue may block the palatal shelves in their migration toward the midline. A small lower jaw (micrognathia) can produce a small oral cavity, and in this case a normal sized tongue will block the fusion of the palatal shelves. This mechanism accounts for the increased incidence of cleft palate in the Pierre Robin anomalad (see Chapter 50).

The person counseling the parents should point out the various functions that are affected by the anomaly: chewing, swallowing, hearing, and speaking. He should indicate that each will be dealt with at the optimum period of the patient's growth. At the primary interviews, the physician should not overwhelm the already stressed parents. However, at each step along the route of rehabilitation and at subsequent follow-up visits, more of the future developmental and reconstructive time table should be discussed.

Definition

A cleft palate is a fissure in the roof of the oral cavity. The classic terms were uranoschisis (ouranos, Greek for sky or roof of the mouth + schisis, fissure), alluding to a cleft of the hard palate, and staphylorschisis (staphyle, Greek for uvula), alluding to a cleft of the soft palate or velum (Latin for a structure resembling a veil). Embryologically, the palate - or preferably "secondary palate" (see Chapter 39) - is the roof of the mouth situated posterior to the incisive foramen, from the canine teeth, posteriorly, including the hard and soft palates, but excluding the premaxilla with its four incisor teeth.

A cleft of the secondary palate may be complete, i.e. extending from the uvula to the incisive foramen, or it may be incomplete to various degrees - bifid uvula, cleft of the velum, or a submucous cleft, in which the palate may appear normal despite a diastasis of musculature in the velum and an absence of palatine bones in the hard palate. It may also be complete with an associated alveolar cleft (cleft of the primary and secondary palates), producing collapse of the maxillary segments towards the midline.

History

In surgical annals, no more heroic chapter has been written than that of the repair of cleft palate. The earliest palatoplasties were performed without anaesthesia, without control of the airway, and, in the face of alarming hemorrhage, without the benefit of blood transfusion. It is amazing that surgeons persevered and that patients acquiesced when hope of success was so minimal. However, by the time anesthesia was first used for repair of the cleft palate by Colles in 1867, all of the basic techniques for closure of the cleft had been described. Closure of the cleft of the velum (staphylorrhaphy) had been successfully achieved by von Graefe of Germany in 1817 and by Roux of France in 1819. In addition, after the advent of anesthesia, Jonathan Mason Warren (1867) had reported success in the closure of 88 of 100 cases of soft palate clefts. Closure of the hard palate (uranoplasty) had been attempted, and the operation was enhanced in 1826 by the contribution of Dieffenbach, who employed bilateral relaxing incisions within the alveolus and around the maxillary tuberosity.
so as to convert the soft tissues on either side of the cleft into bipedicle flaps. Von Langenbeck (1861) realized that nothing less than the bilamellar cover of the hard palate - the mucosa and periosteum or mucoperiosteum - would remain closed without wound disruption. Following the introduction of anesthesia to the field of cleft palate surgery, pharyngoplasty and palatal lengthening procedures were added to the armamentarium of surgeons who performed these operations. The introduction of endotracheal anesthesia by Magill in the early 1920's added additional safety to palatal surgery.

In the longitudinal follow-up of cleft palate repairs, it was noted that the central third of the face failed to develop as fully as the rest of the physiognomy, a finding presumably secondary to surgical trauma from wide elevation of the mucoperiosteum. Since the maxillary bones are membranous in origin, they develop by successive deposition of lamellae beneath the periosteum until the age of 5. Thus, by elevating the mucoperiosteal bipedicle flaps, the surgeon can deprive the maxilla of the blood supply necessary for this type of bony growth. The result is that the central face is relatively flat, producing a dish-face appearance (see Chapter 42).

To obviate the problem of facial underdevelopment, Dieffenbach (1845) extended his relaxing mucoperiosteal incisions through bone. By eliminating the undermining between the relaxing incisions and the cleft, he hoped to obtain both cleft closure and normal facial skeletal development. The "bone-flap operation", usually performed in two stages, has survived to modern times, having been championed in the USA by Warren B. Davis (1940); G. V. I. Brown, Hyslop and Wynn, and Peer have also advocated the technique.

Because of a similar reasoning, Schweckendiek in Germany (1962), as well as Slaughter and Pruzansky (1954) in the USA, avoided early operation for uranoschisis and recommended closure of only the velum between the ages of 12 and 24 months, so that speech would develop unabatedly while the growth potential of the maxilla was still preserved. They obturated the bony cleft, closing it when union of the velum brought the cleft margins into close approximation so that the remaining cleft could be closed without the need for wide surgical undermining. Alternatively, the bony cleft could be closed at the age of 5, by which time it is believed, bony growth of the central face shifts its locus from beneath the periosteum to suture lines between facial bones.

In an attempt to improve the speech results following palatal repair, various retrodisplacement procedures were advocated by Ganzer (1917), Halle (1925), Ernst (1925), Veau and Ruppe (1922), Moorehead (1928), Kilner (1927), Wardill (1928), Peet (1961), and Reidy (1962). This procedure produces an "M" incision anteriorly, elevates virtually all of the mucoperiosteum of the hard palate, releases the aponeurotic attachment from the posterior margin of the hard palate, and "pushes back" the soft tissues of the hard and soft palates by suturing the "M" as an inverted "U" - a variation of the "V-Y" advancement principle.

Dorrance (1925) developed a variation in retrodisplacement. He made a relaxing incision within the alveolar ridge from one maxillary tuberosity to the contralateral one, thereby producing a transverse flap (posteriorly based) which was mobilized toward the posterior pharyngeal wall.
Several historic concepts of uranoplasty must be mentioned, only to be condemned. One was the attempt to close the cleft of the hard palate by turning over the mucoperiosteum of one palatal shelf as a flap, based upon the cleft margin - turning it over as one turns the page of a book - then tucking it under the periosteum of the opposite palatal shelf. Not only was the flap poorly designed from the point of view of blood supply, but also mucoperiosteum was lost to both palatal shelves.

Another form of uranoplasty to be deprecated was the "forced compression" procedure by Brophy (1894), whereby the palatal shelves were brought into apposition by the insertion of wires placed through the two maxillary processes and twisted over lead plates on the buccal side of the alveolar ridges. When the cleft margins came into apposition as the result of twisting the wires, Brophy sutured the mucoperiosteum to close the cleft between the premaxilla and palatal shelf. Brophy instituted this procedure at 10 days to 3 weeks of age. Referring to "forced compression" in 1905, Berry prophetically stated: "... violent operations upon the maxillary bones ... I think, may be left to those who ... overcome difficulties by force (rather) than by craft ..."

