Clin. Otolaryngol. 1980, 5, 61–74

REVIEW

Management of velopharyngeal incompetence in dysarthria: a historical review

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Keywords *velopharyngeal incompetence dysarthria speech symptomatology palatial lift pharyngeal flap neuromuscular treatment*

There are organic disorders other than cleft palate which produce velopharyngeal (V-P) incompetence. Some of these were discussed by investigators in the fields of speech pathology, medicine, and dentistry.[1–13] Many speech pathologists are concerned about individuals with V-P incompetence associated with dysarthria. Dysarthria will be defined for the purposes of this discussion as a speech disorder resulting from weakness, paralysis, incoordination, or alteration in the tone of the speech musculature due to central and/or peripheral nervous system impairment. The speech symptomatology of individuals with dysarthria varies depending upon the neuroanatomic type of dysarthria present.

In cases of V-P incompetence due to clefts of the velum, the speech symptomatology is dependent upon the extent of V-P inadequacy, faulty habits of articulation, and associated dental problems. In cases of V-P incompetence secondary to neurologic conditions, the speech symptomatology is dependent not only upon the extent of V-P inadequacy and the type of neurologic condition, but on the involvement of remaining structures of the speech mechanism.

Although some controversy exists in the literature with regard to the speech symptomatology exhibited by individuals with clefts of the velum, most investigators agree that faulty habits of articulation, whereby the speaker experiments with compensatory activities of the articulators to reduce hypernasality, are the chief causes of reduced intelligibility of speech.[14–22] The data as reported by these investigators suggest that there does not appear to be any particular pattern to the development of faulty articulation habits by these individuals. Rather, the habits greatly vary from one individual to another as do the resultant speech symtomatology.

Darley, Aronson & Brown[13] reported that the effect on the listener of V-P incompetence due to palatal weakness or paralysis, as may occur in several types of dysarthria, is similar to that of the varying degrees of hypernasality and associated articulation defects heard in indi-

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0307-7772/80/0200-0061 $02.00

viduals with clefts of the velum. However, the speech difficulties associated with palatal weakness are generally not due to faulty habits of articulation, but are more often the result of widespread neuromuscular impairment of the speech mechanism. Honjow, Isshiki & Kitajima[18] attempted to explain the difference in speech symptomatology between individuals with V-P incompetence due to cleft palate and those associated with paresis or paralysis by explaining that articulation difficulties associated with cleft palate are due not only to V-P incompetence, but also to the fact that the condition often exists at the time of articulatory development when the likelihood of the development of compensatory (faulty) habits of articulation is greatest. The individual with acquired V-P incompetence due to neurologic disease, on the other hand, who probably has already developed articulatory proficiency, is not likely to develop compensatory articulator adjustments even in the presence of marked hypernasality. Any speech difficulty he exhibits is dependent upon the type and extent of neuromuscular impairment of the speech mechanism and is typically characterized by varying combinations of respiratory, phonatory, resonatory, and articulatory difficulties.

The primary objectives of this paper are to delineate the adverse effects of three types of dysarthria on V-P competence, to describe the speech symptomatology commonly associated with each type, and to review the methods of prosthetic, surgical, and speech management found to be successful with each type.

Classification of dysarthrias

Some of the early dysarthria classification systems were based on neuroanatomic site of lesion, etiology, and associated disease conditions, and gave rise to several terms, including cerebellar dysarthria, bulbar nuclear dysarthria, dysarthria due to disorders of sensibility, dysarthria with severe epilepsy, and several designations of mixed dysarthria.[23–25] A more recent classification system was proposed in which 6 major types of dysarthria—flaccid, spastic, ataxic, hypokinetic, hyerkinetic, and mixed—were outlined.[13] This classification system is primarily based on what is happening to the muscles whose dysfunction causes the dysarthria. Supplementary information regarding the probable neuroanatomic site(s) of lesion responsible for producing each of the 6 types of dysarthria is also included. This 6-fold classification system of dysarthria will be used below.

