Optimal Timing of Cleft Palate Closure

Rod J. Rohrich, M.D., Edward J. Love, M.D., H. Steve Byrd, M.D., and Donnell F. Johns, Ph.D.

Dallas, Texas

Learning Objectives: After studying this article, the participant should be able to: 1. Understand the speech, hearing, and growth implications of cleft palate repairs. 2. Understand the benefits and risks of early versus late palatal closure. 3. Formulate a plan for the timing of palatal closure. 4. Describe the long-term side effects associated with the timing of cleft palate closure.

Treatment objectives for the cleft palate patient—normal speech, normal maxillofacial growth, and normal hearing—are closely related. Controversy about the timing of cleft palate surgery is directed at the need for early palatoplasty for improved speech and hearing versus delayed hard palate repair for undisturbed facial growth. This controversy as to the value of early versus delayed closure continues into the present. The authors present an updated argument regarding this controversy along with a comprehensive literature review. They also present a logical algorithm based on the literature and their personal experience. (Plast. Reconstr. Surg. 106: 413, 2000.)

The functional goals of cleft palate surgery are normal speech, hearing, and maxillofacial growth.1,2 It is generally thought that speech and hearing are improved by early cleft palate repair (before 24 months of age)3 and that delayed closure (after 4 years) is associated with less retardation of midfacial growth.4,5 The primary goal in the timing of cleft palate surgery, therefore, is to provide adequate palatal function for the development of normal speech without interfering significantly with maxillofacial growth. This article reviews the literature in the controversial area of cleft care and proposes one logical algorithm for treating such patients.

Speech

Early cleft palate repair increases the likelihood of normal speech development.3,6 Delayed treatment may interfere less with midfacial growth, but speech development tends to be poor.7 If closure of the palate is delayed past 2 years and the mechanisms for speech have already developed, the chances for normal speech are significantly diminished.8 Oropharyngeal movements preliminary to speech apparently begin in the intrauterine period. As reported by Oiler et al.,9 the babbling and cooing of infants may be an important prelude to normal speech development.

Because most physiologic functions are optimally learned at an early age, it is logical that speech is also best learned early.10 It is thought that this occurs before 2 years. For this reason, speech pathologists recommend early palatal closure to restore the normal velopharyngeal mechanism and to enhance the chances for normal speech.3,11–13

Kaplan suggests that the ideal age for palatal repair is 3 to 6 months.14 This recommendation is based on the theory that the palate must be functional when palate-related sounds are first learned to avoid poor speech development and integration. Because the repaired palate has limited mobility for an additional 3 to 6 months because of postoperative edema, Kaplan advocates palatal repair at 3 to 6 months of age so that the palate can be functioning normally at 9 to 12 months. This was noted clinically by Wardill as early as 1937.15 Wardill believed that the ideal time to repair the palate was at younger than 1 year of age. However, because of the limitations of pediatric anesthesia at that time, he was unable to practice this himself.

In a study by Dorf and Curtin,3 12 months of age was used as an arbitrary dividing point between early and late palatal closure. They
found a 10 percent incidence of pharyngeal and glottal articulation abnormalities (common compensatory components in cleft palate speech) in those children who had repairs before 1 year and an 86 percent incidence of articulation abnormalities in those who had repairs after 1 year. This difference was statistically significant. Henningsson and Isberg\textsuperscript{16} made similar observations. The articulation errors were more prone to develop with late palatal repair because the pharyngeal and glottal abnormalities occurred as a compensatory mechanism.\textsuperscript{17}

In 1944, Schweckendiek\textsuperscript{18} advocated a two-stage repair with early closure of the soft palate. The hard palate was left open with the rationale that this would allow normal development of the maxilla. The oronasal fistula was occluded by a prosthesis until the hard palate fistula was closed at 15 years of age. Schroder,\textsuperscript{19} in the first description of the speech results from this protocol in 1966, was critical of Schweckendiek’s procedure. Schroder stated that most of the patients needed a pharyngoplasty and that the delayed hard palate closure did not prevent growth disturbances of the maxilla.