The final historical development which has improved results in the management of cleft patients has been the advent of the multidisciplinary cleft palate team or clinic. Ideally, in such a clinic the plastic surgeon, pediatrician, dentist, oral surgeon, orthodontist, prosthodontist, social worker, speech pathologist, anesthesiologist, otolaryngologist, audiologist, psychologist, psychiatrist, and nurse combine skills in coordinating an individualized treatment plan designed to achieve the best results at the most propitious period during the child's growth and development. In this way the shortest time is expended while minimal trauma is created for the child and parents.

The Goals of Surgery

Construction of an Air- and Water-tight Velopharyngeal Valve. The construction of an air- and water-tight velopharyngeal valve is an essential requisite for normal speech. The non-cleft velopharyngeal valve is a complex mechanism which possesses two essential parts: the velum or soft palate and the pharynx. The velum produces a flaplike closure of the oral pharynx and receives additional assistance from the pharynx, which moves forward and medially to clutch the flap.

The velar movement in speech is supplied by the paired levator muscles, which arise from the inferior pyramids of the temporal bones. They then pass anteriorly, inferiorly, and medially to produce the muscular sling, which, when contracted in speech (through nerve impulses mediated via the vagus nerve), produces the levator papilla, an elevation upon the nasal side of the velum (see Chapter 41). In sagittal view, this papilla forms a right angle genu, which is drawn upward and backward to approximate the posterior pharynx at the height of the atlas.

The midline pharynx then moves forward, producing a bulge which seals off the nasal pharynx so that air will not escape nasally in speech and fluid will not escape during deglutition. The "pharyngeal clutch" is produced by the superior constrictor muscle, which arises from the pharyngeal tubercle of the occipital bone, the medial face of the pterygoid
process, the pterygoid hamulus, the pterygomandibular raphé, the mandible, and the tongue. The superior constrictor muscle is innervated by the vagus nerve (see Chapter 41).

Another paired muscle is involved, presumably, in deglutition and in opening the eustachian tubes: the tensor of soft palate. It arises from the scaphoid fossa and the spinous angle of the sphenoid bone, as well as from the cartilaginous and membranous portions of the eustachian tube. It runs forward, medially, and downward to the pterygoid hamulus and turns medially around the hamulus to join its counterpart from the opposite side; it also attaches to the palatine bones. The tensor veli tenses the velum, presumably in preparation for swallowing, and opens the eustachian tubes. It is innervated by the mandibular branch of the trigeminal nerve. In its downward and medial course from its origin in the skull, the tensor is situated lateral to the levator muscle (see Chapter 41).

The timing of the surgery is almost as important a factor in obtaining normal speech as is the surgical procedure used. Surgical closure of the velum before the onset of speech produces normal speech in a higher percentage of cases than if the closure is accomplished after speech has begun. Although connected speech begins at about 18 months in the noncleft infant, there is a delay in language development in the cleft palate group. Nevertheless, this group will catch up with the noncleft population. The objective should be to have the infant start speaking with a normal speech mechanism. Since speech is a habit, beginning to speak with faulty anatomy almost assures the patient of faulty speech. Even though the anatomy is later corrected, the reversal of bad speech habits remains a herculean job for the patient and speech pathologist.

In attempting to construct a well-functioning air valve for speech, three procedures which enjoy contemporary popularity have been devised: (1) closure of the palate with reconstruction of the levator muscle sling; (2) V-Y retroposition of the palate; and (3) simultaneous closure of the palate and primary pharyngeal flap.

**Preservation of Hearing.** In general, hearing is best preserved with early closure of the velum; thus, the eustachian tubes and the middle ears are protected from dryness and cold, which profoundly affect the physiology of this region. A majority of cleft palate patients will develop serous otitis. The latter is thought to result from malfunction of the opening-closing mechanism of the eustachian tubes due to improper function of the tensor muscles. With improper middle ear aeration, there is absorption of gases (O₂, N₂, CO₂), producing an unequal pressure on either side of the tympanic membrane and causing its collapse. Adherence of the tympanum to the middle ear ossicles may occur, or a cholesteatoma (squamous epithelial granuloma) may form. A serous transudate can collect in the middle ear. Treatment consists of continuous aeration by means of transtympanic plastic tubes.

**Preservation of Facial Growth.** The membranous bones of the face grow by subperiosteal lamellar deposition of bone until the age of about 5 years, when growth shifts to the suture lines of the skull. Consequently, the more periosteal removal or scarring secondary to palate repair, the greater will be the maxillary deformity. The solution to this problem has been extremely gentle and limited periosteal elevation, if it is needed to achieve closure of the palatal cleft. Alternatives include avoidance of periosteal elevation, as in the bone-flap operation, or obturation of the hard palate cleft with closure deferred until growth shifts to the suture lines.
**Functional Occlusion and Esthetic Dentition.** Occlusion has been achieved in the past by conventional orthodontic therapy performed on the permanent dentition. In the last 15 years, early "maxillary orthopedics" have shifted the maxillary segments when the alveolus has been cleft (complete cleft of the primary and secondary palates), and the aligned segments have been unified by a bone graft (see Chapter 48). The feeling was that, in so doing, lateral medial collapse and orthodontic therapy would be avoided. This hope has not been realized in spite of hard and often exquisite work by its proponents (see Chapter 48). Bone grafting has not prevented collapse. It may be due to the truism that scars perpendicular to the cleft augment collapse, while those running in a parallel direction do not. Such perpendicular scars were the product of mucoperiosteal and mucosal flaps raised to provide mucosal coverage of the grafts. A basic surgical truism was violated: one should obtain surgical goals with as few operations and procedures as possible.

**Surgery**

**General Health.** As a requisite for surgery, the infant should be free of acute infection and should have a haemoglobin of at least 10 grams. However, nasopharyngeal mucous or mucopurulence, which coexists with an open palatal cleft, is no contraindication to surgery.

**Anesthesia.** General endotracheal anesthesia is utilized with atropine as the only premedication. The patient is allowed no solid food for 12 hours and no liquids for four hours prior to surgery. For a 20-pound infant, 0.3 mg of atropine is administered 45 minutes prior to induction. The infant is positioned supine with the head in extension upon a water-circulating thermal blanket for maintenance of body temperature. The rectal temperature is monitored, a stethoscope is taped to the chest wall, and a 2-inch blood pressure cuff is placed on one arm.

