The Levator Veli Palatini Muscle in Cleft Palate Anatomy and Its Implications for Assessing Velopharyngeal Function: A Literature Review

Seunghee Ha*

Department of Audiology & Speech Pathology, University of Tennessee at Knoxville

Background & Objectives: The levator veli palatini (LVP) muscle is most commonly reported as the most abnormal muscle in the population with cleft palate and the muscle plays a significant role in velar elevation, and therefore speech production. ***Methods:*** This article reviewed the literature focusing on anatomic aspects of the LVP muscle in normal and cleft palate populations and addressed the significant implication of the studies in clinical services for individuals with cleft palate. ***Results & Conclusion:*** Literature addressed that abnormal location and volume of the LVP muscle is closely related to velopharyngeal insufficiency and a complete evaluation of the LVP muscle along with the velopharyngeal mechanism is required to provide the most appropriate and effective treatments to individuals with cleft palate. Continuous improvements of MRI technique lead to effective identification of LVP muscle structures and better clinical decision-making for surgical procedures and speech therapy in individuals with cleft palate. (***Korean Journal of Communication Disorders*** 2007;12;77-89)

Key Words: levator veli palatini muscle, anatomic aspects, cleft palate, MRI

Ⅰ. Introduction

In individuals with repaired cleft palate, it is estimated that 10 % to 25 % have residual velopharyngeal insufficiency (Bradley, 1997; Marrinan, LaBrie & Mulliken, 1998; Witt & Marsh, 1997). Individuals with residual velo-pharyngeal insufficiency show persistent nasalized speech and abnormal articulation patterns. As a result, secondary surgical management or speech therapy is necessary to improve the speech problems. There are several possible factors to account for surgical failure and residual velopharyngeal insufficiency. Gross anatomic abnormalities and severity of the cleft in patients are closely related to surgical failure. The abnormal velopharyngeal muscle structures which remain even after primary palatoplasty may be another significant factor. In particular, the levator veli palatini (LVP) muscle plays a significant role in velar elevation and it is

Received January 19, 2007; final revision received February 20, 2007; accepted February 22, 2007.
* Correspondence to Seunghee Ha, PhD, Department of Audiology & Speech Pathology, University of Tennessee at Knoxville, USA, e-mail: shha610@gmail.com, tel.: +1 865 974 4802

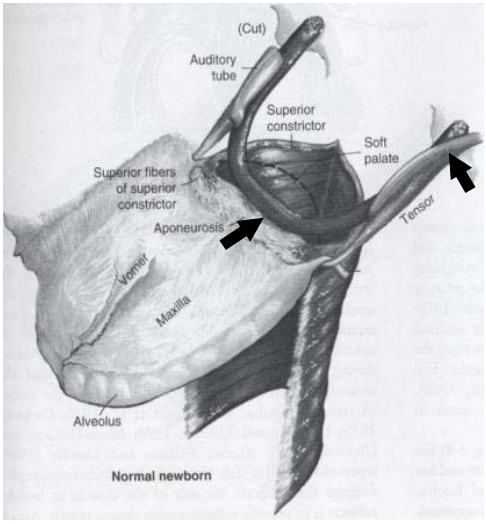
most commonly reported as the most abnormal muscle in the population with cleft palate. Therefore, the LVP muscle is of great clinical and research interest. An understanding of anatomic and physiologic characteristics of the LVP muscle would contribute to better clinical managements in population with cleft palate. Information obtained utilizing videofluoroscopy and videoendoscopy techniques which are commonly used for evaluation of the velopharyngeal mechanism has limited usefulness because of the relative inaccessibility of detailed muscle structures. Recent technical advancements of instrumental evaluation such as MRI expand our understanding of the LVP muscle in normal and abnormal anatomy and furthermore, make it possible to identify normal/abnormal distribution and quantity of the LVP muscle. The purpose of this study is to review literature focusing on anatomic aspects of the LVP muscle using a variety of techniques and to address the clinical implication of the LVP muscle in assessment and treatment of population with cleft palate.

Ⅱ. Anatomy of the Levator Veli Palatini Muscle

1. LVP Muscle in the Normal Anatomy

Normal speech production and swallowing require velar movement, which results in coupling and decoupling of the oral and nasal cavities. The LVP muscle is the primary muscle responsible for velar elevation during speech and swallowing. Most anatomic investigations of the velopharyngeal muscles have been conducted using dissection and histological techniques. Literature on normal / abnormal anatomy of the velopharyngeal muscles shows that sufficient muscle volume of the LVP and its inserted position relative to the soft palate are crucial factors to determine velopharyngeal competence for speech and swallowing (Dickson, 1972; Hoopes et al., 1970). <Figure - 1> shows the LVP muscle in relation to the palate, auditory tube, pharynx and other muscles in a normal newborn. The LVP muscle originates from the lower surface of the petrous portion of the temporal bone and courses along the surface under the auditory tube. The muscle descends and inserts into the soft palate where it blends with levator fibers from the opposite site (Kuehn & Moon, 2005). The LVP muscle has a broad insertion extending into the middle and posterior third of the soft palate (Boorman & Sommerlad, 1985; Dickson et al., 1974; Huang, Lee & Rajendran, 1998; Kuehn & Kahane, 1990). In the normal mechanism, no muscle tissue attaches directly to the hard palate (Dickson, 1972; Kuehn & Kahane, 1990). The paired levator muscles form a muscular sling effectively to serve velar elevation. In particular, the angle of the inserting LVP muscle allows the velum to be pulled along a superior and posterior path that may be linear or somewhat curved (Kent, Carney & Severeid, 1974; Kuehn, 1976). Huang, Lee & Rajendran (1998) observed the LVP and other velopharyngeal muscles in 18 fresh cadaveric specimens of normal adults. They reported that the LVP muscle forms a muscular sling and its muscle fibers occupy the middle 50 percent of the velar length measured from the posterior nasal spine to the tip of the uvula. They also reported that the fibers of each muscle bundle of the levator lay in a transverse orientation without significant overlap across the midline even though this is in contrast to the findings of Kuehn & Moon (2005). In their histologic study, Kuehn & Moon (2005) found that levator fibers cross the midline to mingle with levator fibers on