V-P function in different types of dysarthria

Endoscopic, ultrasound, multiview video-fluoroscopic, and electromyographic (EMG) investigations have been performed to help define the anatomy and physiology of the V-P mechanism during speech and nonspeech acts. Dickson[26] presents a particularly useful overview of the data in this area and is an excellent reference source for the interested reader. For the purposes of this discussion, however, we will explore only those data which help clarify the motor innervation of the V-P musculature. As reported by Dickson only 2 comprehensive studies of the motor nerve supply to the V-P musculature are described in the literature.[27, 28] Each study was based on evoked EMG recordings from various muscles of the

V-P mechanism in rhesus monkeys. The rhesus monkey was studied because previous investigations showed that the basic anatomy and physiology of the V-P mechanism of this animal are similar to those of human beings.[29, 30] The data obtained by directly stimulating various cranial nerve fibers within the skull indicated that those muscles thought to be most responsible for V-P closure, i.e. the levator veli palatini, uvularis, and superior constrictor muscles, receive dual motor innervation via the facial nerve and via branches of the pharyngeal plexus derived from the glossopharyngeal and vagus nerves. This dual nerve supply and the musculature they innervate comprise the final common pathway, or lower motor neuron (LMN) system, responsible for V-P activity. Normally, this system works in harmony with other components of the LMN system which initiate muscular activity in other segments of the speech mechanism. However, before the components of the LMN system can work in harmony to induce sufficient, graded, and synchronous V-P and other speech musculature activities they must be stimulated, governed, and regulated by specific nerve tracts of the upper motor neuron (UMN) system which originate from different areas of the nervous system.

Disease of or damage to those components of the LMN and/or UMN system which regulate the activity of the V-P musculature may result in V-P incompetence, the characteristics of which will vary depending upon which system is disturbed and the extent of its impairment.

SPASTIC DYSARTHRIA

Bilateral lesions of corticobulbar and selected extrapyramidal fibers of the UMN system produce spastic paralysis of the speech musculature, sometimes referred to as pseudobulbar palsy. Spastic muscles are generally stiff, move slowly through a limited range, and tend to be weak. The effects of such muscular impairments on speech vary depending upon which muscles of the speech mechanism are affected. However, the impaired speech of the individual with pseudobulbar palsy is known as spastic dysarthria. Spastic dysarthria is generally characterized by reduction, weakening, or loss of articulatory, phonatory, and/or resonatory control, accompanied by spasticity and hyperreflexia of the affected musculature.[1, 11–13, 31] Several muscle groups of the speech mechanism are typically involved. When the lingual and labial muscles are involved, articulation is slow, dragging, and indistinct, resulting in markedly reduced alternate motion rates (AMR) and impaired consonant production skills. Involvement of the laryngeal mechanism is a common occurrence in pseudobulbar palsy, resulting in hoarse and weak phonation accompanied by monopitch, monoloudness, reduced stress, and/or intermittent periods of strained-strangled voice. Because the V-P mechanism is richly innervated, it is highly susceptible to neuromuscular impairment. V-P incompetence and resultant hypernasality is second to imprecise articulatory skills as the most prominent speech symptom associated with pseudobulbar palsy. Nasal snorting, however, is not a common feature of this disorder.[11–13]

Simultaneous recordings of intraoral air pressure, rate of nasal air flow, and the speech signal to demonstrate 5 different patterns of V-P dysfunction exhibited by 5 children with dysarthria secondary to spastic, athetoid, or mixed cerebral palsy were performed.[7] Two of the children were classified as spastic cerebral palsied. One exhibited a gradual opening and the

other exhibited a gradual closing V-P phenomenon during AMR tasks. The gradual opening condition was characterized by good V-P closure during the initial segments of the AMR tasks, followed by a gradual rise in nasal air flow, a drop in intraoral air pressure, and marked hypernasal resonance in later segments of the speech tasks. The converse condition was characterized by a gradual closing of the V-P port and a reduction of hypernasal resonance as the AMR segments continued over time.