Anesthesia induction is by open-drop, semi-closed system or by the Reese modification of the Ayre technique. The choice of agents used is a personal one, depending upon the experience of the anesthesiologist. When the proper level of anesthesia has been reached, the infant is intubated orally without relaxant. Sterile plastic endotracheal tubes (No. 16, 18, or 20) are used without cuffs. A nonrebreathing technique with an Ayre T-tube is utilized. The endotracheal tube is held in situ on top of the tongue by the blade of a Dott or Dingman mouth gag. The desired level of anesthesia is usually maintained with nitrous oxide and oxygen in a 50 per cent concentration with halothane (0.5 to 1.0 per cent). The operation should not last longer than one hour, since the risk of laryngeal edema increases.

An infusion of 5 per cent dextrose and water is started by microdrip, the infusion bottle containing only one-third of the infant's fluid requirement for 24 hours.

When the operation is completed, the pharynx and larynx are carefully aspirated. Anesthesia is discontinued, and the infant is allowed to breathe pure oxygen for several minutes. When respirations are regular, the endotracheal tube is removed very gently at the end of inspiration. An oropharyngeal airway is carefully inserted so as not to damage the surgical closure, and the patient is positioned prone with the head turned on one side. The patient should not be removed from the operating room until respiratory excursions are adequate and the pharyngeal reflexes have returned.
A tracheotomy set should accompany the infant to the recovery room. If signs of laryngeal edema appear, treatment with high-humidity oxygen, antihistamines, and intravenous steroids is instituted.

Formation of the Levator Sling

Union of the levator muscles into an end-to-end relationship so as to produce a levator sling is one step in an operation which also incorporates a relaxing incision, either of the V-Y retroposition or von Langenbeck type.

Veau (1931) stated that suture of the cleft muscles of the velum was indispensable. The anatomy of the cleft muscles, when repaired by reorientation (from sagittal to coronal direction) and end-to-end union, resembles two "U's" interlocking to form an "X". The first "U" forms the levator sling, the second the palatopharyngeal sling. The levator and palatopharyngeus muscle bundles, in the cleft velum, attach to the palatine bones at the posterior aspect of the cleft hard palate. Freed as a sheet, the muscle is developed by dissection into a plump bundle which is joined to its counterpart on the opposite side. Kriens (1970) has recommended intravelar veloplasty with end-to-end midline union of the cleft musculature to reconstruct the levator sling.

The palatopharyngeal sling courses from the levator papilla in a lateral direction and posteriorly; it encircles the pharynx and, when contracting, produces Passavant's ridge.

The formation of the levator sling is usually performed with a V-Y retroposition procedure, to be discussed in the following section.

V-Y Retroposition ("Push-Back") Procedure

The operation for a complete cleft of the secondary palate (postalveolar cleft) differs from that for a complete cleft of the primary and secondary palates (prealveolar cleft). The two techniques narrated are basically those of the Oxford plastic surgeons (Kilner, Peet, Osborne, Reidy, Patterson, Calnan). Their techniques are illustrated in a paper by Peet (1961).

A small amount of anesthetic solution (0.5 per cent) with epinephrine is injected into the mucoperiosteum to reduce blood loss. The cleft margins are incised; an S-shaped incision is made starting along the pterygomandibular raphe, curving it around the maxillary tuberosity posteriorly, then bringing it forward and medial to the alveolus. When the scissor blades are spread, the wound margins are distracted down to the hamulus, which is fractured medially. The wound is packed temporarily with moist gauze. The incision is continued forward (medial to the alveolar ridge) to a point behind the canine tooth. From this point, the incision continues at right angles to the apex of the cleft. In extending the incision down to bone, the lesser palatine vessels are severed and clamped. Thus, with the cleft, a V-shaped flap is formed.

The flap is now elevated at the mucoperiosteum-bony interface back to the posterior palatine foramen; the descending palatine vessels are dissected and preserved.
Three important steps follow: first, the nasal mucosa is separated from the posterior nasal spine; next, the nasal mucosa is freed from the superior surface of the palatal shelves; finally, the mucosa of the lateral pharyngeal wall is freed from the medial pterygoid plate up to the base of the skull. Strands of connective tissue tethering the soft palate to the maxillary tuberosity or to the posterior nasal spine are also divided.

The contralateral flap is similarly developed. The palate is closed in layers, starting with the nasal mucosa, which is closed with 4-0 chromic catgut. Several nylon vertical mattress sutures are placed in the uvula. The muscularis and mucosal layers are closed as one, using interrupted and vertical mattress sutures. A single suture placed in the nasal mucosa at the junction of the hard and soft palates incorporates the oral mucosal-muscularis layer and is tied to obliterate any "dead space". The anterior ends of the flaps are sutured to each other and to the triangle of mucoperiosteum over the incisive foramen.

A variation of this technique can be employed if the cleft traverses the alveolus. In the four-flap technique, the incision inside the alveolus is extended more anteriorly. The right angle incision at the cleft margin is made as before. However, four mucoperiosteal flaps are raised, two based anteriorly and two posteriorly. The nasal mucosa is sutured to that of the vomer to provide closure of the nasal layer. The four-flap technique can also be used with vomer flaps in the closure of bilateral clefts. As in the Oxford technique, the oral mucoperiosteal flaps are approximated, and vertical mattress sutures are used to join the velar musculature in the midline.

**Palatoplasty and Primary Pharyngeal Flap: The Author's Technique**

If the palatoplasty fails and the patient speaks with rhinolalia aperta, speech therapy is usually instituted at about the school age and is continued until improvement ceases. If speech remains imperfect, a secondary palate lengthening procedure is proposed, commonly the addition of a pharyngeal flap (see Chapter 52).

It occurred to the author (Stark and De-Haan, 1960) that the ideal time to perform the pharyngeal flap procedure is when the cleft palate is open and there is an unimpeded view of the pharynx, a principle applied by Burian (1954) and designated by him as "pharyngeal fixation". The flap can be developed, bleeding controlled by electrodesiccation, the donor site closed, and the flap sutured to the velum after the palate has been repaired. To do this in every case, however, would be to subject 60 to 75 per cent of the total cleft palate patients to a needless addition of a pharyngeal flap, as this percentage would speak normally with palatoplasty alone. There is, as yet, no means available of determining which infants with cleft palate have palatal shelves severely deficient of mesoderm, as opposed to those who have mesodermally sufficient palates which are merely cleft. The addition of a pharyngeal flap to the palatoplasty adds a small amount of operating time and has virtually no side effects. Adding this step to whatever type of palate repair that has been performed increases the number of patients with acceptable speech beyond the expected 60 to 75 per cent for palatoplasty alone to approximately the 90 per cent level. Although the flap in many cases may be unnecessary, the author feels it is a safe "ounce of prevention" which allows 15 to 25 per cent of the total number of patients to avoid speech therapy and secondary procedures to collect velopharyngeal incompetence.
For 20 years, the author has combined pharyngeal flap with palatoplasty of the von Langenbeck type as the primary palatal operation at the age of 1 year. The V-Y retroposition operation has not been combined with the primary pharyngeal flap at this age, since this procedure tends to force an excessive amount of soft tissue into the tiny oropharynx. However, retroposition of the V-Y type can be used with a pharyngeal flap as a secondary procedure for the improvement of speech.