the opposite side. Anteriorly, the levator muscle is attached to the posterior margin of the aponeurosis of the tensor veli palatini with overlap on its oral surface medially. Huang, Lee & Rajendran (1998) confirmed that the LVP muscle is well placed to function as the primary mover in velar elevation.



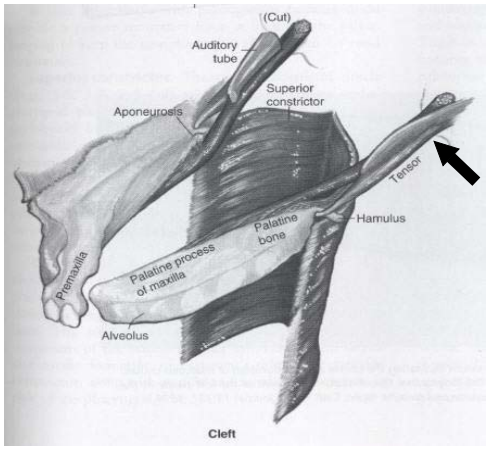
<Figure - 1> Superolateral view of the palate and pharynx showing the LVP muscle and other muscles in a normal newborn. Arrows indicate the LVP muscle sling (Millard, 1980).

In addition to velar elevation, some researchers have presented evidence that the LVP muscle plays a role in lateral pharyngeal wall movement for normal velopharyngeal closure for speech (Beery, Rood & Schramm, 1983; Bell-Berti, 1976; Honjo, Harada & Kumazawa, 1976; Lavorato & Lindholm, 1977; Yamawaki, Nishimura & Suzuki, 1999; Ysunza et al., 1999). Although there is controversy about which muscle is responsible for lateral pharyngeal wall movement during speech, several investigators have argued that the LVP muscle contributes to mesial movement of the lateral wall component of normal velopharyngeal closure. These studies have included various techniques such as electromyographic and cinematographic analyses conducted by Bell-Berti (1976) and Honjo, Harada & Kumazawa (1976), and nasoendoscopic analyses by Lavorato and Lindholm (1977). Counter evidence indicating that the levator muscle may not be responsible for the lateral pharyngeal wall movement, at least at the level of the hard palate, was provided by Iglesias, Kuehn & Morris (1980). However, using rapid MRI technique Yamawaki, Nishimura & Suzuki (1999) reported that the LVP muscle contraction contributes to the lateral pharyngeal wall movement on phonation. In their study the transverse plane of rapid MRI indicated that medial movement of the lateral pharyngeal walls was coincident with inward displacement of the eustachian tube cartilage, which was caused by contraction of the LVP muscle. The level of this movement was clearly shown in the plane 5mm lateral to the mid-sagittal plane on rapid MRI. This sagittal plane could display the actual side of medial movement of the lateral pharyngeal wall and could not be obtained by other methods of imaging such as multiview fluoroscopy and radiography. Yamawaki, Nishimura & Suzuki (1999) showed that MRI information of serial changes within a short period has great potential to shed light on anatomic and physiologic features of the velopharyngeal mechanism, for which other techniques have limitations.

2. LVP Muscle in the Population with Cleft Palate

Cleft palate anatomy has been well described in the literature (Dickson, 1972; Latham, Long & Latham, 1980; Maue-Dickson & Dickson, 1980). Some studies recorded anatomical findings in cleft palates at the time of intraoperative muscle dissection in patients undergoing cleft palate repairs (Koch, Grzonka & Koch, 1998, 1999;

Mehendale, 2004; Sommerlad et al., 1994, 2002). Latham, Long & Latham (1980) demonstrated convergence of the fibers of the LVP, palatopharyngeus, and uvulus muscles to form a compact bundle that inserts into the posterior nasal hemispine and medial margin of the bony cleft palate. Muscle fibers course from one rigid attachment to another, thereby leading potentially to a situation of isometric contraction. Velar muscles are oriented in the wrong direction, that is, anteroposteriorly instead of transversely. Therefore, to achieve an anatomic reconstruction in palatal repair, it appears logical that the velar muscles should be detached from their abnormal attachments to the posterior-medial edges on each side of the clefted bony palate and then moved into a position where they could more effectively raise the velum (Moon & Kuehn, 1997).



<Figure - 2> Superolateral view of the palate and pharynx, showing the LVP muscle in a newborn with a cleft. Two arrows indicate the origin points of the LVP muscle. (Millard, 1980).

