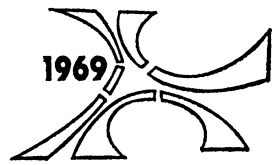
Palatal Lift Prosthesis for Treatment of Anatomic and Neurologic Palatopharyngeal Insufficiency



JUAN B. GONZALEZ, D.D.S., M.S.
ARNOLD E. ARONSON, Ph.D.
Rochester, Minnesota

Several organic conditions other than cleft palate produce palatopharyngeal insufficiency and resultant hypernasality and nasal emission. These conditions were recognized by Lermoyez in 1892 (*10*), and since then many other clinicians and investigators have also reported on this subject (*2*, *6–8*, *11*, *12*). Congenitally short soft palate, congenitally deep nasopharynx, and paralysis of the soft palate are three major examples. Hypernasality and nasal emission may also occur in varying degrees and durations in patients with normal palates who have undergone tonsillectomy and adenoidectomy (*6–11*, *14*).

In these patients, loss of intraoral pressure causes reduced intelligibility of consonant sounds. After diagnosis of palatal insufficiency, it is common to refer the patient to a speech clinician for therapy. The clinician often tries to produce better palatopharyngeal closure by training the soft palate and pharyngeal structures to function to their maximal potential. Often this potential is reached without significant reduction of the palatopharyngeal inadequacy, and the speech defect is altered only slightly or not at all.

Whereas surgical and prosthetic methods for reducing the palatopharyngeal space have been applied with success in patients with overt clefts of the hard and soft palate, or soft palate, similar techniques have not been extensively applied to the problems of congenital short palate or palatal paresis (*8*, *9*, *11*, *12*, *14*). Cartilage implants (*9*, *13*), silicone implants (*5*), and, more recently, Teflon paste implants (*15*) have been used for the correction of palatopharyngeal insufficiencies. The pharyngeal flap operation has been used also (*11*). These procedures have yielded mixed results in the treatment of congenital anatomic insufficiencies and disappointing results in the management of patients with neurologic palatopharyngeal insufficiencies.

Dr. Gonzalez is associated with the Section of Dentistry and Oral Surgery and Dr. Aronson with the Sections of Speech Pathology and Neurology, Mayo Clinic and Mayo Foundation, Rochester.

This paper was presented at the 1969 International Congress on Cleft Palate, Houston.

Gibbons and Bloomer (*8*) used a palatal lift prosthesis in a patient who had flaccid paralysis of the soft palate resulting from bulbar poliomyelitis. The prosthesis successfully elevated the soft palate and diminished the palatopharyngeal gap; there was a definite improvement in the speech.

This 5-year study represents an assessment of the use of the palatal lift prosthesis and modifications of it for the treatment of patients having congenital and acquired anatomic or neurologic palatopharyngeal insufficiencies without overt clefts.

Materials and Methods

Thirty-five patients (ages 4 to 72 years) were selected for study after oral and cineradiographic examinations demonstrated that adequate retention for the prosthesis could be attained and elucidated the residual muscular activity present in the palatopharyngeal region. The selection also was based on the cause of the deficiency and on whether surgical treatment was contraindicated because of physiologic, psychologic, or economic reasons.

Types of Patients. Nineteen patients had complete or partial paralysis of the soft palate from neurologic diseases. Ten of these had spastic paresis from upper motor neuron system damage: six from traumatic brain injuries, three from cerebral vascular accidents, and one from degenerative central nervous disease. Five had flaccid paralysis of the soft palate: three from myasthenia gravis, one from bulbar polio, and one from diphtheria. Four had combined spastic and flaccid paralysis of the soft palate from amyotrophic lateral sclerosis.

Sixteen patients had palatopharyngeal deficiencies as a result of a short soft palate or a large nasopharynx. Ten of these had what appeared to be congenitally short palate, three had short palate resulting from operations to close clefts, two had what appeared to be congenital large nasopharynx, and one had agenesis of the levator veli palatini muscle. In seven of these patients, hypernasality and nasal emission developed only after adenoidectomy and tonsillectomy; the anatomic insufficiencies had not been diagnosed before the operation.

The speech problems common to all patients were hypernasality, nasal emission, and weak consonant production with resultant decrease in intelligibility of speech. However, patients with neurologic disease had additional problems of articulation, phonation, and respiration, because of paralysis of lips, tongue, larynx, and respiratory muscles.