**Technique.** A suture is placed in each avascular half for traction. A saline and epinephrine solution (1:100000) is injected into the posterior pharyngeal wall, into the soft tissues of the velum, and into the mucoperiosteum of the hard palate. After a wait of not less than five minutes for the vasoconstriction effect, the vertical lateral margins of the pharyngeal flap are incised, the wound margins distracted by spreading the scissors, and the wounds packed with saline-epinephrine gauze pledgets. Any remaining bleeding is controlled by electrocoagulation. The flap is undermined at the superior constrictor-prevertebral fascia interface. Traction sutures are placed in the two sides of the flap. The flap end is transected with a right angle scissors.

It appears to be of little import whether the flap is based superiorly or inferiorly as far as speech results are concerned. Indeed, after approximately six months it is difficult to determine, upon lateral roentgenogram, in which direction the flap was based, since the base migrates to a midposition somewhere between the two extremities. The presence of a friable adenoid pad constitutes the only indication for basing the flap superiorly, since, if it were based inferiorly, flap disruption might result as the sutures cut through the adenoidal tissue at the end of the flap.

The donor site in the midline of the pharynx is closed. This diminishes the likelihood of morbidity from sepsis with its potential complication, mediastinitis. In addition, primary closure reconstitutes the hemisphincter that is the superior constrictor and, in addition, diminishes the transverse diameter of the oropharynx.

The palatoplasty begins with incising the margins of the cleft, preferably removing a single, continuous strip of epithelium. In this way, the surgeon is assured that no epithelial remnant remains which could produce a fistula.

Relaxing incisions identical to those of the V-Y retroposition are made with the exception of the right angle extension from the canine tooth to the anterior extremity of the cleft, i.e. it is made along the pterygomandibular raphe, around the maxillary tuberosity, inside the alveolus to a point slightly beyond the anteriormost extremity of the cleft. In the velum, the wound margins are distracted by spreading the scissors. The wounds are packed with pledget soaked with saline-epinephrine solution.

Mucoperiosteum is released from the underlying maxillary and palatine bones by using a periosteal elevator. The dissection must be performed with care, moving the elevator from side to side and avoiding forceful forward pushing. By spreading the scissors carefully, the descending palatine vessels and the anterior palatine bundle can be identified and preserved as they emerge from the greater palatine foramen.
The soft tissue tethering (aponeurosis of the soft palate) at the posterior end of the hard palate can be handled in several ways. It can be dissected free of the nasal surface of the palatal shelves, as described in the V-Y retroposition procedure, or it can be transected. The latter procedure leaves an unepithelized area on the nasal surface that may be resurfaced in the manner of Cronin (1957), who uses a right angle knife to incise the nasal mucous membrane in its posterior part, then dissects and retrodisplaces it to cover the raw area. It may be surfaced with an island flap which is transplanted with its neurovascular pedicle to the raw nasal defect (Millard, 1966). Finally, if a pharyngeal flap is used with stronger posterior traction force than is exerted anteriorly by granulation and cicatrization of the nasal defect, nasal lining is not necessary.

The decision whether or not to fracture the hamulus is an individual one. Fracture of this bone, which slackens the pull of the tensor muscles, has been a traditional step in cleft palate repairs since the time of Billroth (1889). It was felt that the incidence of wound disruption or fistula formation was lessened if the pull of the paired tensor muscles were diminished in the postoperative period. Recently, it has been suggested that the opening-closing mechanism of the eustachian tubes may be impaired by hamular fracture (Kriens, 1970). In a serial radiographic study, Thomson and Harwood-Nash (1972) reported that the fractured hamulus eventually reverted to its preoperative anatomical position.

The palate is closed in layers using medium caliber silk sutures. The nasal surface is closed, the knots being tied on the nasal mucosal side. The mucosa-muscularis of the velum is treated as one layer and sutured with vertical mattress and interrupted sutures. The bipedicled mucoperiosteal flaps are approximated with silk over the hard palate.

The inferiorly based pharyngeal flap is pulled forward, and its farthest advance upon the velum is marked. The underlying uvula-velar mucosa is removed, and the flap is inset using approximately six interrupted sutures. If the flap has been based superiorly, a small hinged flap of nasal mucosa with muscle is turned back from each uvular segment before the velum is closed in this area. The pharyngeal flap is sutured to the donor sites of the hinged flaps, and the hinged flaps are sutured to the pharyngeal flap.

Packs are not employed, even in the open wounds that result from the relaxing incisions. These wounds quickly epithelize without misadventure.

The hypopharynx is suctioned, and a traction suture of medium silk is placed in the anterior part of the tongue as a potential aid to the airway during the postoperative period. The infant remains in the recovery room until fully reacted.

In the postoperative period, the infant should be fed a clear fluid diet for four days. Milk and soft ice cream may be added until the eight day, when soft food (blenderized foods, eggs, bread minus the crusts, and so forth) is allowed. A regular diet may be resumed by the fifteenth day. All of these feedings should be taken from a glass or cup. Straws or spoons introduced into the oral cavity are prohibited. Non flexible sleeves should be worn to keep the fingers out of the oral cavity. Antibiotics are not routinely used. The febrile response during the first few postoperative days usually subsides as hydration improves.
The speech results in 32 patients with primary pharyngeal flap combined with palatoplasty performed at 1 year of age and tested five years postoperatively were "normal" in 90 per cent (29) of the patients and only slightly less than normal in the other three patients.

Hearing in all of these patients was sufficiently good not to require a hearing aid: 20 had no hearing loss, a bilateral loss of 0 to 15 decibels was present in six, and a loss of 15 to 30 decibels was found in five, while one patient had a loss of greater than 30 decibels.

Of the 32 patients, seven had a cleft of the secondary palate alone, in which there was no maxillary collapse. Of the remainder, 14 were found to have normal occlusion at the age of 5; six had an anterior crossbite; three had a unilateral collapse.

Additional operations have been performed at the age of 1 year, but either the patients have not reached the determinate age of 5 years or have not yet been evaluated. Many more have had a primary pharyngeal flap with palatoplasty, though after speech had begun. These cases demonstrated the herculean problem of speech reversal (see Chapter 52).

Complications

A number of complications may be associated with the surgical correction of cleft palate: impaired airway, hemorrhage, wound disruption or dehiscence, fistula formation, stertorous breathing, or imperfect speech due to a physiologically short and/or immobile velum.