Many researchers have focused on the functional implications of cleft anatomy with emphasis on the LVP muscle which is immobilized by its abnormal insertion into the cleft margin (Maue-Dickson & Dickson, 1980). In infants born with cleft palate, the course and insertion of the LVP muscle is markedly abnormal. <Figure - 2> illustrates a superolateral view of the palate and pharynx, showing the position of the LVP muscle in a newborn with a cleft.

The LVP muscle fibers in the cleft palate anatomy are attached to the posterior nasal spine and medial margin of the bony cleft palate, instead of forming the levator sling. The literature has provided information about abnormal insertions (wrong placement), underdeveloped muscle volume, impaired muscle function, and insufficient or disorganized muscle tissue. The LVP muscle generally inserts, together with fibers of the palatopharyngeus, into the posterior-medial margin of the cleft hard palate (Dickson, 1972; Koch, Grozonka & Koch, 1998; Latham, Long & Latham, 1980). The abnormal attachments of the LVP muscle inevitably result in isometric contraction of the muscle. Therefore, surgeons must attempt to detach the LVP muscle fibers from the abnormal attachments to the posterior-medial edges of each side of the clefted bony palate for an anatomic reconstruction during palatal repair.

The pathomorphology of the LVP muscle in cleft palates is well-documented in the literature (Cohen et al., 1994; Dickson, 1972; Koch, Grozonka & Koch, 1998; Lindman, Paulin & Stål, 2001; Maue-Dickson & Dickson, 1980). Lindman, Paulin & Stål (2001) showed that the LVP muscle of children with cleft palate is morphologically different from that of the normal adult. They found that the LVP muscles in infants with cleft palate had a greater amount of connective tissue, a lack of contractile tissue within the muscles and a significantly smaller mean diameter of the fibers than those of normal adult subjects. They assumed that the differences might be related to different stages in maturation of the muscles, changes in functional demands with growth and age, and/or a consequence of the cleft. They also suggested that the morphologic

differences might be one possible explanation of a persistent postsurgical velopharyngeal insufficiency in some patients with cleft palate. Cohen et al. (1994) compared the morphologic status of all soft palates in fetuses with cleft palate to age-matched and nonmatched control specimens. They attempted to test the hypothesis that soft palate muscles are abnormal in cleft palate. They found that soft palate muscles in the cleft specimens showed delayed development in the 10 - to 12.5 - week fetuses. Older cleft specimens did not show striking morphologic delays, but were remarkably disoriented and disorganized, especially close to the medial epithelial edge of the cleft. These abnormalities persist throughout prenatal life and are present in the soft palate muscle at the time of palate repair in the majority of children with cleft palates (Cohen et al., 1993). Cohen et al. (1993) also found that the LVP muscle is the most abnormal muscle in prenatal infants with cleft palate and in some fetuses the LVP muscle could not be distinguished as a discrete muscle.

Given the importance of the LVP muscle for velar elevation and the most common and serious abnormality of the muscle in infants with cleft palate, proper surgical repositioning of the LVP muscle is critical with regard to subsequent velar elevation for speech and swallowing. Hoopes et al. (1970) suggested that the relatively anterior insertion of the LVP muscle near the posterior border of the hard palate is less favorable for velar elevation, and therefore result in velopharyngeal insufficiency. In addition to proper repositioning of the LVP muscle across the midline, sufficient muscle mass to provide adequate velar elevation is essential for successful surgical repair. Lewin, Heller & Kojak (1975) explained that muscular hypoplasia accounts for most of the failures in achieving velopharyngeal competence after primary palate surgery. The literature on the LVP muscle in cleft palate anatomy suggests that we require a reliable method for measuring insertion points and volume of the muscle in the soft palate either before or after repair.

Unlike clinical managements in the past where a pharyngeal flap was routinely considered for individuals with repaired cleft palate who were showing velopharyngeal insufficiency, various secondary surgical procedures for velopharyngeal insufficiency have been recently performed (Furlow, 1986; Sommerlad et al., 2002). In addition, it is reported that speech therapy focusing on improvement of muscle strength such as the continuous positive airway pressure (CPAP) therapy might result in reduction of hypernasal speech in populaiton with velopharyngeal dysfunction (Kuehn et al., 2002). Given these recent various treatment options for velopharyngeal insufficiency, it is important to determine which type of clinical treatment would provide the best result based on anatomic features specific to individuals with cleft palate. Therefore, to make an appropriate clinical decision and to improve the success rate of clinical managements, it is essential to perform more complete anatomic and physiologic evaluation using a reliable method for identification of distribution and quantity of the LVP muscle.