In 1979, Perko\textsuperscript{20} compared a group of patients who underwent one-stage palatal repair with a group who had undergone delayed hard palate closure and stated simply that “the temporarily remaining cleft in the hard palate does not disturb speech development to a relevant degree.” No speech data were provided on these patients, however.

In 1984 in a long-term follow-up study, Rohrich et al.\textsuperscript{2} reported on the incidence of speech deficiencies in 38 patients treated with delayed versus early closure of the hard palate. The overall speech intelligibility, articulation, and nasal escape were significantly reduced in the delayed closure group. This study showed conclusively that, in the long-term evaluation of cleft palate patients, delayed closure of the hard palate significantly decreased the patient’s chance for normal speech development.

In the same year, Henningsson et al.\textsuperscript{21} described speech intelligibility, hypernasality, nasal air emission, and articulation in a similar group of patients. The early closure group had significantly better intelligibility and fewer articulation errors than the delayed closure group. The articulation problems of the delayed closure group paralleled those found by our group\textsuperscript{2} and included frequent glottal stops, pharyngeal fricatives, and velar substitutions.

During this same period, Witzel et al.\textsuperscript{22} reviewed the rationale for delayed palatal closure and its effect on speech. They found that the data for a beneficial maxillofacial growth response with delayed repair were lacking and noted severe speech problems with delayed palate closure methods. They also pointed out the significance of oronasal fistulas in the development and persistence of articulation defects. Bzoch\textsuperscript{23} found that speech development was hampered by the use of an obturator (which was advocated by Gillies and Fry\textsuperscript{4}), primarily because of an inadequate seal. Noordhoff et al.\textsuperscript{24} reconfirmed this observation in an independent study.

In an attempt to link the type of operative procedure with its effect on speech, Trier and Dreyer\textsuperscript{8} compared the results of patients undergoing von Langenbeck’s palatoplasty without reconstruction of the levator veli palatini muscle with those of patients undergoing the same procedure but with intravelar veloplasty. They examined a total of 43 patients, who were divided equally between the two study groups and who had an average follow-up of 4 years and 7 months. All the patients underwent the palatoplasty at 14 to 16 months. The authors reported better speech and superior velopharyngeal function after intravelar veloplasty. They recommended careful reconstruction of the levator sling at the time of the palate repair.\textsuperscript{25–27}

In contrast, Holtmann and associates\textsuperscript{28} analyzed the early speech results obtained from V-Y pushback, the von Langenbeck technique, or the von Langenbeck technique with pharyngeal flaps in 62 patients. They found no differences among the treatment groups in terms of late complications, velopharyngeal insufficiency, or need for speech therapy. However, patients with the V-Y pushback required more blood transfusions, and those who had the von Langenbeck procedure with pharyngeal flaps had more airway obstruction. It seemed that the pharyngeal flap with a primary palatorrhaphy was unnecessary in 75 percent of the patients. These authors recommended only the von Langenbeck palatoplasty for primary cleft repair. Their speech results were early, with the latest assessment performed at 8 years.

In 1989, in a speech analysis study by Harding and Campbell,\textsuperscript{29} 48 patients treated with early versus delayed hard palate surgery were
compared. In general, delaying hard palate closure caused more persistent, noticeable, and abnormal speech patterns until after hard palate repair. However, both groups had tongue displacement and airstream problems. These findings reflect those of Dorf and Curtin and Cosman and Falk.

Speech Data Summary

The earlier the repair to the palate, the sooner the soft palate can begin to function normally and the sooner speech can begin integrating and developing without abnormal patterns. Furthermore, normal levator function and concomitant optimal speech results can be achieved by the release and repositioning of the levator muscle, without resorting to a mucoperiosteal pushback. In 1996, a long-term, multidisciplinary study described 44 patients who were similarly matched except for the timing and technique of hard palate repair. The study revealed significantly greater speech deficiencies with delayed hard palate closure, specifically in articulation, nasal resonance, nasal emission, and overall intelligibility assessment. The study is unique for its long-term follow-up. The average age at follow-up in the early closure group was 17.0 years versus 18.2 years in the late closure group. We noted a persistent palatal fistula rate in the late closure group of 35 percent in comparison with 5 percent for the early closure group. Therefore, the recent data are quite conclusive that speech integration and normal speech pattern development are superior with early palatal repair.