Impaired Airway. The airway may be compromised at the time of surgery by secretions and bleeding and in the postoperative period by edema. To combat aspiration at the time of surgery, the anesthetic agent should be administered by endotracheal tube. The latter may be responsible for the development of postoperative laryngotracheal edema, which is treated by steam inhalation and the administration of systemic steroids. Tracheotomy, a dangerous procedure in an infant, is rarely indicated. In the immediate postoperative period, maintenance of an adequate airway is enhanced by placing a traction suture in the tip of the tongue, so that the tongue may be pulled away from the posterior pharynx.

Hemorrhage and Blood Loss. With the use of a chemical vasoconstrictive agent (epinephrine in saline, 1:100000 to 1:200000), bleeding is rarely brisk and is easily controlled with electrocoagulation. Blood replacement should rarely be necessary. Few accurate studies of blood loss exist. Tempest (1958) found that in palatoplasties operative blood loss averaged 140 mL, while Dingman, Ricker, and Job (1949), using epinephrine as a vasoconstrictive agent at the time of surgery, reduced the average blood loss to 60 mL. In the author's series, blood loss following the injection of epinephrine in saline (1:175000) into the soft tissues (velum and mucoperiosteal flaps) during combined palatoplasty and transfer of a primary pharyngeal flap was 60 mL (patients under the age of 2 years) and 90 mL (patients from 2 to 12 years).

Wound Disruption or Dehiscence. Suture of the mucoperiosteal flaps and velum under tension invites wound disruption when the patient cries, speaks, or eats. Sedation will help to control crying. Absence of tension reduces the danger of wound disruption, as has
neutralizing of the pull of the tensor muscles by hamular fracture. It remains to be seen whether wound disruption will increase if the hamulus is left intact because of concern for the middle ear problems.

Wound dehiscence may be caused by underlying systemic disease or by the chronic administration of high doses of corticosteroids.

**Fistula.** Failure to take the precautions mentioned above may result not only in wound disruption but also in the production of fistulas. An added and common cause of fistula is residual epithelium because of the failure to excise completely or to incise the cleft margins prior to palatoplasty. Fistulas may close spontaneously, or closure may be facilitated by infrequent application of silver nitrate cauterization to the margins of the fistula. Unless they are 5 mm or more in diameter, fistulas rarely account for either nasal escape of food or nasal speech. The treatment of palatal fistulas is discussed in Chapter 47.

**Nasal Speech.** Rhinolalia aperta, or speech typical of cleft palate, will result if palatoplasty is deferred until after speech has begun (delayed operation) or if the palate, even though closed surgically, has a short anterior-posterior dimension. Short anteroposterior development results from inept, traumatic surgical technique or from a mesodermal deficiency of the velum. If palatoplasty is performed prior to the onset of speech, the problem of speech reversal from rhinolalia aperta to velopharyngeal competence is usually obviated. If the palatoplasty is a palate lengthening procedure of the retropositioning or pharyngeal flap type, the likelihood of development of a short anteroposterior palatal dimension is minimized.

**Secondary Surgery.** With a failed palatoplasty in which rhinolalia aperta has persisted in spite of at least six months of speech therapy, the patient usually needs a V-Y retroposition or a pharyngeal flap operation.

The V-Y retropositioning operations if performed essentially as outlined for the primary operation.

In the secondary pharyngeal flap operation, the short, often scarred velum may form a barrier to easy exposure of the pharynx. Consequently, the velum may have to be divided in the midline and the two halves retracted.

Pharyngoplasty techniques to correct velopharyngeal incompetence are outlined in Chapter 52.

**Submucous Cleft Palate**

*Miroslav Fára, R. C. A. Weatherley-White*

The submucous cleft is the rarest form and appears anatomically to be the least serious form of cleft palate. However, it may cause imperfect palatopharyngeal closure, and surgical treatment or speech therapy may eventually be required. Curiously, the surgical results in terms of speech proficiency have been distinctly worse than those achieved following closure of the complete cleft.
**Diagnosis.** In order to establish the diagnosis of submucous cleft, the examination of the patient must demonstrate the following clinical findings: (1) a median depression or notch in the hard palate; (2) failure of the soft palate muscles to join in the midline, although the mucous membrane appears to be intact (zona pellucida); and (3) a bifid uvula.

The diagnosis is facilitated by the finding of a strikingly thinner midline area between the bulging muscles on either side of the soft palate. In more severe cases, when the nasal and oral mucous membranes are in contact between the separated muscles, the zone is usually whitish or partly translucent (zona pellucida). The notch in the hard palate may be of varying length; the bony cleft can reach the incisive foramen.

If the gag reflex is induced, it tends to exaggerate the defect in the hard palate. The soft palate muscles bulge when they contract, and the central part of the soft and hard palates elevates and flattens. A light source placed in the nasal cavity shows the defect by transillumination. Palpation confirms the actual size of the cleft in the hard palate.

A cleft of the uvula alone is not a reliable sign of a submucous cleft, as its incidence in the healthy population is greater than 1 per cent. However, in a patient with a submucous cleft of the soft and hard palates, the uvula is rarely undivided and thus should alert the clinician to the possibility of a submucous cleft.

Rarely, congenital perforations of the anterior hard palate are seen in conjunction with submucous cleft; examples of these are seen in the figure.

The most important functional symptom of submucous cleft is a speech disorder resulting from incomplete velopharyngeal closure as the consequence of a short or poorly mobile palate. Middle ear disease with consequent hearing loss may also be a frequent related finding.

**Differential Diagnosis.** The submucous cleft should not be confused with two pathologic conditions of the palate which also cause a disorder of speech. The first is the inadequate palate without cleft which is anatomically deficient ("short palate"); the second is one which is functionally abnormal because of a neurologic disorder.

In these conditions, confusion may occur if a coincidental cleft of the uvula is also present. However, the muscular diastasis is not apparent, and instead of a dent in the posterior edge of the hard palate, one can palpate an intact posterior spine.

**Incidence in the United States.** There are no figures which demonstrate the absolute incidence of the anomaly. Most series derive from a large number of cleft palate repairs; in these reports the incidence of submucous clefts ranges from 5 to 10 per cent of the cleft palate population. It is generally described as a "rare" variant of cleft palate, and the inferred incidence from these reports would be 1 in 10,000 to 1 in 20,000.