Ⅲ. Clinical Implications of the LVP Muscle for Assessing Velopharyngeal Function

1. Instrumental Evaluation of the Velopharyngeal Mechanism

A thorough understanding of anatomic and physiologic features of the velopharyngeal mechanism is required in the

diagnosis and treatment of patients with cleft palate. To obtain complete anatomic and physiologic information, several techniques have been used in evaluation of the velopharyngeal mechanism. Videofluoroscopy and videoendoscopy are the most commonly used methods for visualizing and evaluating the velopharyneal mechanism in clinical settings. Videoendoscopy appears to be the only method capable of directly visualizing the velopharynx in real time without the need for ionizing radiation. However, it is an invasive method, and thus it is difficult to perform on children. It also depicts only one imaging perspective (typically from above the velopharynx) and provides very limited information on underlying velopharyngeal muscles. Currently, videofluoroscopy is also frequently used in the clinical evaluation of the velopharyngeal mechanism. The technique provides images of velopharyngeal movements typically in the sagittal plane. However, it uses ionizing radiation and produces superimposed images that might obscure differences between bone and soft tissue. Another disadvantage is limitation of soft tissue contrast. Because of these limitations of the two techniques, thorough evaluation about muscle structure in vivo could not usually be performed in the past. The disadvantages of the two techniques support the application of MRI, which is noninvasive, easily repeatable, and does not use ionizing radiation (Beer et al., 2004). Furthermore, the completely free choice of the image plane in MRI helps clinicians to capture a variety of images and obtain more detailed information on structures which other techniques are unable to detect. MRI also allows us to observe the underlying muscles and other soft tissues due to its excellent soft tissue contrast. MRI procedures appear to be the best to identify muscles and discriminate muscles from the surrounding soft tissues.

Technological advances of MRI make it possible to image palates of babies born with cleft palate and obtain detailed anatomic information such as muscle tissue orientation and distribution before palatal surgery. Using MRI procedures, it is possible to identify patient-specific dysfunction and perform individual-specific surgical treatments; therefore, it eventually leads to improved surgical outcomes. On the basis of the information obtained from MRI procedures with regard to muscle size, orientation, and location, the normal velopharyngeal mechanism can be better emulated during reconstructive surgical procedures. Moreover, MRI techniques allow clinicians and researchers to more thoroughly investigate the LVP muscle in normal subjects and in patients with cleft palate. Such information has significant implications for preoperative planning (Ettema et al., 2002; Kuehn et al., 2001, 2004)

2. MRI studies on the LVP muscle

Recently, a substantial number of studies investigated the LVP muscle in normal subjects and in individuals with cleft palate using MRI techniques (Ettema et al., 2002; Ha, 2006; Ha et al., in press; Kuehn et al., 2001, 2004). Kuehn et al. (2001, 2004) observed the LVP muscles in subjects with submucous cleft palate or cleft palate using presurgery and postsurgery MRI evaluations. Using MRI, Kuehn et al. (2001) evaluated patients suspected of having occult submucous cleft palate. MR images in the study provided evidence of an interruption of levator veli palatini muscle tissue in the midline and a substantial attachment of levator muscle tissue to the posterior of the hard palate. They also found that bilateral encapsulating sheaths interrupted the normal progression of the levator muscle sling across the midline. The MR images after surgical repair indicated that the LVP muscle sling was crossing the midline. Kuehn et al.

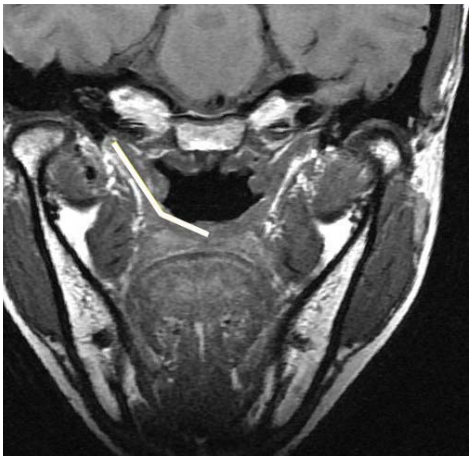
(2004) also focused on the LVP muscle with particular attention in the MRI evaluation of patients with cleft palate before and after primary palatoplasty. They qualitatively described the muscle volume, original insertion location, and repositioning of the LVP muscle in seven infants with cleft palate before and after surgery. The results from the study suggest that infants with cleft palate may possess LVP muscle volume that is proportionately similar to infants and adults with normal intact palates. Thus, if the divided levator bundles are repositioned properly to form a sling across the velar midline, the prognosis for normal speech may be favorable in those patients. On the other hand, if muscle volume is proportionately small, prognosis for normal speech is less favorable. Kuehn et al. (2001, 2004) argued that the ability to image velopharyngeal muscles has important implications. The anatomic information such as the angle of levator origin and muscle volume contributes to better clinical decisions regarding surgical management or behavioral therapy.

Ettema et al. (2002) provided detailed anatomic information of the LVP muscle in normal adults using MRI. They obtained both static and dynamic images of the velopharyngeal region, specifically the LVP muscle and reported quantitative measures of the configuration of the LVP muscle at rest and during speech tasks consisting of nasal and nonnasal sounds mixed with vowels (high vs. low), consonants (bilabial vs. alveolar), or both. Regarding the configuration of the LVP muscles, specifically, they measured (1) the distance between origins of the LVP (reference line), (2) length, (3) thickness of LVP muscles, and (4) angles of origin in relation to the reference line and the belly of the muscle along its course into the velum. Among these measurements, the angle of origin is associated with the relation between the base of the skull and the origin and course of the LVP muscle. They reported that the levator muscle is at its greatest length during rest and becomes progressively shorter for nasal consonants, low vowels, high vowels, and fricative consonants. The angle of origin in their study became smaller (more acute) corresponding to the degree of muscle contraction and velar elevation. The investigators indicated that detailed information on muscle change in MR images could help to detect coarticulatory effects. With regard to angle of origin, nasals following fricative consonants are different from nasals preceding fricative consonants.