FLACCID DYSARTHRIA

Unilateral or bilateral lesions of the motor units of the cranial nerves produce flaccid paralysis of the speech musculature, sometimes referred to as bulbar palsy. Flaccid muscles are weak and flabby; in severe cases, muscles are paralyzed. The effects of such muscular impairments on speech vary depending upon which and how many motor units are impaired. The term applied to the speech of the individual with bulbar palsy is flaccid dysarthria. Flaccid dysarthria is characterized by impairment of all types of movement of the affected speech musculature—voluntary, automatic, and reflexive. The muscles of resonation, articulation, and/or phonation may be involved in the flaccid paralysis and such involvement may occur unilaterally or bilaterally. Unlike pseudobulbar palsy, which affects overall movement patterns of several different speech musculature groups, bulbar palsy is typified by selective impairment of individual muscles of the speech mechanism and their specific functions. The 3 most deviant dimensions of speech associated with bulbar palsy are hypernasality, imprecise consonant production, and breathy voice, in that order.[11-13] Hypernasality in bulbar palsy is often due to V-P incompetence secondary to a lesion of the pharyngeal plexus. Imprecision in tongue consonant production is often due to a lesion of the hypoglossal nerve. Breathy voice in bulbar palsy is due to hypoadduction of the vocal folds and is ordinarily a manifestation of impairment of the recurrent laryngeal branches of the vagus nerve. It should be noted that isolated disturbances of a single cranial nerve (mononeuropathy) occasionally occur in bulbar palsy producing speech signs specific to that cranial nerve.

MIXED DYSARTHRIA

The above discussions have presented neurologic conditions with associated pure types of dysarthria, that is, disorders of motor speech due to single motor system disease. Mixed dysarthria results from impairment of more than one motor system. Individuals with this condition display a mixture of speech symptoms depending upon the motor systems involved. Three common types of neurologic disease associated with mixed dysarthria are amyotrophic lateral sclerosis (ALS), multiple sclerosis (MS), and Wilson's disease. This section will describe only the mixed dysarthria of ALS since the mixed dysarthria of the other two diseases infrequently involves the V-P mechanism.

ALS involves progressive, diffuse degeneration of UMNs and LMNs. As corticobulbar and extrapyramidal fibres and specific cranial nerve motor units become involved in the degenerative process, a variable combination of spastic and flaccid dysarthria, or mixed

dysarthria, occurs. The 3 most common deviant speech dimensions in ALS are imprecise consonant production, hypernasality, and harsh voice, in that order.[11–13] Because ALS is progressive, these speech problems become increasingly severe throughout the course of the disease resulting in unintelligibility in advanced cases. The prognosis for life is averaged at 5 years post onset; however, it has been reported that some individuals live for as long as 20 years with ALS.

Methods of prosthetic, surgical, and/or speech rehabilitation of V-P incompetence in dysarthria

PROSTHETIC MANAGEMENT

Gibbons & Bloomer[2] were the first to describe the successful use of a palatal lift type (Figure 1) of prosthesis in facilitating V-P closure in a patient with flaccid dysarthria secondary to bulbospinal poliomyelitis. Since then several investigations have tested the efficacy of prosthetic management of V-P dysfunction in individuals with different types of dysarthria.

A programme of prosthetic treatment of V-P dysfunction in 11 children with cerebral palsy was performed.[8] Ten children studied were fitted with a specially designed palatal lift, and one was fitted with a bulb-type obturator. Although prosthetic management of their palatal paresis resulted in significant improvement in the speech of the children, the investigators concluded that prosthetic management would be of minimal clinical assistance to the neuro-