Maxillofacial Growth

The timing of palatal surgery and its effect on maxillary growth are likewise controversial. There is no doubt that the timing of palatal surgery affects maxillary growth. However, two other major factors must be considered. First, operative procedures to repair the primary palate (lip and alveolus) may be as harmful to maxillary growth as the palatal repair itself, causing alveolar collapse and subsequent growth impairment. Second, in cleft cases, there are varying degrees of inherent maxillary deficiency, because no one cleft is similar to another. It is obvious that these inherent growth deficiencies vary with the individual cleft types.

Several studies have suggested that severe facial growth disturbances are associated with combined cleft lip and palate repair and not with an isolated cleft palate closure. This helps to differentiate between growth impairment in patients with combined cleft lip and palate and those with a cleft of the palate only.

There are three general types of maxillofacial growth: anteroposterior, vertical, and transverse (alveolar arch). It is important to differentiate these because the transverse (alveolar arch) growth problems can usually be corrected orthodontically and they do not alter facial aesthetics as significantly as anteroposterior and vertical growth deficiencies, which usually must be corrected by orthognathic surgical techniques.

Patients with an isolated cleft palate may have midfacial hypoplasia, which is related to the inherent growth limitation of the congenital anomaly. Retrusion after repair may be minimal, and it is not greater than that in patients with unrepaired clefts of the secondary palate or submucous clefts. The isolated cleft palate must be considered separate from the cleft palate associated with the cleft lip because of the difference in the maxillary growth potential of each. Thus, facial retrusion in an isolated secondary cleft palate, as opposed to a cleft lip and palate, is related primarily to its inherent maxillofacial congenital deficiency and secondarily to growth impairment induced by the operative closure of the cleft palate.

In 1921, Gillies and Fry observed narrowing and posterior displacement of the maxillary arch in patients who had surgical repair of the hard palate. They proposed closing only the soft palate and obturating the hard palate with a prosthesis. In an interesting follow-up of a select group of 10 patients who had repairs that followed these recommendations, Walter and Hale reported that the comparison of cephalometric findings with accepted norms and an examination of study casts showed encouraging results for facial and occlusal development. According to the authors, the dental arch width harmony was quite good in this small, select group of patients who had no orthodontic care. These findings concur with a study by Poupard et al., which reported improved palatal occlusion using the Gillies-Fry protocol.

In 1925, Rayner concluded that, in his experience with 125 cleft palate cases, the hard palate repair in the first 2 years of life caused greater dental arch collapse than repair at 3 or
4 years. Hagemann,51 in 1941, on the basis of these studies, proposed delaying surgery until after the eruption of secondary dentition to minimize the degree of transverse alveolar collapse.

In 1944, Schweckendiek18 began early closure of the soft palate and delayed closure of the hard palate (until 12 to 15 years) to allow normal maxillary development. He postulated that this method would allow for normal speech and normal growth of the maxilla. When analyzing Schweckendiek’s results, Bardach et al.52 found excellent facial growth and occlusion, but 81 percent of these patients had a degree of velopharyngeal insufficiency and 86 percent had glottal and pharyngeal articulation problems.

Jolleys,53 in 1954, found no difference in maxillary growth in children who were operated on at 2 years and those operated on between the ages of 3 to 5 years. In 1974, Robertson and Jolleys54 compared actual occlusion and facial profile in cases of early hard palate closure at 12 to 15 months of age with those who had palatal closure at 5 years. They found no differences between the two groups by 4 years.