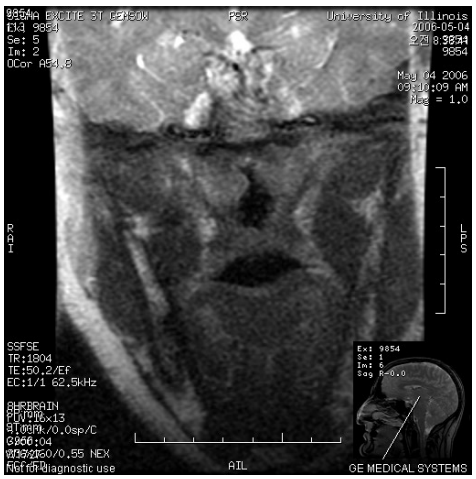
These findings in Ettema et al. (2002) suggest that quantitative measurements can be used for objectively evaluating the distribution, quantity, and function of the muscle before and after palatoplasty. The angle of origin and the length and volume of the LVP muscle appear to be especially useful indicators which can assess the function and capacity of the LVP muscle, and furthermore, velar elevation. One can predict that the most appropriate treatment including surgical protocols and speech therapy for individuals with cleft palate will be determined based on effective presurgical identification of muscle fiber distribution with MRI. In addition, the velopharyngeal mechanism can be evaluated with regard to muscle tissue distribution and orientation in a quantified and objective manner after primary palatoplasty. The findings in Ettema et al. (2002) have important implications regarding anatomic and physiologic aspects of the LVP muscle. In addition, quantitative measurements of the LVP muscles in normal subjects can be utilized usefully as reference data for patients with cleft palate.

Ha (2006) provided anatomic and physiologic information on the LVP muscles in vivo in individuals with repaired cleft plate derived from MRI techniques. <Figure - 3> presents the oblique coronal section of the head in a normal subject which shows the

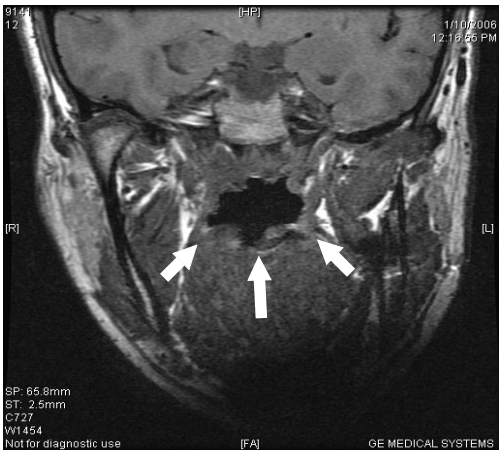
entirety of the LVP muscle sling and illustrates how to measure the length of the LVP muscle. <Figure - 4> shows the dynamic image of a subject with repaired cleft palate during production of the speech sound /a/. Quantitative measurements of the LVP muscle were obtained using the dynamic images to investigate configuration differences of the muscle in terms of oral versus nasal consonants, and high versus low vowels. The result of the study showed that the configuration of the levator muscle sling appeared to vary among subjects with repaired cleft palate and differed from the muscle sling of normal velopharyngeal anatomy. For example, the LVP muscle in one male subject did not form a complete muscle sling and showed a discontinuity <Figure - 5>. Also, the thickness of the muscle bundles in another female subject appeared to be relatively thin <Figure - 6>. The author assumed that the incomplete muscular sling and the thin muscle bundles might result in reduced velar elevation and might be related to mild hypernasality and audible nasal air emission which the two subjects showed at the time of the experiments. In comparison with the Ettema et al. (2002) study, results of this study indicated that the LVP muscle in individuals with cleft palate is shorter in length and it has lesser muscle contractility, larger variability in angles of origin, and lesser muscle thickness. The results suggest that these different anatomic features of the LVP muscle in individuals with cleft palate might result in persistent postsurgical velopharyngeal inadequacy in some individuals with cleft palate despite the primary surgical repair. With regard to dynamic images during speech production, the author found that there were systematic changes of the LVP muscle configuration in terms of vowel and consonant types. LVP muscle angle of origin and length became progressively smaller from rest, nasal consonants, low vowels, high vowels, and fricative consonants. These changes are consistent with those of the normal subjects suggesting that the pattern of neuromotor control is similar between the two groups.



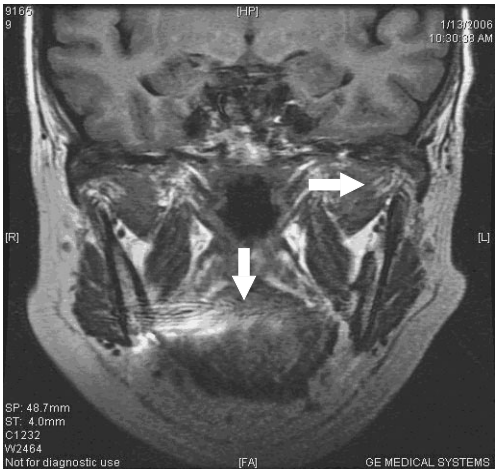
<Figure - 3> Oblique coronal image shows the entirety of the LVP muscle sling in a normal subject. A line represents the length of the right side of the LVP muscle. Two arrows indicate the origin points of the muscle (Ha, 2006).