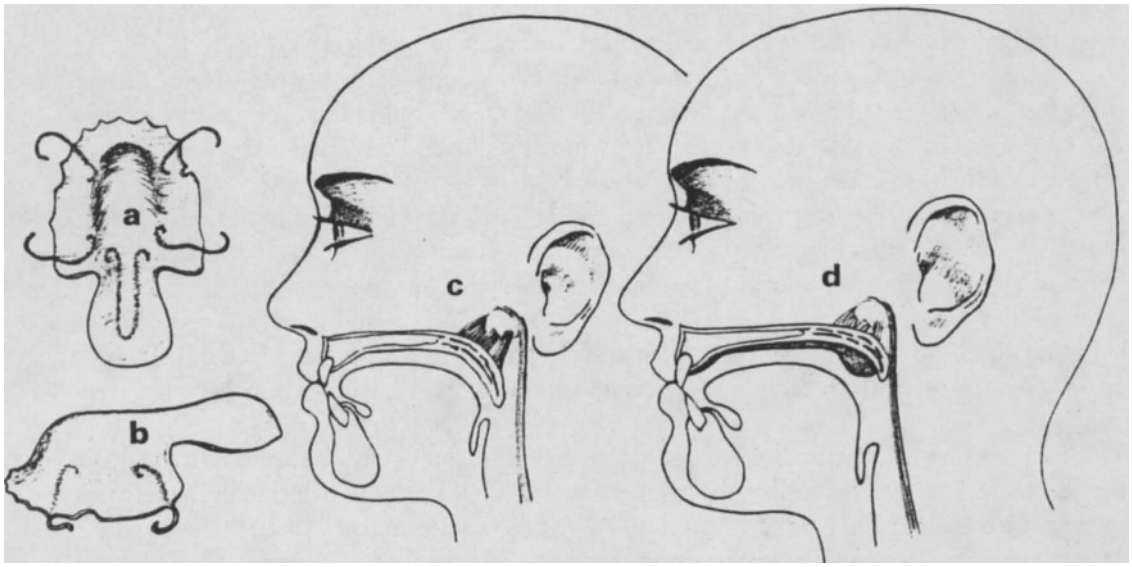

Figure 1 A palatal lift device. a Superior view of the palatal lift. b Lateral view of the lift. c Velopharyngeal incompetence without the lift. d Improvement in velopharyngeal function as a result of the lift.

muscularly handicapped child who had weakness of the tongue, lip, and/or jaw musculature associated with V-P incompetence.

Treatment of V-P incompetence through the use of a 'speech-aid prosthesis' (palatal lift) was reported on 6 patients with neurogenic V-P incompetence all with clinically anatomically normal soft palate structures, who improved in speech intelligibility following prosthetic management.[32] Stimulation of increased movement of the posterior and lateral pharyngeal walls by the prosthesis was noted. These authors concluded that 'if stimulation of additional muscle movement can be obtained with the palatal lift prosthesis and its modification, its use may be indicated to achieve maximum pharyngeal wall movement prior to secondary surgery' (p. 475).

Certain anatomic and physiologic considerations concerning the V-P mechanism for the proper construction of a palatal lift for individuals with V-P incompetence secondary to palatal weakness were reported.[10] Findings indicated that the major anatomic change that occurs with placement of the palatal lift is the reduction in size of the V-P orifice. The important physiologic effects of the palatal lift are an overall reduction in the amount of nasal air flow and a quicker buildup and release of intraoral air pressure during speech.

Success in the management of V-P incompetence in 19 patients with different types of dysarthria (10 spastic, 5 flaccid, and 4 mixed) using a palatal lift was reported.[9] The speech symptomatology common to all patients were hypernasality, nasal emission of air, and weak consonant production with resultant decrease in intelligibility of speech. With the exception of 2 spastic and one mixed dysarthric patients, all patients showed moderate to marked reduction of hypernasality and nasal emission and an increase in speech intelligibility immediately after insertion of the prosthesis. However, patients with severe neuromuscular involvement of other structures of the speech mechanism, such as the lips, tongue, larynx, and/or respiratory musculature, still exhibited residual articulatory and phonatory defects.

A prosthetic management programme of an edentulous patient with isolated palatal paresis of undetermined origin was described.[33] The speech symptomatology of this patient was characterized as moderate to severe hypernasality during conversational speech accompanied by audible nasal emission of air and inconsistent articulation errors. Lateral head X-rays taken during prolongation of vowels and /s/, and cinefluorography during extended speech samples revealed that there was consistent and substantial V-P opening throughout all types of speech efforts. A palatal lift attached to the posterior aspect of an upper denture resulted in a dramatic improvement in the patient's articulation and a marked reduction in hypernasality. Follow-up cinefluorographic analysis with the prosthesis in place showed more upward movement of the palate during speech than was achieved prior to prosthetic intervention.