The patients with anatomic insufficiency had glottal stop substitutions which apparently developed as compensation for their inability to produce pressure consonants. One patient had symptoms of early stuttering. The gag reflex and nasal regurgitation were present in approximately half of the patients with neurologic disease and in all patients with anatomic insufficiency. Cineradiographic examination of some patients with paresis of the soft palate showed considerably more movement of the soft palate than would have been predicted on the basis of oral examination alone.

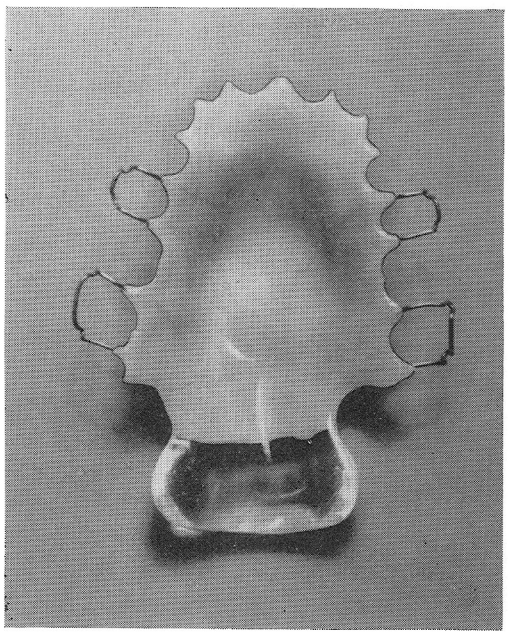


FIGURE 1. Palatal lift prosthesis.

All of the patients had had speech therapy and many had reached a plateau of achievement.

PROSTHESES. Two types of prostheses[1] were used. One was very similar in design to the one described by Gibbons and Bloomer (*8*) and was used mostly to treat patients with paresis of the palatopharyngeal region. It consisted of two parts, a retentive portion which fastened to the maxillary teeth or denture (Figure 1) and a lift portion which elevated the soft palate to narrow the palatopharyngeal space (Figure 2). Figure 3 shows the prosthesis in the mouth. Figure 4 shows cephalograms demonstrating how the palatal lift elevates the anatomically normal but physiologically insufficient soft palate in a 21-year-old woman with spasticity and palatal paresis due to traumatic brain injury.

The second type of prosthesis was used mostly for patients with anatomically short palates or large nasopharynx (Figure 5). It was similar in design to the one previously described except that the lift portion was modified by the addition of a pharyngeal piece which elevated the soft palate and also narrowed or closed the palatopharyngeal space. Figure 6 shows frames of cineradiographic films demonstrating the prosthesis in a 7-year-old girl with congenitally short palate.

In all patients, special effort was made to elevate the soft palate to the level of the palatine plane or to place the pharyngeal portion of the lift obturator just above the median tubercle of the atlas (*1*) (Figures 2 and 5).

[1] The technique of prosthesis design and construction will be discussed in a subsequent article.