<Figure - 4> Dynamic image of a subject with repaired cleft palate during production of the speech sound /a/. Angle and location of the section are shown in the inset of the figure (Ha, 2006).



<Figure – 5> Oblique coronal magnetic resonance image of a male subject with cleft palate. The arrows depict the LVP muscle bundle. This subject shows disconnection of the levator veli palatini muscle sling (Ha, 2006).



<Figure – 6> Oblique coronal magnetic resonance image of a female subject with cleft palate. The arrows depict the LVP muscle bundle. This subject shows relatively thin LVP muscle sling (Ha, 2006).

The Ha (2006) study confirmed that the velopharyngeal mechanism can be evaluated with regard to muscle tissue distribution and orientation in a quantified and objective manner using MRI techniques. Particularly, sufficient muscle length and volume of the LVP and its inserted position relative to the velum might be crucial factors to determine velopharyngeal competence for speech and nonspeech activities. Effective identification of muscle fiber distribution and quantitative measurements of the muscle using MRI techniques will eventually contribute to the most appropriate treatment including surgical protocols and speech therapy for individuals with cleft palate. For example, if the abnormal attachment of the LVP muscle to the hard palate in a patient with cleft palate is identified using MRI, which results in velopharyngeal insufficiency and hypernasal speech, secondary palatoplasties such as veloplasty, and palate re-repair which place an emphasis upon the anatomic restoration of the displaced LVP to a more normal transverse orientation would be the best option to treat residual velopharyngeal insufficiency (Sommerlad et al., 2002). In addition, if patients show normal anatomic features of the velopharyngeal mechanism including the insertion points of the LVP muscle, but thin and insufficient muscle thickness like the female speaker with cleft palate in the Ha (2006) study <Figure - 6>, behavioral speech therapy focusing on improvement of muscle strength would be a more appropriate option to treat hypernasal speech. Continuous positive airway pressure (CPAP) therapy is an example of speech treatments to improve muscle strengthening (Kuehn et al., 2002).

Ⅳ. Conclusion

Clinical services for patients with cleft palate should be based on complete understanding of anatomic and physiologic characteristics of the velopharyngeal mechanism. The LVP muscle has been of clinical and research interest because of its importance for velar elevation and normal speech and

abnormalities in individuals with cleft palate. There is general agreement in the literature of the LVP muscle in cleft palate anatomy that the abnormal location / attachment, underdeveloped muscle volume, and insufficient muscle tissues are closely associated with velopharyngeal dysfunction. In the past, thorough investigation of the muscle in vivo had shown limitations due to the relative inaccessibility of detailed muscle structures. Technological advancements of MRI have led to better investigation of the LVP muscle in normal and abnormal anatomy and furthermore, have allowed us to identify normal/abnormal distribution and quantity of the LVP muscle and more reliably to evaluate the muscle structures. Several recent studies investigated the LVP muscle in individuals with or without cleft palate using MRI and showed the usefulness of MRI in the evaluation of the muscle structures. These studies suggest that it is possible to identify patient-specific dysfunction, to perform individual-specific surgical treatments, and to design speech therapy focusing on improvement of abnormal muscle structure or function using MRI procedures. Effective identification of muscle fiber distribution and quantitative measurements of the muscle using MRI techniques will eventually contribute to the most appropriate treatment including surgical protocols and speech therapy for individuals with cleft palate

Continued research efforts should be made toward a comprehensive understanding of anatomic and physiologic characteristics of the normal and abnormal velopharyngeal muscle structures including the LVP muscle if clinicians are to provide more appropriate and optimal clinical services to individuals with cleft palate and other craniofacial anomalies. In particular, it is necessary to evaluate the integrity of the velopharynx for speech purposes in individuals born with cleft palate. More complete anatomic information of the velopharyngeal mechanism is needed in a larger number of individuals with cleft palate considering possible influential factors of anatomic features including cleft palate type, age, gender, and speech ability.

In future studies, the relationship among anatomic information of the LVP muscle, velopharyngeal function, and speech ability should be investigated in the cleft palate population. Specifically, it would be important to determine to what extent muscle length and angle of origin of the LVP muscle predict velopharyngeal function and speech ability. It would be important to divide speakers with cleft palate into groups according to velopharyngeal function or resonance problems. Then the quantitative measurements of the LVP muscle at rest and during speech could be compared between groups. Such a study would contribute to an understanding about how far or to what extent abnormality of the LVP muscle causes less effective velar function and hypernasalized speech. Continued efforts using MRI are currently underway in my laboratory to investigate more complete anatomic features of the velopharyngeal region in individuals with cleft palate and relationships with speech production.

References

Beer, A. J., Hellerhoff, P., Zimmermann, A., Mady, K., Sader, R., Rummeny, E. J., & Hanning, C. (2004). Dynamic near-real-time magnetic resonance imaging for analyzing the velopharyngeal closure in comparison with videofluoroscopy. *Journal of Magnetic Resonance Imaging, 20(5),* 791-797.

Beery, Q. C., Rood, S. R., & Schramm, V. L. (1983). Pharyngeal wall motion in prosthetically managed cleft palate adults. *The Cleft Palate Journal, 20(1),* 7-17.