Success using a palatal lift for a patient with V-P incompetence secondary to multiple cranial nerve damage was reported.[34] Speech was unintelligible and decidedly hypernasal, accompanied by audible nasal air escape. The velum was described as extremely long and flaccid with no movement during phonation or gagging. However, during sustained production of /s/ the patient was capable of exciting limited velar movement, as revealed by lateral radiographs. When a palatal lift was fitted, articulation was judged to be significantly more

intelligible, particularly after the patient had used the prosthesis for more than 2 months. Improvements in resonance balance and nasal air escape were noted with the prosthesis in place.

Success in improving the speech intelligibility of two patients with palatal paralysis secondary to severe bulbar palsy using a palatal lift was reported.[35] The speech symptomatology of these patients prior to prosthesis insertion was described as severe hypernasality, nasal air emission, severe articulatory impairment, and severely diminished vocal volume. With prosthesis in place, each patient exhibited marked improvement in articulatory proficiency and overall speech intelligibility, although residual dysarthric features were still present. Only one patient experienced an appreciable reduction in hypernasality using the prosthesis.

Reduced hypernasality and improved speech intelligibility using a palatal lift in a 7-year-old boy with V-P incompetence of unknown etiology was reported.[36] Speech therapy prior to the use of the palatal lift had produced no improvement in speech.

The assets and liabilities of palatal elevation and/or palatopharyngeal prosthodontic stimulation for individuals with V-P incompetence secondary to neurologic disease were reviewed.[37] It was determined that if the neurologic disorder is localized to the V-P region and the patient has minimal or no difficulty with articulation, prosthetic treatment is optimal; patients who exhibit additional involvement of the tongue, lips, or larynx usually respond less favorably to prosthetic therapy. Five basic objectives in making the palatal lift and/or pharyngeal section prostheses for individuals with V-P incompetence secondary to neurologic disease were listed. The most significant were 1 to reduce hypernasality and nasal escape of air by palatal elevation, 2 to increase V-P function by constant and continuous stimulation, and 3 to increase neuromuscular response by gentle stimulation and speech exercises.

One of us (D.F.J.) has observed increased muscular motility after the fitting and wearing of a palatal lift. Observations of this phenomenon were made objectively. Frontal cineradiographic views of a patient at rest and during phonation were studied and measured on a 0- (no medial movement) to 5-point (maximal medial movement) equal interval psychophysical rating scale of lateral pharyngeal wall displacement, in the same manner as reported earlier.[38] These measurements were made both before the patient was fitted with a prosthesis and after a period of at least three months of wearing it. Little to no improved movement in the soft palate was noted as a result of wearing the prosthesis, as observed on lateral cineradiographic views. However, significant improvement in mesial movement of the lateral pharyngeal walls has been noted. For example, patients with 0–1 movement pre-fitting, increased to ratings of 3–4 lateral pharyngeal wall movement after wearing the palatal lift.

The major contra-indications to the use of a prosthesis in the management of V-P incompetence in individuals with dysarthria, as indicated by several of the studies reviewed, are a inadequate retention, b a hyperactive gag reflex not responsive to desensitization or specific appliance modification, c a very spastic or stiff soft palate that does not tolerate elevation, and/or d extreme lack of cooperation on the part of the patient.

SURGICAL MANAGEMENT

While paresis, paralysis, and incoordination of the V-P mechanism has been recognized for

many years, until recently this condition has received little attention in the surgical literature. The surgical results on a series of nine patients with V-P incompetence due to bulbar palsy were reported.[3] The surgical preference was for an inferiorally based posterior pharyngeal flap. Following this surgery all patients demonstrated significant improvement in articulation and diminished or abolished hypernasality.

Three of 6 children with palatal paresis secondary to cerebral palsy made sufficient gains in speech intelligibility following pharyngeal flap surgery.[8,39] Postoperative failure in 3 cases was attributed to lack of intrinsic motivation for self-improvement in each case. The conclusions drawn were that in children with cerebral palsy prosthetic management of palatal paresis may insure greater success in the rehabilitation of speech than surgical intervention.