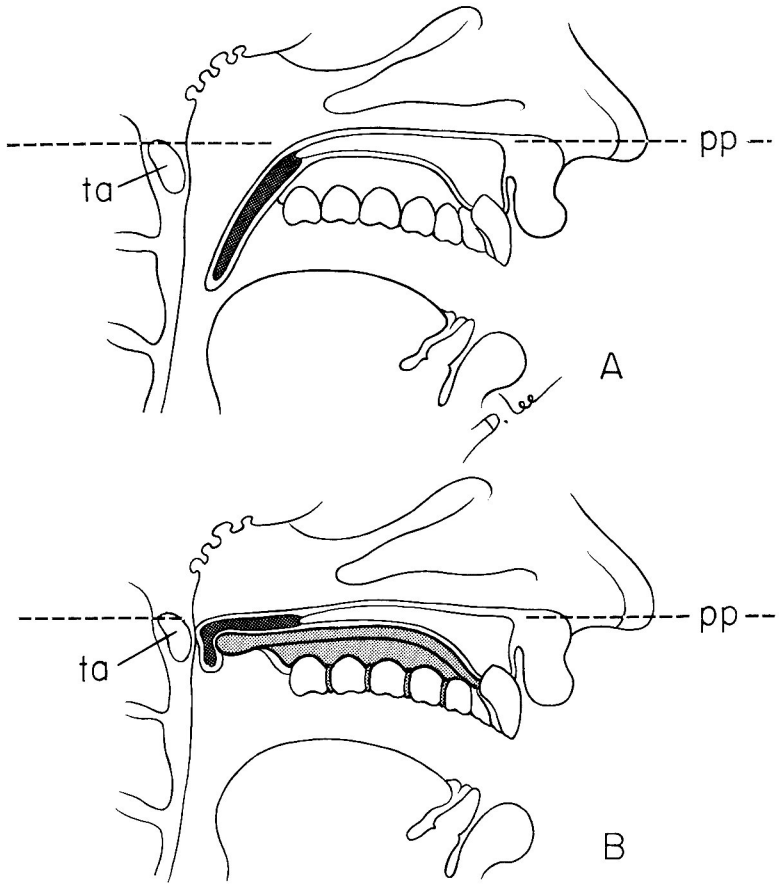


FIGURE 2. *A*, Anatomically normal but paralyzed soft palate. *B*, Palatal lift in place, elevating soft palate to produce palatopharyngeal closure (pp, palatal plane; ta, median tubercle of the atlas).

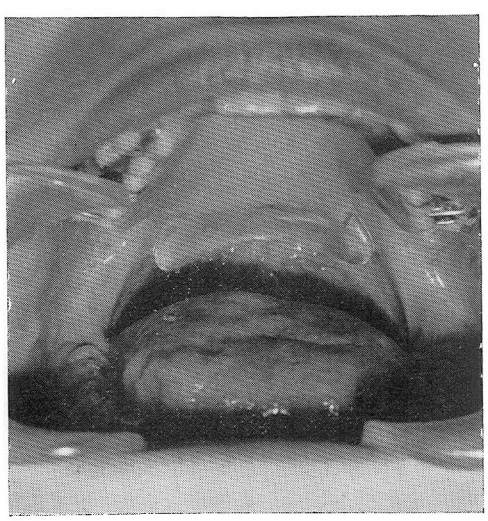


FIGURE 3. Palatal lift in position in mouth.

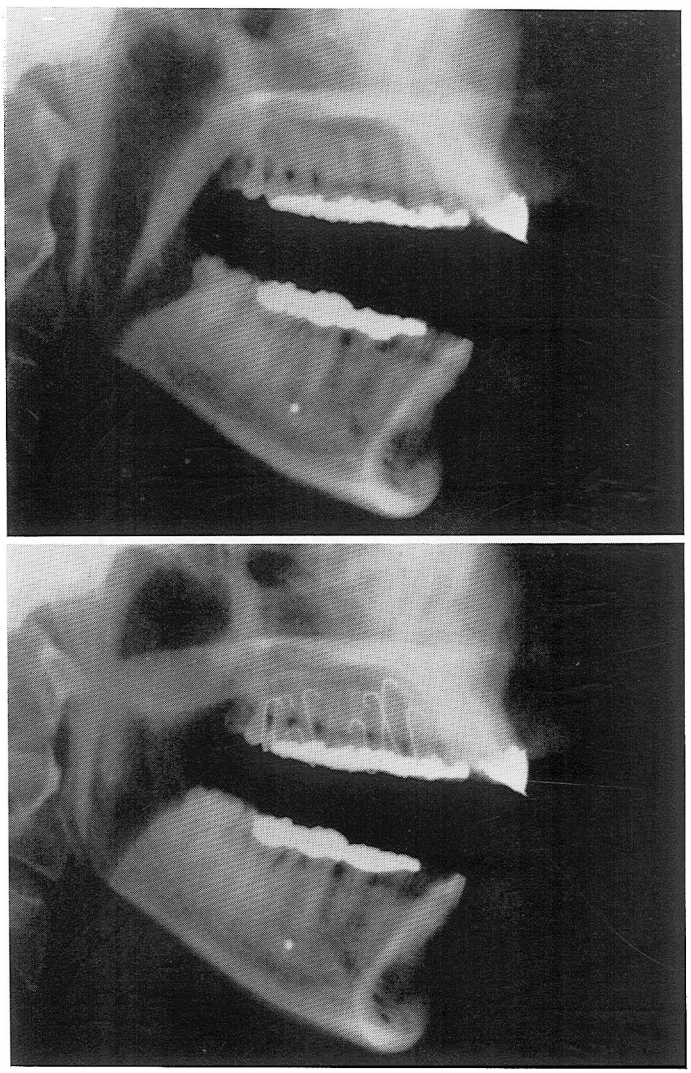


FIGURE 4. Cephalograms of patient with paralyzed soft palate saying "ah"; *upper*, without prosthesis; *lower*, with prosthesis.