Bell-Berti, F. (1976). An electromyographic study of velopharyngeal function in speech.

Journal of Speech, and Hearing Research, 19(2), 225-240.

Boorman, J. G., & Sommerlad, B. C. (1985). Levator palati and palatal dimples: Their anatomy, relationship and clinical significance. *British Journal of Plastic Surgery, 38(3),* 326-332.

Bradley, D. P. (1997). Congenital and acquired velopharyngeal inadequacy. In K. R. Bzoch (Ed.), *Communicative disorders related to cleft lip and palate.* Austin, TX: PRO-ED.

Cohen, S. R., Chen, L., Trotman, C. A., & Burdi, A. R. (1993). Soft-palate myogenesis: A developmental field paradigm. *The Cleft Palate-Craniofacial Journal, 30(5),* 441-446.

Cohen, S. R., Chen, L. L., Burdi, A. R., & Trotman, C. A. (1994). Patterns of abnormal myogenesis in human cleft palates. *The Cleft Palate-Craniofacial Journal, 31(5),* 345-350.

Dickson, D. R. (1972). Normal and cleft palate anatomy. *The Cleft Palate Journal, 9,* 280-293.

Dickson, D. R., Grant, J. C., Sicher, H., Dubrul, E. L., & Paltan, J. (1974). Status of research in cleft palate anatomy and physiology: July, 1973-Part 1. *The Cleft Palate Journal, 11,* 471-492.

Ettema, S. L., Kuehn, D. P., Perlman, A. L., & Alperin, N. (2002). Magnetic resonance imaging of the levator veli palatini muscle during speech. *The Cleft Palate-Craniofacial Journal, 39(2),* 130-144.

Furlow, L. T. (1986). Cleft palate repair by double opposing Z-plasty. *Plastic and Reconstructive Surgery, 78(6),* 724-738.

Ha, S. (2006). Magnetic resonance imaging of the levator veli palatini muscle in speakers with repaired cleft palate. Unpublished doctoral dissertation. University of Illinois, Urbana-Champaign.

Ha, S., Kuehn, D. P., Cohen, M., & Alperin, N. (in press). Magnetic resonance imaging of the levator veli palatini muscle in speakers with repaired cleft palate. *The Cleft Palate-Craniofacial Journal.*

Honjo, I., Harada, H., & Kumazawa, T. (1976). Role of the levator veli palatini muscle in movement of the lateral pharyngeal wall. *Archives of Oto-Rhino-Laryngology, 212(2),* 93-98.

Hoopes, J. E., Dellon, A. L., Fabrikant, J. I., & Soliman, A. H. (1970). Cineradiographic assessment of combined island flap pushback and pharyngeal flap in the surgical management of submucous cleft palate. *British Journal of Plastic Surgery, 23(1),* 39-44.

Huang, M. H., Lee, S. T., & Rajendran, K. (1998). Anatomic basis of cleft palate and velopharyngeal surgery: Implications from a fresh cadaveric study. *Plastic and Reconstructive Surgery, 101(3),* 613-627; discussion 628-619.

Iglesias, A., Kuehn, D. P., & Morris, H. L. (1980). Simultaneous assessment of pharyngeal wall and velar displacement for selected speech sounds. *Journal of Speech and Hearing Research, 23(2),* 429-446.

Kent, R. D., Carney, P. J., & Severeid, L. R. (1974). Velar movement and timing: Evaluation of a model for binary control. *Journal of Speech and Hearing Research, 17(3),* 470-488.

Koch, K. H., Grzonka, M. A., & Koch, J. (1998). Pathology of the palatal aponeurosis in cleft palate. *The Cleft Palate-Craniofacial Journal, 35(6),* 530-534.

Koch, K. H., Grzonka, M. A., & Koch, J. (1999). The pathology of the velopharyngeal musculature in cleft palates. *Anatomischer Anzeiger, 181(1),* 123-126.

Kuehn, D. P. (1976). A cineradiographic investigation of velar movement variables in two normals. *The Cleft Palate Journal, 13,* 88-103.

Kuehn, D. P., Ettema, S. L., Goldwasser, M. S., & Barkmeier, J. C. (2004). Magnetic resonance imaging of the levator veli palatini muscle before and after primary palatoplasty. *The Cleft Palate-Craniofacial Journal, 41(6),* 584-592.

Kuehn, D. P., Ettema, S. L., Goldwasser, M. S., Barkmeier, J. C., & Wachtel, J. M. (2001). Magnetic resonance imaging in the evaluation of occult submucous cleft palate. *The Cleft Palate-Craniofacial Journal, 38(5),* 421-431.

Kuehn, D. P., Imrey, P. B., Tomes, L., Jones, D. J., O'Gara, M. M., Seaver, E. J., Smith, B. E., Demark, D. V., & Wachtel, J. M. (2002). Efficacy of continuous positive airway pressure for treatment of hypernasality. *The Cleft Palate-Craniofacial Journal, 39(3),* 267-276.

Kuehn, D. P., & Kahane, J. C. (1990). Histologic study of the normal human adult soft palate. *The Cleft Palate Journal, 27(1),* 26-34; discussion 35.

Kuehn, D. P., & Moon, J. B. (2005). Histologic study of intravelar structures in normal human adult specimens. *The Cleft Palate-Craniofacial Journal, 42(5),* 481-489.