The effects of a Teflon and glycerin mixture injected along the area of 'Passavant's line' in one patient with neurogenic V-P incompetence were studied.[40] Improved speech resulted from this implantation technique. Teflon injections into the nasopharynx in 12 patients with V-P incompetence were performed.[41] Consistent improvement in resonance were obtained. These results indicated that Teflon is an excellent implant material for the correction of velopharyngeal insufficiency in selected cases. However, it appears that none of the 12 patients studied collectively had 'neurogenic' V-P incompetence since the criteria for patient selection were such that 'patients with hypernasal speech but with good levator activity—patients with poorly defined or erratic levator action—were excluded from this treatment' (p. 20).

The implantation or injection of other alloplasts including silicone rubber and silastic pillows has been reported. Implantation of fascia, bone, and cartilage into the posterior pharyngeal wall has also received some attention in the literature. Presently, there are many techniques for secondary correction of V-P incompetence and the reader is referred to Yules[42] for a review of various surgical procedures, pharyngeal wall implants, dental prostheses and speech therapy methodologies used in the management of V-P insufficiency associated with an anatomically deficient mechanism.

For surgical correction of V-P incompetence the posterior pharyngeal flap procedure appears to be the most popular approach up to this time. While there has been a considerable amount of conflicting information as to the manner in which the flap functions (dynamic vs. static) and the type of flap to be used (inferiorally based vs. superiorally based, for example) the literature has, for the most part, been focused on secondary procedures used in the cleft palate population. A resurgence of interest in surgical correction of neurogenic V-P incompetence is evidenced by several recent reports.[43–46]

Seventy patients were studied, 42 with V-P incompetence due to causes other than cleft palate.[43] Of these, 15 patients had 'idiopathic V-P insufficiency' associated with neurological impairment due to central nervous system dysfunction 'ranging from severe cerebral palsy to minimal brain dysfunction' (p. 352). Eight of these 15 patients received a broad, superiorally based pharyngeal flap which included the entire posterior wall of the pharynx. Either improved or acceptable resonance for all 8 patients following this surgical procedure were reported.

Forty-one patients with submucous clefts of the palate and 32 cases with 'occult' submucous cleft were studied.[44] Five 'occult' cases had palatal paresis due to primary neuromuscular disease. Surgical procedures included levator muscle sling construction, palate push-back, and

pharyngeal flap. Excellent speech results were obtained *except* with patients having palatal paresis.

Ninety-eight patients with V-P incompetence without overt cleft were studied.[45] Of these, 19 were diagnosed as having 'palatal paresis'. Surgery was performed on 9 patients, ranging in age from 4 to 60 years, all of whom had had speech therapy prior to surgery without significant improvement. The surgical procedure most frequently used was a palatal push-back combined with a superiorally based pharyngeal flap. Two of the 9 patients had implants, one a silicone rubber pillow and the other a cartilage implant behind the posterior pharyngeal wall with reported minimal benefits. Results of surgical management were disappointing regardless of the methods used. The conclusions drawn were a the combination of palatal push-back and superiorally based pharyngeal flap is inappropriate for patients with neurogenic V-P incompetence, b palatal lifts and obturators may be the treatment of choice, at least in patients with cerebral palsy, and c patients with palatal paresis need obturation, and that good results may be achieved by using, wide, lined pharyngeal flaps or a 'lateral port control' pharyngeal flap.

Several investigators have discussed the level of attachment of the pharyngeal flap as it related to speech results in individuals with V-P incompetence and have stressed the importance of maintaining a superior-posterior vector motion of the velum as it relates to the base of the flap on the posterior pharyngeal wall.[47,48] These investigators maintain that a 'low based' pharyngeal flap produces traction in an inferior direction and thus 'tethers' the velum. A significant number of patients with V-P incompetence following pharyngeal flap surgery have been reported.[4,49] Results of these studies revealed that the superiorally based pharyngeal flap provided far superior postoperative speech results as compared to the inferiorally based flap.

The role of the lateral pharyngeal walls in pharyngeal flap surgery has also been the subject of a number of studies.[50–52] Results of these studies indicated that mesial movement of the lateral walls is essential for V-P closure, since the flap can serve only as a vertical obturator and cannot close the lateral ports around the flap.