METHOD OF EVALUATION. Each patient's contextual speech was tape-recorded and cephalograms and cineradiographic films were made prior to and after construction and placement of the prosthesis. Judgments of changes in the speech were made by the authors on the basis of actual clinical observations and review of the tape recordings. The changes noted were reduction of hypernasality and nasal emission and increase in overall speech intelligibility. The initial and subsequent changes were noted as: none, slight, moderate, or marked. Each patient's initial and subsequent reactions to wearing the prosthesis and the tissue changes from wearing the

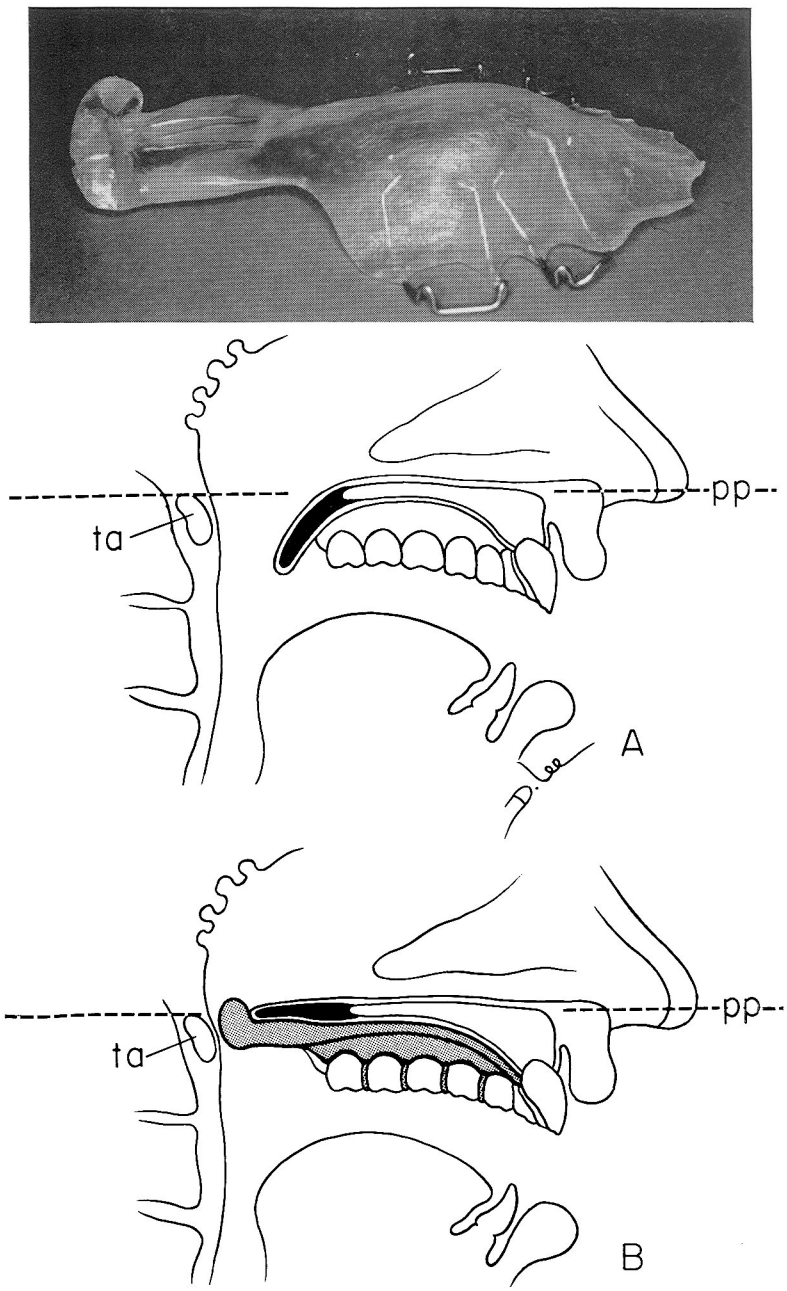


FIGURE 5. *Upper*, modified palatal lift with an obturator; *lower*, A, congenital anatomic insufficiency of palatopharyngeal region, B, palatal lift-obturator in place, elevating the soft palate and obturating the palatopharyngeal space (pp, palatal plane; ta, median tubercle of the atlas).