In 1983, Robertson and Jolleys55 abandoned the delayed closure of the hard palate in favor of early closure, having demonstrated no significant differences in facial growth. This correlated with the results of the Oxford Cleft Palate Study,33,34,56 which showed no statistical difference between dental arch width and facial growth when comparing early closure at 10 months versus late closure at 48 months. Furthermore, a statistically increased incidence of persistent palatal fistula occurred in patients with delayed palatal closure (35 percent) compared with those with early palatal closure (5 percent).

In a large series of 2000 patients, Koberg and Koblin57 noted no statistical difference in the severity of growth retardation after surgery up to 8 years. Early palatal surgery before 1 year of age did not cause greater maxillary growth inhibition than surgery at any other age. Interestingly, most midfacial retusion occurred from palatal surgery during the second phase of maxillary growth at 8 to 15 years. The authors concluded that, for hard palate surgery not to affect maxillary growth, the operation should be delayed until after age 15.

Koberg and Koblin57 further compared maxillofacial growth relationships in 1033 patients. They noted the greatest growth disturbances came from the Veau pushback method, followed by the von Langenbeck relaxing incisions method. The least amount of facial retrusion was noted in the two-stage operation of soft palate repair at 1 to 2 years and hard palate repair after age 12. Studies by Jolleys55 and Palmer et al.58 confirmed the deleterious effects of the pushback technique; however, Aduss59 and Bishara et al.38,60 found no difference in facial growth when the V-Y pushback was used.

The proposed advantage of V-Y pushback is to lengthen the palate and to achieve better speech; this supposed advantage has not been substantiated.60–62 This technique cannot be justified routinely because of the higher risk of midfacial retrusion, arch collapse, and palatal fistula. More importantly, there seems to be no speech benefit. The benefit of the pushback method is derived more from the release of the abnormal attachments of the levator and paltapopharyngeal muscles and the reconstitution of the levator veli palatini as an adjunct to palatal repair. Soft palate muscle release, retropositioning, and reconstruction of the muscle sling without a hard palate pushback obtain this functional lengthening. This results in good velar function and better speech development, without the growth inhibition caused by the anterior scar of the pushback.

It does not seem that the timing of the cleft palate repair is the major deterrent to facial growth interference; instead, the surgery itself may cause the deformity. The most severe deformities are seen in children who had their palates repaired between the ages of 8 to 12 years, which is the most rapid phase of maxillary growth.57

HEARING

Conductive hearing loss in patients with cleft palate has been known to occur for more than a century; eustachian tube dysfunction is the primary cause of middle-ear disease.65–67 In 1978, Bluestone68 reported an incidence of hearing loss in the cleft palate population ranging from 0 to 90 percent; the average was 50 percent. In addition, Paradise69 noted that otitis media in the cleft palate patient is almost universal. The incidence of hearing loss is reduced if the palate is closed early.26–72
Hearing has long been neglected in the controversy surrounding the timing of palatal closure. Yet a child’s ability to learn a language and to articulate intelligently depends primarily on his or her hearing capacity.73,74 A significant hearing loss, even one that is short-lived, during the active period of child development may have permanent and irreversible effects at the level of the brain stem.10

According to Chaudhuri and Bowen-Jones,70 children who had palatal repair before 1 year had a 10 percent incidence of hearing loss, compared with 60 percent for those who had surgery at an older age. Although it is accepted that these children have middle-ear effusion, it is not known at what stage middle-ear malfunction occurs. The child with a cleft palate may have a malfunction at birth.

Yules75 concluded that approximately 50 percent of the cleft palate population suffered some hearing loss and that 94 percent have ear diseases arising from early serous otitis media. Middle-ear disease remains a problem well into later life.

Too-Chung71 conducted an interesting study using tympanometry in infants to assess middle-ear function. He found that all cleft palate children had normal aeration of the middle ear at birth and that this remained normal until 17 weeks of age. Closure of the cleft palate at or before 4 months of age decreased middle-ear complications.