Surgical procedures performed on a series of young adult patients with acquired neurogenic V-P incompetence, many of whom had previously undergone unsuccessful management regimes including combinations of speech therapy, palatal massage, muscle retraining programmes, and the wearing of palatal-lift prostheses were reported.[46,53] Each patient was comprehensively studied and underwent preoperative neurologic, speech, multi-view cineradiographic, panendoscopic, manometric, and surgical evaluations. The motility of each lateral pharyngeal wall, as determined by some of these measures, was rated on an equal-appearing interval scale at rest and during sustained phonation of vowels, fricatives, and affricatives and during connected speech consisting of test phrases. Subsequently, a solution of 4% xylocaine was topically sprayed in the pharyngeal area. The patient was requested to sustain the vowel /a/ as the most medial excursion of each lateral pharyngeal wall was located and marked at the level of the palatal plane on the oral side. The methods of marking these landmarks included surgical weck clips, silver nitrate sticks, methylene blue marking pens, and disposable cauterizing units. A third marking was made in the midline at or above the level of the palatal plane on the nasal side; above the tubercle of the atlas and approaching the level of

the clivus. The lateral markings denote the desired area for the lateral incisions of the pharyngeal flap and the midline marking denotes the desired level (height) for the base of the flap. After the marking was complete, the patient was requested to sustain /a/ again so that the landmarks and lateral markings of the outlined flap could be visualized per-orally. Caution against marking the flap after the patient is supine and a general anesthesia has been administered was emphasized, since under this condition the V-P musculature is flaccid, the lumen of the V-P area is narrowed medially, and the anterior-posterior distance between the velum and posterior pharyngeal wall is reduced. It was further warned that the surgeon might well make a much narrower flap with a lower base if the desired landmarks have not been marked and the flap outlined while the patient is awake in the seated position, responsive, and, most importantly, phonating.

The surgical management of these patients was accomplished by a tailor-made superiorally based posterior pharyngeal flap. In selected cases, catheters were introduced transnasally to maintain lateral port apertures on either side of the flap in the manner described by Hogan.[54]

While data are still being collected on the efficacy of the surgical procedure outlined above, these authors report improved speech intelligibility and reduced hypernasality in patients treated thus far.[46]

SPEECH REHABILITATION

The goal of speech therapy for individuals with V-P incompetence, regardless of the underlying pathophysiological mechanism, is to induce greater V-P activity in order to reduce hypernasal resonance and nasal air emission and increase speech intelligibility. Although most of the data available concerning the effects of speech therapy on V-P dysfunction were collected from individuals with anatomically deficient palates, certain inferences may be drawn with regard to the efficacy of speech therapy in individuals with neuromuscularly impaired palates. Several therapeutic techniques have been proposed for improving V-P functioning and speech, including blowing, sucking, gagging, pushing, muscle stimulation, and articulation exercises. These exercises were designed to induce more effective voluntary control of the V-P musculature as well as increase its strength, tone, bulk, and mobility. While some investigators have reported varying degrees of success in eliminating or reducing hypernasal resonance and associated speech symptomatology using one or more of these exercise techniques,[1,55-59] others have reported that these techniques do not bring about significant improvement in V-P functioning.[60-64] In view of the conflicting evidence, the speech clinician who uses such exercises should guard his prognosis, especially if he is working with an individual whose neurologic disorder may involve more than just the V-P component of the speech mechanism.

Despite the fact that the three types of dysarthria known to have an adverse effect on the V-P musculature, i.e. spastic, flaccid, and mixed, present distinctive clusters of speech symptomatology, it has been suggested that they can be approached in the speech rehabilitation programme as if they have much in common (p. 272).[13] Our clinical experience supports the above contention that it is legitimate to make certain generalizations when applying a traditional speech therapy format for the improvement of articulation, phonation, and/or reson-