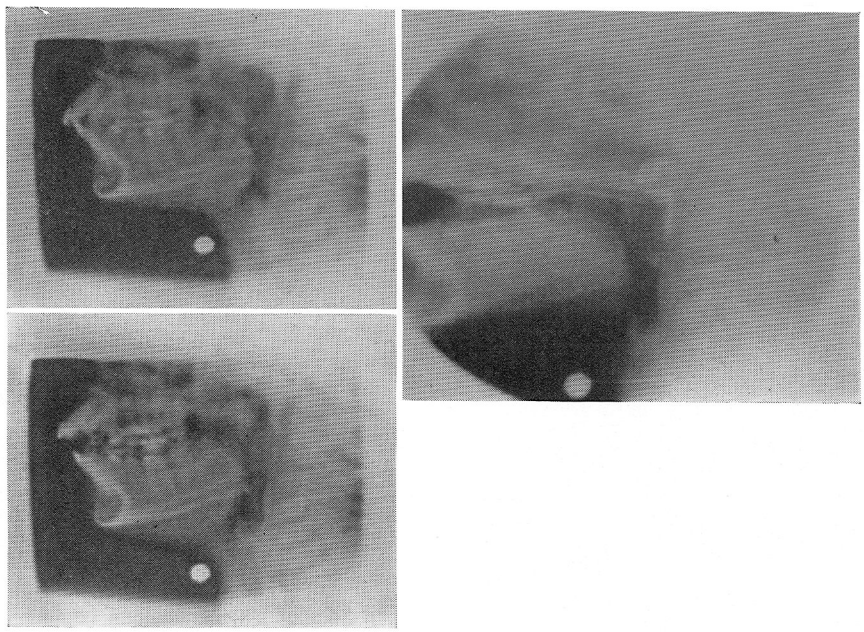


FIGURE 6. Cineradiographic frames of patient with congenitally short soft palate; *upper left,* at rest; *lower left,* in maximal movement during speech, without effecting palatopharyngeal closure; *right,* with palatal lift-obturator in place, effecting palatopharyngeal closure during speech.

prosthesis were also noted. Follow-up observations were made at 3 months and at 1 and sometimes 2 years.

Results

Patients with neurologic deficits showed moderate to marked reduction of hypernasality and nasal emission and increase in speech intelligibility immediately after the placement of the prostheses (Table 1). Closure or near-closure of the palatopharyngeal space provided additional intraoral air pressure. However, patients with severe muscular paralysis of the lips, tongue, larynx, or respiratory musculature could not utilize this potential to full advantage and, therefore, articulatory and phonatory defects remained. The articulatory problem improved somewhat once the patient became accustomed to wearing the prosthesis.

The subsequent changes noted also varied, depending on the cause of the deficiency. One patient with neurologic deficit due to trauma (N4) showed little sign of improvement in spite of the prosthesis. The patient with degenerative central nervous disease (N10) and one with amyotrophic lateral sclerosis (N17) showed progressive loss of the initial improvement until death 2 years later. Two patients with myasthenia gravis (N12 and N14) had marked improvement in their speech initially, and they wore the pros-

TABLE 1. Speech improvement in patients with neurologic palatopharyngeal insufficiency.

pt.	*cause*	*improvement**									*residual articulation defects**		
		immediate			*3 months*			*1 year*					
		hyper-nasality	*nasal emission*	*intelli-gibility*	*hyper-nasality*	*nasal emission*	*intelli-gibility*	*hyper-nasality*	*nasal emission*	*intelli-gibility*	*immediate*	*3 months*	*1 year*
N1	Trauma	+++	+++	+++	...	...	...	+++	+++	+++	++	...	0
N2†	Trauma	++	++	++	...	...	...	+++	+++	+++	+	...	+
N3†	Trauma	++	++	++	+++	+++	++	+++	+++	+++	++	+	+++
N4	Trauma	+	+	+	+	+	+	No further improvement			+++	+++	
N5	Trauma	+++	+++	++	+++	+++	++	...	...	...	++	++	...
N6	Trauma	++	++	++	+++	+++	++	...	...	...	++	+	...
N7	CVA‡	++	++	+	++	++	++	+++	+++	+++	+	+	+
N8	CVA	++	++	+	+++	+++	++	...	...	...	+++	++	...
N9	CVA	++	++	++	+++	+++	+++	...	...	...	++	+	...
N10§	Degener.	+++	+++	+++	+++	+++	++	++	++	++	+	+	++
N11	Polio	++	++	++	++	++	++	+++	+++	+++	0	0	0
N12†	MG‖	+++	+++	++	+++	+++	++	+++	+++	+++	+	+	0
N13	MG	+++	+++	+++	+++	+++	+++	+++	+++	+++	0	0	0
N14	MG	+++	+++	+++	+++	+++	+++	...	...	...	+	0	...
N15	Diphther.	++	++	+	++	++	++	+++	+++	++	+++	+++	++
N16	ALS¶	+++	+++	++	...	...	...	+++	+++	++	+	...	+
N17§	ALS	++	++	++	+++	+++	+++	+++	+++	+	+	+	+++
N18	ALS	+++	+++	++	+++	+++	++	+++	+++	++	+	+	++
N19	ALS	+++	+++	++	+++	+++	++	+++	+++	++	++	++	++