In 1986, Watson et al.72 found that, in the long-term follow-up of patients with early versus late closure of the hard palate, those with late closure had a significantly higher incidence of otitis media or of the number of ventilation tubes inserted. They concluded that the late closure of the hard palate had an adverse long-term effect on hearing and suggested frequent follow-up of these patients throughout adolescence and adulthood to assess their otologic status. This is in agreement with an earlier study by Bennett76 on hearing loss in the older cleft palate patient.

After early repair of the palatal defect, eustachian function may still be abnormal, and hearing impairment may result.77 Subtelny12,78 found that, anatomically, cleft palate patients have a wider intertubercular distance and corresponding decrease in the height of the nasopharynx, which affects the angle of inclination to the eustachian tube and the action of the tensor tympani dilator muscle. This was confirmed by Maue-Dickson et al.79

The cartilaginous portion of the eustachian tube may also be poorly developed, as shown by Rood and Stool.80 The pressure differential between the nasopharynx and the tympanic cavity may reduce ventilation and drainage of the middle ear. This may initiate an inflammatory response from the regurgitation of food that subsequently affects the ciliary activity of the mucus-lined tube. The nasopharyngeal orifice is opened by the action of the tensor palatine muscle, and failure of this action leads to eustachian tube obstruction.

Bluestone81 has demonstrated that increased middle-ear secretions occur if the viscosity of the secretions is high. Interruption of this cycle at an early age by the installation of a ventilating grommet may alter this viscosity and break this vicious cycle concomitant with early palatal closure. This can prevent recurring otitis media, subsequent middle-ear infections, and long-term hearing loss.82

The prophylactic insertion of ventilating grommets at the time of the closure of the cleft palate not only saves the patient an anesthetic, but also significantly decreases the chances of recurrent middle-ear infection and subsequent long-term hearing loss. This also allows for better speech and language development at this early, critical developmental age.

DISCUSSION

On the basis of our own long-term studies in this area2,33,34,72 and a comprehensive review of the literature, we have created a goal-oriented, pragmatic approach for the management of the cleft palate patient that has evolved over the past 15 years. The ultimate goals in each cleft palate child are normal speech, maxillofacial growth, and hearing.

An early, two-stage palate repair is advocated in the management of patients with cleft lip and palate (Table I). The recommended sequence involves closure of the soft palate at 3 to 6 months of age, with secondary closure of the residual hard palate at 15 to 18 months of age. This sequence takes advantage of the early physiology and growth that occurs in the soft palate, which is vital in the development of speech. Furthermore, it avoids the potential pitfalls of growth disturbance related to early periosteal undermining of palatal and vomerine tissue. This repair sequence also provides total palatal closure before connected speech evolves.83 If the soft palate is repaired at the
time of lip repair, an additional anesthetic is avoided.

The complete release of the levator mechanism from the palatal aponeurosis extending laterally beyond the insertion of the tensor tympani tendon and the stripping away of all tethering muscle fibers from both the lining and the posterior margin of the bone are essential. This extensive release of the muscle allows retrodisplacement within a lining of oral and nasal mucosa. This sequence results in the development of normal speech in approximately 75 percent of patients. Another 10 percent develop mobile but short palates, whereas the remaining 15 percent have neurogenic palates. Those children with mobile but short palates are considered treatment failures; however, neurogenic palates are thought to be a consequence of the clefting rather than a failure of technique. A similar subgroup of children with neurogenic palates has been identified in a group of children requiring pharyngeal flaps after one-stage, pushback palatal repair. From these observations, we think that a near-ideal palatal closure would result in normal speech in approximately 85 percent of patients.

In an effort to identify shortfalls of this procedure, children with short, mobile velas were identified, and their maxillary arch configurations were evaluated. In this study, there was a direct correlation between incompetent speech and the magnitude of the cleft. This correlation pertains to the magnitude of the clefting in the secondary palate and relates specifically to the occurrence of bilaterality. A paucity of lining is thought to restrict the development of normal velar length. From these observations, our treatment sequence has evolved. The cleft lip and the soft palate are closed at 3 to 6 months of age. We realize that the lip repair can cause maxillary retrusion; however, facial retrusion from lip surgery can occur, regardless of the age of the patient at surgery. With reconstruction of the anterior palatal arch through the cleft lip repair and posterior maxillary arches through the soft palate repair, there is a molding effect, with alveolar arch alignment. We perform an intravelar veloplasty with release and retropositioning of the levator veli palatini muscles. As previously described, all surfaces, including the nasal mucosa and mucoperiosteum, remain intact, and there are no raw surfaces. We avoid any mucoperiosteal undermining at this age.