ance difficulties in individuals with different types of dysarthria. However, we also feel that generalizations cannot as easily be made when applying certain neuromuscular facilitation techniques for the improvement of V-P dysfunction associated with different types of dysarthria. For example, whereas facilitation techniques such as the application of pressure, icing, brushing, stroking, and vibration to the velar musculature may be occasionally successful in eliciting and developing hidden potentials of the weak V-P mechanism in individuals with flaccid dysarthria,[1,55,65] inhibition techniques such as prolonged icing, pressure to muscle insertion points, slow and irregular stroking and brushing, and desensitization of hyperreflexias of the velar musculature may be prescribed to help bring about more cooperative agonistic-antagonistic V-P muscle activity in individuals with spastic dysarthria.[66–70] The neuromuscular treatment approach for the individual with V-P incompetence associated with mixed dysarthria may vary in accordance with whether such incompetence is characterized by a predominance of flaccid or spastic V-P muscle activity.

If the neuromuscular treatment regimen does not result in significant reductions in V-P incompetence and hypernasality after a period of 2 months of intensive therapy, it is unlikely that continuing the programme will be of further help. However, even if significant mitigation of V-P incompetence is accomplished through neuromuscular training, the individual may still require prosthodontic and/or surgical intervention to establish a more fully functioning V-P mechanism. Further, since the rehabilitative measures may be of some assistance in effectuating only more adequate V-P functioning, any associated speech symptomatology that existed prior to these management procedures will probably require continued attention by the speech clinician. The individual whose neurologic condition severely affects the V-P as well as other components of the speech mechanism may never be able to develop the delicately coordinated movements of the speech musculature necessary for normal speech production.

Discussion

V-P incompetence has long been recognized by practitioners in the field of medicine, dentistry, and speech pathology as a serious condition which must be fully investigated so as to discover ways to remediate or prevent its disabling effects on speech and vegetative activities. Unfortunately, as we examine the literature in this area we discover that there are limited data available regarding this condition and its treatment in individuals with dysarthria.

It is not clear why greater strides towards an understanding of V-P incompetence in dysarthria have not been made. It would be difficult to argue that individuals with this condition cannot be helped. For it has been established by investigators reviewed above that the correction of V-P incompetence due to dysarthria is attainable. Firstly, prosthetic management has been shown to be quite an effective procedure in the treatment of V-P incompetence in certain individuals with dysarthria. However, much more research is needed in this area to substantiate present findings and perhaps to refine the techniques of design and construction of various types of prostheses. Secondly, although the few steps that have been taken to determine the effectiveness of pharyngeal flap surgery for individuals with palatal dysarthria have led to mixed results, additional, long-term studies of a sufficiently large number and variety of individuals with neurogenic V-P incompetence must be performed before such a procedure is

unequivocally adopted or abandoned. Furthermore, there have been only a few attempts reported on the possible benefits of other surgical procedures, such as Teflon injection or pharyngeal muscle implants for the improvement of V-P incompetence in dysarthria. Lastly, the shortage of investigations of V-P incompetence in dysarthria by investigators in speech pathology is perhaps the greatest enigma of all, since the speech clinician is invariably called upon to design and implement rehabilitative programmes for dysarthric individuals. The question arises then as to how the speech clinician can design differential therapeutic programme for the improvement of V-P incompetence in individuals with dysarthria if there are few or no data to support the usefulness of certain traditional therapeutic and neuromuscular facilitative techniques for the rehabilitation of V-P incompetence in individuals with different types of dysarthria.

In conclusion, V-P incompetence is a prominent condition in the individual with spastic, flaccid, or mixed dysarthria. As in the successful management of the individual with an anatomically deficient V-P mechanism, a multidisciplinary approach may be the best procedure for the treatment of V-P incompetence in the individual with a neuromuscularly impaired V-P mechanism. However, until further research is conducted in the areas of prosthetic, surgical, and speech rehabilitation for V-P incompetence in individuals with dysarthria, the most beneficial treatment programme for this population will remain ill defined.

Acknowledgements

The authors wish to thank Drs Frederic L. Darley and Arnold E. Aronson for their editorial comments during the preparation of this manuscript. Requests for reprints should be addressed to James P. Dworkin, Program in Speech Pathology, 224 Taylor Education Building, University of Kentucky, Lexington, Kentucky 40506.

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