* Symbols: +++ = marked; ++ = moderate; + = slight; 0 = none.
† See Table 3 for 2-year findings.
‡ Cerebrovascular accident.
§ Patient died between 1 and 2 years after prosthesis made.
‖ Myasthenia gravis.
¶ Amyotrophic lateral sclerosis.

thesis as a supportive measure until their muscles regained complete strength and activity to effect palatopharyngeal closure. Several of the neurologic patients claimed that, when they were wearing the prosthesis, less effort was needed for speaking.

The patients with anatomic insufficiency initially showed moderate improvement in the reduction of hypernasality and nasal emission and increased speech intelligibility with the prosthesis in place (Table 2). However, residual compensatory or nonrelated articulation errors remained and required additional articulation therapy. Compensatory glottal stops (the phenomenon found in children with clefts of the palate) were frequent, as were wrinkling of the brow and pinching of the nares. The frequent occurrence of breathy voice and low volume was interpreted as the patients' way of reducing the conspicuousness of their speech. One patient stuttered, and it was suspected that this was related to her reaction to the speech defect because she was quite conscious of the nasality problem. These patients showed marked improvement in their speech.

In four patients, follow-up studies showed improved palatopharyngeal efficiency with the prosthesis removed, suggesting that better closure was perhaps partly a result of the mechanical stimulation of the musculature by the prosthesis. This phenomenon was noted approximately a year after placement of the prostheses. This phenomenon has also been observed by others (*3*, *4*) with similar types of prostheses.

Tolerance to the prostheses was favorable in all patients except in three with anatomic deficiency. These patients were treated surgically by pharyngeal flap and palatoplasty procedures. Some patients experienced irritation of the soft palate mucosa by the prosthesis at first, but this disappeared after proper adjustment of the size and shape of the lift section. The patients have been wearing the prosthesis from 3 months to 5 years.

Discussion

It has been argued that, if there is any residual palatopharyngeal movement, a palatal lift will not remain in place but will be dislodged by such movement (*8*). In spite of the active soft palates in both groups of patients, this dislodgement was not observed; the prostheses remained firmly in place during swallowing and speaking. Another objection to the prosthesis has been that pressure from it will result in inflammation of the mucosa of the soft palate and in orthodontic movement of the teeth. In no patient did the former happen after proper adjustment of the size, shape, and position of the lift or lift-obturator section. No changes in the occlusal schemes have been noticed after treatment was instituted except for changes in mixed dentition by some of the patients.

There are several advantages in the use of the palatal lift prosthesis. The device is applicable in virtually all patients with paresis of the palatopharyngeal region and in most patients with an anatomic deficiency. It seems to be most effective in the treatment of speech defects in patients

TABLE 2. Speech improvement in patients with anatomic palatopharyngeal insufficiency.