A major benefit of the simultaneous closure of the lip and soft palate at an early age is that it narrows the hard palate gap so that less extensive hard palate surgery is required later. Dental casts are made at the time of the repair of the cleft lip and soft palate and again at the time of repair of the hard palate. The patient is monitored at 3-month intervals to assess the narrowing of the hard palate gap. The hard palate is repaired at 15 to 18 months of age. Because the width of the hard palate cleft diminishes significantly, a much less extensive procedure must be performed. In many cases, only paring of the medial palatal edges with elevation of enough nasal and palatal mucosa to attain a tension-free, two-layer closure is done. Lateral or anterior incisions or mucoperiosteal elevations are avoided if possible. If mucoperiosteal elevation is needed, the von Langenbeck procedure is preferred over palatal pushback procedures.

It can be argued that a two-stage approach in the isolated cleft palate necessitates a second anesthetic that can be avoided with a one-stage repair. To obtain comparable velar lengthening, this repair would need to be done before 1 year of age. Until growth has been documented through the development of both the primary and secondary dentition, with one-stage repairs before 1 year of age, it should be considered that these early, one-stage repairs place the child at greater risk for the distur-

### TABLE I

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<tr>
<th>Birth</th>
<th>3 to 6 Months</th>
<th>15 to 18 Months</th>
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<tr>
<td>Begin active orthopedic appliance for maxillary alveolar arch alignment</td>
<td>Repair of cleft lip and intravelar veloplasty (closure of anterior and posterior palatal arches)</td>
<td>Repair of residual hard palate cleft using less subperiosteal undermining</td>
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<tr>
<td>Continue possible appliance application*</td>
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* After the lip is closed, the active phase of treatment is complete, and a small obturator appliance is used to retain alignment and to provide a more normal tongue position. Keeping the tongue out of the cleft may allow further closure of the residual cleft palate.

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bance of both transverse and anteroposterior maxillary arch development. This approach is in concert with the primary goals of normal speech, retaining normal maxillofacial growth, and decreasing the incidence of otitis media and subsequent long-term hearing loss in these patients.

Rod J. Rohrich, M.D.
Department of Plastic Surgery
UT Southwestern Medical Center
5323 Harry Hines Boulevard
Dallas, Texas 75390-9132
rrohri@mednet.swmed.edu

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REFERENCES


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Self-Assessment Examination follows on page 422.
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1. THE ULTIMATE GOAL IN THE TIMING OF CLEFT PALATE SURGERY IS TO MAINTAIN SPEECH WITHOUT INTERFERING WITH MAXILLOFACIAL GROWTH.
   A) True
   B) False

2. THE ULTIMATE GOAL IN EACH CLEFT PALATE CHILD IS:
   A) Normal speech
   B) Maxillofacial growth
   C) Hearing
   D) All of the above

3. RANDOMIZED CONTROLLED STUDIES HAVE SHOWN A GREATER RISK FOR POOR SPEECH DEVELOPMENT IF THE PALATE IS CLOSED AFTER 2 YEARS OF AGE.
   A) True
   B) False

4. CONDUCTIVE HEARING LOSS IN CLEFT PALATE PATIENTS IS DUE TO DYSFUNCTION OF:
   A) Tympanic membrane
   B) Levator palatini
   C) Eustachian tube

5. LOSS OF HEARING FOR SHORT PERIODS OF TIME HAS NO EFFECT ON A CHILD'S ABILITY TO DEVELOP NORMAL SPEECH.
   A) True
   B) False

To complete the examination for CME credit, turn to page 528 for instructions and the response form.