Although asymptomatic submucous cleft is recognized, it has been felt to occur only infrequently and primarily in clefts of minimal proportions. Kelly (1910) assumed that a hyperactive pharyngeal constrictor mechanism compensated for the palatal defect, but no recent authors have attempted to explain abnormalities which in some cases can produce
defective speech patterns and in others permit the acquisition of normal speech. In light of
the prevailing philosophy that submucous clefts should be operated upon when recognized,
it would seem important to try to define those factors which would predispose toward
velopharyngeal incompetence. There is no information concerning the relative frequency of
asymptomatic versus symptomatic submucous clefts.

In 1970, the Cleft Palate Clinic of the University of Colorado School of Medicine
addressed itself to these unresolved questions (Weatherley-White and associates, 1972). The
thrust of the project was twofold: first, to determine the incidence of submucous cleft palate
in the normal population; and second, to define those factors, anatomical and otherwise, which
cause pathological speech. Inherent in this latter goal was the development of diagnostic
criteria for surgical repair.

To accomplish the first objective, a survey was made of the 10,836 children then
enrolled in the Denver School System. All children were examined by the school physician
or nurse after pretraining to recognize submucous cleft palate, and any abnormalities of the
lips or palate (including repaired cleft) were reported. All reported children were then
examined by two pediatricians from the Cleft Palate Clinic, and if a submucous cleft were
suspected, the children were examined by a plastic surgeon and a speech pathologist.

To qualify as a submucous cleft subject, the patient had to demonstrate all three of the
principal signs: hard palate notch, muscle diastasis, and bifid uvula. Several children with a
bifid uvula and a small notch in the hard palate but no muscle diastasis were thus excluded
from the study.

The findings of the school survey are shown in Table 45-1.

Table 45-1. Incidence of SMCP in a Random Population

<table>
<thead>
<tr>
<th>Total number of children surveyed</th>
<th>10,836</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal palate</td>
<td>10,599</td>
</tr>
<tr>
<td>Suspected abnormality</td>
<td>237</td>
</tr>
<tr>
<td>Submucous cleft palate</td>
<td>9</td>
</tr>
<tr>
<td>Bifid uvula</td>
<td>108</td>
</tr>
<tr>
<td>Repaired cleft palate</td>
<td>9</td>
</tr>
</tbody>
</table>

There were 108 bifid uvulae, an incidence of 1:100, a figure which corresponds to
other studies. There were nine submucous clefts, or an incidence of 1:1,200, far higher than
had been previously assumed. Of these nine patients, seven were entirely asymptomatic; one
had a very mild speech defect corrected by a brief period of speech therapy, and one had had
recurrent episodes of serous otitis. There were nine repaired clefts identified in the same
group of school children, a similar incidence to that of submucous cleft.

These nine children were included in a larger group of 61 patients with newly
diagnosed submucous cleft palate referred to the Cleft Palate Clinic at the University of
Colorado School of Medicine. All received the following battery of tests, described in more
detail elsewhere (Weatherley-White and associates, 1972):
1. Each patient was examined by a pediatrician, a plastic surgeon, and otolaryngologist.

2. A detailed speech evaluation was performed.

3. Cineradiography was performed in most instances.

4. Other tests included audiograms, impedance studies, psychometrics, and others necessary to confirm associated abnormalities.

The results of our study can be summarized as follows.

**Speech Evaluation.** Only patients 5 years and older underwent speech evaluation; there were 44 eligible patients, and the results are listed in Table 45-2.

<table>
<thead>
<tr>
<th>Speech Evaluation</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>22</td>
</tr>
<tr>
<td>Abnormal due to V-P incompetence</td>
<td>11</td>
</tr>
<tr>
<td>Mildly defective</td>
<td>3</td>
</tr>
<tr>
<td>Moderately defective</td>
<td>7</td>
</tr>
<tr>
<td>Severely defective</td>
<td>1</td>
</tr>
<tr>
<td>Abnormal due to other defects</td>
<td>11</td>
</tr>
</tbody>
</table>

The table is unavoidably biased toward an incorrectly high percentage of abnormal speakers, owing to the tendency to refer the abnormal speaker more readily for medical evaluation. In the school survey, which gives a more representative incidence of speech problems in submucous cleft, only one of the nine patients had a speech defect. It can also be seen that, although 50 per cent of the submucous cleft patients had speech abnormalities of some sort, they were just as often due to other speech defects as to true velopharyngeal incompetence. The very high incidence of other speech defects was due mainly to mental or motor retardation and to hearing losses secondary to serous otitis media and other inner ear disturbances. A majority of patients (40 out of 61) had associated congenital anomalies.

**The Degree of Severity of the Cleft.** The degree of the muscular cleft was classified as "minimal" if the muscular diastasis was less than 0.25 cm wide and involved less than half of the soft palate; as "moderate" if the diastasis was between 0.25 and 0.5 cm wide and involved more than half of the soft palate; and as "severe" if the diastasis was 0.5 cm or more wide and extended into the bony cleft. Table 45-3 summarizes the number of patients in each group correlated with their respective speech performances.

<table>
<thead>
<tr>
<th>Degree of Cleft</th>
<th>Number</th>
<th>Normal Speech</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal</td>
<td>14</td>
<td>7 (50%)</td>
</tr>
<tr>
<td>Moderate</td>
<td>15</td>
<td>7 (50%)</td>
</tr>
<tr>
<td>Severe</td>
<td>10</td>
<td>6 (60%)</td>
</tr>
</tbody>
</table>
The severity of the palatal cleft would seem to be a logical basis for classification of submucous cleft palate, but in this study no correlation between the degree of cleft and the speech proficiency was found. There was an approximately equal number of abnormal speakers in each anatomical group. No accurate prognosis for speech can therefore be made by anatomical identification alone.

**Length of the Palate.** The patients were divided on the basis of lateral cineradiography into three groups, depending on the ratio of the length of the soft palate to the depth of the pharynx. The optimum length of the soft palate is at least 1.5 times the depth of the pharynx, so that closure can be made with the active middle third of the palate. If the soft palate is between 1.0 and 1.4 times the depth of the pharynx, closure will occur along the posterior third of the soft palate. These two situations were classified as "adequate" and "marginal", respectively. Finally, if the palate is shorter than the pharyngeal depth (a ratio of less than 1.0), closure will be anatomically impossible. Table 45-4 shows the correlation of relative palatal length to speech proficiency.

**Table 45-4. Relative Palatal Length and Speech**

<table>
<thead>
<tr>
<th>Ratio of Palate to Pharynx</th>
<th>No</th>
<th>Normal</th>
<th>V-P Incompetence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adequate</td>
<td>10</td>
<td>7 (70%)</td>
<td>0</td>
</tr>
<tr>
<td>Marginal</td>
<td>16</td>
<td>6 (37%)</td>
<td>8 (50%)</td>
</tr>
<tr>
<td>Short</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

There were no patients with adequate palatal length who had typically "cleft palate" speech; however, three patients with adequate palatal length had speech defects due to other reasons and radiographically demonstrated very uncoordinated palates. By contrast, 50 per cent of the patients with a marginal palatal length had velopharyngeal incompetence with its characteristic stigmata. Presumably all "short" palates would have demonstrated this abnormality.