pt.	*cause*	*improvement**									*residual articulation defects**		
		immediate			*3 months*			*1 year*					
		hyper-nasality	*nasal emission*	*intelli-gibility*	*hyper-nasality*	*nasal emission*	*intelli-gibility*	*hyper-nasality*	*nasal emission*	*intelli-gibility*	*immediate*	*3 months*	*1 year*
A1	Short palate	++	++	++	Surgical correction						++		
A2	Short palate	+	+	+	++	++	++	++	++	++	+	++	++
A3†‡	Short palate	++	++	++	++	++	++	+++	+++	+++	+	+	+
A4‡	Short palate	+	+	+	++	++	++	+++	+++	+++	+++	++	+
A5†	Short palate	+++	+++	+++	+++	+++	+++	+++	+++	+++	+	+	0
A6†	Short palate	++	++	++	...	...	...	+++	+++	+++	+	...	0
A7	Short palate	+++	+++	++	+++	+++	++	Surgical correction			+++	+++	
A8	Short palate	+++	+++	++	+++	+++	++	+++	+++	+++	+++	+	+
A9†	Short palate	+++	+++	++	+++	+++	++	+++	+++	++	+++	+	+
A10	Short palate	++	++	++	+++	+++	++	+++	+++	++	++	++	+
A11§	Short palate‖	++	++	++	++	++	++	+++	+++	+++	+	+	0
A12	Short palate‖	+++	+++	++	+++	+++	++	+++	+++	++	++	+	+
A13	Short palate‖	++	++	++	++	++	++	+++	+++	+++	+	0	0
A14†‡	Large nasoph.	++	++	++	++	++	++	+++	+++	+++	+	+	0
A15†‡	Large nasoph.	++	++	++	++	++	++	+++	+++	+++	++	++	+
A16	Muscle agen.	++	++	++	+++	+++	++	...	...	...	++	+	...

* Symbols: +++ = marked; ++ = moderate; + = slight; 0 = none.

† After tonsillectomy and adenoidectomy.

‡ No further improvement after 1 year; speech same with and without prosthesis; prosthesis removed.

§ See Table 3 for 2-year findings.

‖ After operation to close cleft.

TABLE 3. Observations at 2 years.

pt.	*improvement**			*residual articulation defects**
	hypernasality	*nasal emission*	*intelligibility*	
N2	+++	+++	+++	+
N3	+++	+++	+++	0
N12	+++	+++	+++	0
A8	surgical correction			
A11	+++	+++	+++	0

* Symbols as in Table 1.

with paresis of the palatopharyngeal mechanism (this group of patients experienced fatigue less readily during speech with the prosthesis in place). Improvement in speech is almost immediate. Although there is some initial discomfort, adaptation occurs rapidly.

Three of the 35 patients could not tolerate the prosthesis or showed no improvement in their speech. Despite the presence of active gag reflexes in the majority of patients, the prosthesis in the posterior region of the mouth did not stimulate this reflex once adaptation occurred and adequate retention was provided. Initially, the presence of the prosthesis stimulated hypersalivation and had some adverse effects on the articulation. Speech therapy helped to correct these adverse effects. Articulation defects improved rapidly after adaptation to the prosthesis. However, those defects attributable to paresis of other speech structures in addition to the soft palate and compensatory to anatomic deficiencies required prolonged therapy. To facilitate adaptation, we advised that the palatal lift be worn for several minutes at a time alternating with periods of rest until it was worn for most of the day. The prosthesis was not worn overnight and the same hygienic procedures were followed as for any other dental prosthesis.

There are limitations to the use of a palatal lift. It should not be used when adequate retention for it cannot be attained, when the patient has a very spastic or stiff soft palate that does not tolerate elevation, or when the patient is uncooperative.

Our experience suggests that the palatal lift prosthesis can be used: a) to correct or considerably improve palatopharyngeal closure, b) to stimulate the pharyngeal musculature, and c) as a supportive type of prosthesis until muscles regain complete strength and activity to effect palatopharyngeal closure. This mode of treatment can be used as a temporary or a permanent means to correct hypernasality and nasal emission in speech.

Report of Illustrative Case

Myasthenia Gravis. A 17-year-old girl was first seen at the Mayo Clinic about 1 year after the onset of myasthenic symptoms. She underwent thymectomy in October 1965, and, 12 days postoperatively, had good strength in her extremities but considerable residual bulbar weakness. There was definite improvement when

0.2 ml of edrophonium (Tensilon) was given intravenously; ambenonium (Mytelase), 10 mg before meals, was prescribed by her neurologist.

Shortly thereafter, in November 1965, she was seen for speech evaluation and showed a severe bulbar dysarthria-dysphonia. She had severe hypernasality and nasal emission. Her voice showed severe breathiness. Articulatory precision was poor. On prolongation of a vowel, a voice "tremor" or irregularity of pitch was noted. After a brief period of speaking, all phonatory and articulatory aspects of speech deteriorated, the patient going from moderate intelligibility to almost complete unintelligibility. Because of the severe palatopharyngeal problem, construction and placement of a palatal lift prosthesis was tried.