Latham, R. A., Long, R. E., Jr., & Latham, E. A. (1980). Cleft palate velopharyngeal musculature

in a five-month-old infant: A three dimensional histological reconstruction. *The Cleft Palate Journal, 17(1),* 1-16.

Lavorato, A. S., & Lindholm, C. E. (1977). Fiberoptic visualization of motion of the eustachian tube. *Transactions. Section on Otolaryngology. American Academy of Ophthalmology and Otolaryngology, 84(3 Pt 2),* ORL 534-541.

Lewin, M. L., Heller, J. C., & Kojak, D. J. (1975). Speech results after Millard island flap repair in cleft palate and other velopharyngeal insufficiencies. *The Cleft Palate Journal, 12,* 263-269.

Lindman, R., Paulin, G., & Stål, P. S. (2001). Morphological characterization of the levator veli palatini muscle in children born with cleft palates. *The Cleft Palate- Craniofacial Journal, 38(5),* 438-448.

Marrinan, E. M., LaBrie, R. A., & Mulliken, J. B. (1998). Velopharyngeal function in nonsyndromic cleft palate: Relevance of surgical technique, age at repair, and cleft type. *The Cleft Palate-Craniofacial Journal, 35(2),* 95-100.

Maue-Dickson, W., & Dickson, D. R. (1980). Anatomy and physiology related to cleft palate: Current research and clinical implications. *Plastic and Reconstructive Surgery, 65(1),* 83-90.

Mehendale, F. V. (2004). Surgical anatomy of the levator veli palatini: A previously undescribed tendinous insertion of the anterolateral fibers. *Plastic and Reconstructive Surgery, 114(2),* 307-315.

Millard, D. R. (1980). *Cleft craft: The evolution of its surgery, III: Alveolar and palatal deformities.* Hagerstown, MD: Lippincott Williams & Wilkins.

Moon, J. B., & Kuehn, D. P. (1997). Anatomy and physiology of normal and disordered velopharyngeal function for speech. In K. R. Bzoch (Ed.), *Communicative disorders related to cleft lip and palate.* Austin, TX: PRO-ED.

Sommerlad, B. C., Henley, M., Birch, M., Harland, K., Moiemen, N., & Boorman, J. G. (1994). Cleft palate re-repair: A clinical and radiographic study of 32 consecutive cases. *British Journal of Plastic Surgery, 47(6),* 406-410.

Sommerlad, B. C., Mehendale, F. V., Birch, M. J., Sell, D., Hattee, C., & Harland, K. (2002). Palate re-repair revisited. *The Cleft Palate-Craniofacial Journal, 39(3),* 295-307.

Witt, P. D., & Marsh, J. L. (1997). Advances in assessing outcome of surgical repair of cleft lip and cleft palate. *Plastic and Reconstructive Surgery, 100(7),* 1907-1917.

Yamawaki, Y., Nishimura, Y., & Suzuki, Y. (1999). Eustachian tube cartilage and medial movement of lateral pharyngeal wall on phonation. *Plastic and Reconstructive Surgery, 104(2),* 350-356.

Ysunza, A., Pamplona, M. C., Molina, F., Chacon, E., & Collado, M. (1999). Velopharyngeal motion after sphincter pharyngoplasty: A videonasopharyngoscopic and electromyographic study. *Plastic and Reconstructive Surgery, 104(4),* 905-910.

초 록

구개파열자의 구개올림근에 관한 해부학적 특징과 진단: 문헌연구

하승희
(테네시주립대학교 언어병리-청각학과)

하승희. 구개파열자의 구개올림근에 관한 해부학적 특징과 진단: 문헌연구.『언어청각장애연구』, 2007, 제12권, 제1호, 77-89. 배경 및 목적: 구개올림근은 구개파열을 가진 사람들에게서 가장 비정상적인 구조와 기능을 보이는 대표적인 근육으로서 연구개의 움직임에 중요한 역할을 하고 구개파열자의 말 문제와 밀접한 관련이 있다. **방법:** 본 연구에서는 정상인과 구개파열자를 대상으로 한 구개올림근의 해부학적 특징에 관한 문헌을 살펴보았고, 그 연구들이 구개파열자를 대상으로 하는 평가와 치료에 주는 임상적 의의를 살펴보았다. **결과 및 결론:** 선행연구들은 구개올림근의 비정상적인 삽입점과 근육 부피가 연인두 기능부전과 밀접한 관련이 있으며, 구개파열자에게 보다 적절하고 효과적인 치료를 제공하기 위해서는 연인두 부위와 구개올림근의 보다 세밀한 평가가 필요함을 보여주었다. 최근 자기공명영상술의 발전으로 구개올림근에 대한 보다 면밀한 평가와 이해가 가능해졌고, 구개파열자를 대상으로 2차 구개열 수술과 말치료에 대한 보다 적합한 선택이 가능해졌다.

핵심어: 구개올림근, 해부학적 특징, 구개파열, 자기공명영상술

▸ 게재신청일: 2007년 1월 19일
▸ 수정제출일: 2007년 2월 20일
▸ 게재확정일: 2007년 2월 24일

▸ 하승희(제1저자 및 교신저자): 테네시주립대학교 언어병리-청각학과 교수, e-mail: shha610@gmail.com, tel.: +1 865 974 4802