**Palatal Mobility and Fatigue.** The mobility of the soft palate was measured by the time, in milliseconds, required to accomplish the close-open-close sequence of the word "simple". Mobility is compared to speech performance in Table 45-5.

**Table 45-5. Mobility of the Palate and Speech**

<table>
<thead>
<tr>
<th>Mobility (in msec)</th>
<th>No</th>
<th>Normal</th>
<th>V-P Incompetence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Faster than 150 msec</td>
<td>3</td>
<td>3 (100%)</td>
<td>0</td>
</tr>
<tr>
<td>150-200 msec</td>
<td>10</td>
<td>7 (70%)</td>
<td>3 (20%)</td>
</tr>
<tr>
<td>Slower than 200 msec</td>
<td>5</td>
<td>2 (40%)</td>
<td>2 (40%)</td>
</tr>
<tr>
<td>Lack of coordination (no release)</td>
<td>4</td>
<td>0</td>
<td>2 (50%)</td>
</tr>
</tbody>
</table>

A good correlation between the mobility of the palate and speech performance was found in the normally active (less than 150 msec) group; all of these patients had normal speech. Of the patients who accomplished the "close-open-close" sequence between 150 and 200 msec, 70 per cent had normal speech. Only two patients (40 per cent) in the group slower
than 200 msec demonstrated normal speech. The largest variable in this measurement seemed to be the rate at which the subject said the words "Simple Simon".

If the mobility of the palate during the first and tenth repetition of the words "Simple Simon" were compared, this source of error would tend to be nullified, as each patient would act as his own control. This comparison determined the tendency of the palate to fatigue during conditions of muscular work, and using this concept, patients were grouped into three categories.

1. "Improved mobility", in which the palate accomplished the sequence faster on the tenth than on the first repetition.

2. "No change".

3. "Lessened mobility", in which the palate sustained fatigue during the repetitions and either slowed, or failed to make contact at all, on the tenth.

Table 45-6 shows the division of patients into these categories and the speech performance of each group.

**Table 45-6. Fatigability and Speech**

<table>
<thead>
<tr>
<th>Fatigue Factor</th>
<th>No</th>
<th>Normal</th>
<th>V-P Incompetence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved mobility</td>
<td>11</td>
<td>11 (100%)</td>
<td>0</td>
</tr>
<tr>
<td>No change</td>
<td>5</td>
<td>2 (40%)</td>
<td>3 (60%)</td>
</tr>
<tr>
<td>Lessened mobility</td>
<td>2</td>
<td>0</td>
<td>2 (100%)</td>
</tr>
</tbody>
</table>

An excellent correlation exists between fatigue of the palate and speech proficiency; 100 per cent of the patients with "improved mobility" demonstrated normal speech, and 100 per cent of those whose palates became fatigued during speech demonstrated typical "cleft palate" speech patterns. The "no change" group fell neatly in the middle, 60 per cent having velopharyngeal incompetence and 40 per cent having normal speech.

**History of Diagnosis and Therapy.** In 1825 Roux described the cleft with bifid uvula and thinned central portion of the soft palate. This is considered to be the oldest report of a submucous cleft of any variety. In 1846 Demarquay published an anatomical study of the defect. Passavant alluded to submucous cleft in two of his reports in 1862 and 1865. Von Langenbeck in 1864 divided submucous clefts into three groups according to their location: (1) in the soft palate only; (2) in the hard palate; and (3) in both simultaneously. Trélate described the familial incidence of submucous cleft (father-son) in 1870.

In 1910 Kelly, in the earliest report in the English literature, introduced the term "submucous cleft palate", which became familiar and is used at the present time. Other reports dealing with this defect in subsequent years were contributed by Seeman (1924), Limberg (1927), Dorrance (1930), Veau (1931), Burian (1954), Calnan (1954), Gylling and Soivo (1965), Crikelair, Strikes and Cosman (1970), and Weatherley-White and associates (1972).
Historically, it had been suggested that by training the soft palate sufficient rehabilitation of the muscles could be achieved to make palatopharyngeal closure function adequately. Today, however, this would be considered only a preliminary approach.

Submucous clefts were already treated by surgery in the nineteenth century, and the history of submucous cleft palate surgery includes all of the known procedures used in other types of clefts: retroposition of the palate, push-back procedure with the application of a skin graft, attachment of the soft palate to the posterior pharyngeal wall by means of a superiorly or inferiorly based pharyngeal flap, and different types of pharyngoplasties. From the very outset there was much discussion of the advisability of excising the central mucosal zone between the separated muscles.

**Statistics from the Prague Plastic Surgery Clinic.** At the Clinic of Plastic Surgery in Prague, 83 patients have undergone surgery for correction of a submucous cleft. The represents an incidence of 2.9 per cent of a total of 2846 surgically treated primary and secondary clefts of the palate and 5.4 per cent of 1539 isolated secondary clefts of the palate. In two patients, the submucous cleft was associated with an incomplete cleft of the lip. Twenty patients in the series manifested symptoms of the "syndrome of developmental shortening of the palate" (Fára, Hrivnáková and Sedláčková, 1971).

In three-fourths of the patients, there was a wide parting of the muscles of the soft palate with the typically translucent central zone; in one-fourth of the patients, the cleft was not prominent. In 75 per cent of the cases, the entire uvula was cleft, whereas in the remaining 25 per cent, the uvula was partially cleft. The defect in the posterior margin of the hard palate was extremely variable. A minimal depression in the posterior margin of the hard palate was noted in five patients, a dent of 5 mm in eight cases, and a notch of 5 to 10 mm in 17 cases; the depression extended into the palatal vault in 21 individuals and into the incisive foramen in 14 cases (in five of the 14, the vault disclosed a congenital perforation of the soft tissues); and in 18 cases, there were insufficient records.

There were 36 male and 47 female patients. A familial incidence of cleft was noted in eight patients: brother-sister in two cases, sister-sister in three cases mother-daughter in one case, and more distant relative in two cases. Of the associated familial clefts, the cleft of the palate was either submucous (two cases) or complete (six cases); there was no patient with associated cleft of the lip.

The mean age at the time of surgical correction was 9.89 years. Prior to the year 1945, the mean age was 16.2 years but fell to 9.02 years in the past 25 years. The age at which the operation is performed appears to make a distinct difference in the functional results achieved; the earlier the recognition and correction of the anomaly, the better will be the speech ultimately attained.