The prosthesis increased the intelligibility of her speech considerably, yielded a higher rate of articulation, eliminated hypernasality and nasal emission, and, indeed, created a small degree of denasality. There was increased efficacy of breath control. Subjectively, the patient was well satisfied; one of the most important benefits for her was that, although she still tired from prolonged speaking, her subjective fatigue did not occur as rapidly and when it did she recovered from it more quickly. She was able to sustain a vowel for a longer period with the prosthesis in place. She was able to count to 100 with the prosthesis in place, but only to 50 without it, before becoming completely unintelligible and physically unable to go on. After the initial adjustment of the prosthesis, this patient did not experience any detrimental effects to the hard or soft tissue of the mouth.

Her neurologist noted some overall improvement immediately after insertion of the prosthesis. However, initially, excessive salivation was noted and was attributed to a reaction to the prosthesis. Administration of edrophonium 90 minutes after her last dose of medication produced a definite improvement in the ptosis and tongue strength, but the neurologist did not want to increase the medication until the patient had become accustomed to the prosthesis. He thought that the prosthesis had definitely helped her speech (reduced hypernasality and nasal emission and reduced severity of her dysarthria). At dismissal the prescribed regimen was 10 mg of ambenonium before each meal, and 60 mg of pyridostigmin bromide (Mestinon) between meals, and 180 mg of a sustained release form of pyridostigmin bromide (Mestinon timespan) at bedtime.

The patient was seen again in August 1966 by the neurologist, the speech pathologist, and the prosthodontist. The neurologist noted general improvement in her condition. She was still wearing the prosthesis. The soft palate remained weak (muscle weakness was judged to be grade -3 to -4)[2] with no gag reflex. The tongue was essentially normal. A mild facial weakness was noted. Otherwise her strength was good.

Comparison of tape recordings of her speech without the prosthesis made at this date and in November 1965 showed that her speech had improved in all aspects. The rate of articulatory-phonatory fatigue was reduced. During counting there was breathy dysphonia at 32 and extreme difficulty beyond 135. Without the prosthesis her oral manometer ratio was .50 (2 ounces with nares open and 4 ounces with nares closed). The low strength of expiratory musculature is, perhaps, the reason for the low values even with the nares occluded. With the prosthesis in place there was noticeable improvement in speech, mostly reduced hypernasality of vowels. She counted to 150 without showing difficulty. No adverse tissue reaction to the prosthesis was noted.

Presently her speech is excellent. She does not wear the prosthesis, although she used it for a while because she fatigued less rapidly when talking. She is still taking 30 mg of pyridostigmin bromide in the morning and 10 mg of ambenonium in the evening.

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[2] In the neurologic examination, muscle strength is graded on a four-point scale: 0, normal; -1, mild weakness; -4, severe weakness.

Summary

Thirty-five patients with palatopharyngeal insufficiency in the absence of an overt cleft of the palate were treated with a palatal lift or lift-obturator prosthesis. The insufficiency was due to complete or partial paralysis of the soft palate resulting from neurologic disease in 19 patients and to an anatomic deficiency of the palatopharyngeal region in 16. Cineradiographic films of the palatopharyngeal region and tape recordings of contextual speech were made prior to and after construction and placement of the prosthesis. In almost all the patients with paralyzed soft palates, speech intelligibility was increased immediately. However, articulatory and phonatory defects remained because of paresis of the lips, tongue, larynx, and respiratory musculature. These defects improved after a year or more of speech therapy. All patients with anatomic deficiencies showed immediate improvement in speech upon placement of the prosthesis. However, two could not tolerate it. The major improvements were reduced hypernasality and nasal emission, increased rate of articulation, and increased overall speech intelligibility. Residual, compensatory, or nonrelated articulation errors required additional speech therapy. Tissue tolerance to the prosthesis was good in all patients. The prostheses have remained firmly in place during swallowing and speech. The results of this study indicate that the palatal lift prosthesis can be used as a temporary or a permanent measure for the correction or improvement of palatopharyngeal insufficiencies, as a stimulator of the musculature involved, and as a supportive measure until muscles regain sufficient strength and activity to effect palatopharyngeal closure.

reprints: *Dr. J. B. Gonzalez*
Mayo Clinic
Rochester, Minnesota 55901

Acknowledgment. We thank the members of the Section of Neurology for their cooperation in this study.

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