**Treatment at the Prague Plastic Surgery Clinic.** The indication for operation is hypernasality or nasal emission secondary to velopharyngeal incompetence. Surgery was not recommended for patients with submucous cleft who did not manifest the functional sequelae of the defect. Consequently, the number of submucous clefts operated upon was less than the actual incidence in the general population. The incidence of submucous clefts without a functional defect in patients speaking Czech is, however, relatively small and according to our
investigations does not exceed 10 per cent (Fára and Hrivnáková, 1968). This is in contrast to the results of the University of Colorado study, which demonstrated a much higher incidence (60 to 90 per cent) of asymptomatic submucous cleft palates (Weatherley-White and associates, 1972).

In the majority of our patients, surgical therapy was followed by speech therapy, which had been started before the operation in almost 30 per cent of the group. Massage of the soft palate is considered an inseparable component of the preoperative and postoperative therapy.

The operation, apart from the suture of the muscles in the palatal midline in 81 cases, consisted of primary pharyngofixation (Fára and coworkers, 1970). A superiorly based pharyngeal flap was added in 50 cases, of which nine were tubed (Fára, Hrivnáková and Sedláčková, 1971).

Simultaneous palatal push-back was performed in 47 of the more recent cases. In the first 20 years of the study (36 cases), it was not part of the operative procedure. In the last ten years we have stressed the careful detachment of the muscle insertions from the posterior margin of the palatal plates and their end-to-end suture in the palatal midline (Fára and coworkers, 1971).

In patients in whom the separation between the muscle was narrow and the mucous membrane was well developed, the operation was modified by avoiding any incisions in the oral mucosa, and the surgery on the soft palate was done from the nasal side. After the mucoperiosteum was elevated over the hard palate as one flap (Dorrance, 1933), the muscle insertions at the posterior edge of the hard palate were detached and displaced backward. A 1.5-cm incision was then made in the nasal mucous membrane at the posterior edge of the hard palate.

The edges of the incision were retracted, establishing a quadrangular defect, thus pushing the nasal side of the soft palate posteriorly. Therefore a better approach to the muscles was obtained, enabling them to be sutured in the midline. The superiorly based pharyngeal tube flap was placed into the nasal defect and sutured in place with catgut sutures. Finally, both halves of the uvula were sutured.

**Anatomical Findings.** The operative procedures afforded opportunity to study the condition of the mucous membrane in the central zone of the palate, the direction and insertion of the muscles, and the osseous changes in the cleft region. It was found that the mucous membrane was the most hypoplastic in cases in which the muscles were widely separated and the oral and nasal mucous membranes were fused; the area of fused mucous membrane was routinely excised. In the majority of patients, however, although the muscles were separated, a layer of mesodermal tissue of variable thickness was present between the mucosal layers. After an incision in the midline, it sufficed to separate the layers of the mesodermal tissue in order to expose the muscles for approximation and suture.

In the more severe submucous clefts, the arrangement of the muscles was similar to that in complete or open types of palatal clefts. The muscle bundles were folded forward and attached to the posterior margin of the hard palate. In the less severe cases, the folding was only partial; furthermore, some muscle fibers proceeded from the edges of both lateral muscle
groups toward the midline and even joined the fibers of the opposite side. In view of these variations in the velar muscles, an attempt was made to reconstruct the palatal muscle ring during the surgical procedure.

**Histologic Findings.** Histologic examination was done on the excised specimen transversely removed from half of the length of the soft palate. The excised segments comprised the entire medial zone with the nasal and oral mucosa and included the muscle edges on both sides.

The microscopic appearance of the muscles was directly related to the visible width of the cleft. In the least severe cases, which were few in number, numerous muscle fibers intermingled across the midline of the soft palate. The central zone, however, was considerably thinned. The nasal and oral mucous membranes were well developed.

In the more severe cases, in which the muscles were widely separated, the muscle fibers proceeded parallel to the midline cleft. The central zone of excision consisted predominantly of dense collagenous connective tissue. However, the mucous membrane on the nasal and oral sides did not differ histologically from the mucous membrane in the unaffected palate.

In the most severe cases, the muscles were widely separated, and the muscle fibers ran parallel to the central defect. Between the oral and nasal mucous membrane, there was a thin layer of dense collagenous connective tissue. Both mucous membrane layers showed a reduction in the size of epithelial layers in a lateromedial direction. The oral mucous membrane was far more significantly affected in this respect than the nasal mucous membrane. In the midline, it was formed only by a few layers of strikingly flattened cells.

**Developmental Shortening of the Palate.** The syndrome of developmental shortening of the palate has been described by Sedláčková (1955). The main symptom of this syndrome is hypernasality, and in more serious cases there can be speech problems caused by a shortened and inadequate soft palate with strikingly underdeveloped muscles. Only a small number of these individuals manifest a submucous cleft; the authors have included in this group patients with both a shortened palate and submucous cleft.

These patients are characterized by their unique physiognomy: a wide root of the nose, narrow palpebral fissures, narrow external auditory canals and airways, and hyposmia. The upper lip is vertically shortened; the philtrum is poorly defined. The auricles are short, and the lobules are directed diagonally.

**Conclusions.** The incidence of submucous cleft palate appears to be far higher than previously recognized and may in fact equal that of over cleft (1:1200). The majority of these clefts are entirely asymptomatic, which accounts for the previously held assumption that submucous cleft is a "rare" variant of cleft palate.

This finding makes the recommendation that the anomaly be operated on when recognized quite untenable. With increasing awareness of submucous cleft on the part of general practitioners, pediatricians, and nursing personnel, many more cases will be diagnosed in infancy than heretofore. To operate on all of these at a year or so of age, before speech
patterns have developed, would subject large numbers of children to unnecessary surgery and the attendant risk. Unfortunately, the anatomical classification of submucous cleft palate into mild, moderate, or severe is not useful in determining the prognosis for speech.

The concept of early surgery to prevent abnormal cerebral patterns of speech is, however, quite valid; hence, all patients, when diagnosed, should be referred to a cleft palate clinic to be followed closely for early indication of velopharyngeal insufficiency. Once velopharyngeal inadequacy is established, these children should undergo operative repair without delay.

Velopharyngeal incompetence in submucous cleft palate appears to be related to a relatively short, immobile, and easily fatigued palate. Of these factors, probably the most crucial determinant is the fatigability. As this phenomenon can be clarified only by the use of cineradiography and frame-by-frame analysis, the surgeon must, in practice, still depend on a sophisticated speech evaluation in determining which patients with submucous cleft palate can be aided by surgery.