Research Note

A Dynamic Magnetic Resonance Imaging–Based Method to Examine In Vivo Levator Veli Palatini Muscle Function During Speech

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Purpose: The aim of this study was to develop a method able to quantify levator veli palatini (LVP) muscle shortening and contraction velocities using dynamic magnetic resonance imaging (MRI) throughout speech samples and relate these measurements to velopharyngeal portal dimensions.

Method: Six healthy adults (3 men and 3 women, $M = 24.5$ years) produced syllables representing 4 different manners of production during real-time dynamic MRI scans. We acquired an oblique-coronal slice of the velopharyngeal mechanism, which captured the length of the LVP, and manually segmented each frame. LVP shortening and muscle velocities were calculated from the acquired images.

Results: Using our method, we found that subjects demonstrated greater LVP shortening and higher maximum

contraction velocities during fricative and plosive syllable production than during nasal or vowel syllable production. LVP shortening and maximum contraction velocity positively correlated with velopharyngeal port depth.

Conclusions: In vivo LVP function differs between manners of production, as expected, and an individual's velopharyngeal portal dimensions influence LVP function. These measures, contextualized with the force–length and force–velocity muscle relationships, provide new insight into LVP function. Future studies could use this method to investigate LVP function in healthy speakers and individuals with velopharyngeal dysfunction and how function relates to velopharyngeal anatomy.

The velopharyngeal (VP) mechanism consists of the velum, lateral pharyngeal walls, posterior pharyngeal wall, and associated musculature. Proper function of the VP mechanism is necessary for acceptable and perceptually normal speech production. During oral speech production in healthy individuals, the VP muscles,

primarily the levator veli palatini (LVP), move the velum superiorly and posteriorly into contact with the posterior pharyngeal wall (Huang, Lee, & Rajendran, 1998; Perry, Kuehn, & Sutton, 2013). This action, in conjunction with lateral wall contraction, seals the VP port, completely separating the nasal and oral cavities, which is required for oral speech production. VP dysfunction (VPD) occurs when the VP mechanism is unable to close the port completely and is a common issue for individuals with repaired cleft palate. How VP anatomical variability across healthy speakers affects function is unknown; furthermore, an understanding of how this structure–function relationship differs in individuals with VPD remains limited. The LVP is the primary muscle of VP closure (Bell-berti, 1976), so a method to measure in vivo LVP function is needed to empower investigation of the VP structure–function relationship.

The LVP is one of over 600 skeletal muscles in the body (Luu, Zhang, Pelland, & Blemker, 2015), all of which are characterized by two properties intrinsic to the skeletal muscle and related to muscle force-producing capability: force–length and force–velocity relationships. These two fundamental

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relationships describe how muscle length and contraction velocity affect the muscle's force-generating potential. Maximum isometric active force is produced when a muscle is at or near optimal length, and force decreases as the muscle lengthens or shortens away from optimal length (Close, 1972; Edman, 1966; Gordon, Huxley, & Julian, 1966; Huxley, 1957; Ramsey & Street, 1940). For nonisometric contraction, the velocity of the muscle contraction affects its force-generating potential. As contraction velocity increases, force decreases until a maximum velocity is reached at which no force can be generated (Bahler, Fales, & Zierler, 1968; Hill, 1938).

If muscle force-generating potential is diminished due to either muscle length, muscle velocity, or both, two possibilities exist: either the LVP cannot achieve the force necessary for effective closure of the VP mechanism and thus result to hypernasality and dysfunctional speech, or if closure is possible, greater LVP muscle activation is required to achieve closure, and repeated activations cause fatigue during speech. A computational modeling study predicted that certain advantageous VP anatomies could produce nearly twice the degree of closure, as measured by closure force, as less advantageous anatomies for the same LVP activation level (Inouye, Perry, Lin, & Blemker, 2015). In that study, the disadvantageous anatomies required greater amounts of LVP shortening, moving the muscle further away from optimal fiber length and, consequently, decreasing the force-generating ability of the muscle. These results highlight the need for further investigation into the relationship between VP anatomy and LVP muscle lengths and velocities. In order to test the hypotheses posed above and quantitatively establish what constitutes healthy LVP function, a new experimental method to capture LVP muscle lengths and velocities *in vivo* is needed.

The goals of this study are to (a) develop a new methodology for determining LVP muscle shortening (length change) and muscle velocities during speech production using real-time dynamic magnetic resonance imaging (MRI) and (b) demonstrate our method's utility to advance understanding of *in vivo* LVP function by comparing LVP function in syllables that represent four different manners of production and relating these LVP functional measures to VP anatomy. We describe a method for calculating LVP muscle shortening and contraction velocity from oblique-coronal static and dynamic MRI that can be utilized with any speech sample, and our method is exhibited for a sampling of English syllables. We predict that measures derived from our method will be able to distinguish oral consonants requiring high oral pressures from vowels and nasal consonants, which require lower oral pressures (Andreassen, Smith, & Guyette, 1991). Additionally, we expect that our LVP functional measures will correlate with VP port depth.

Method

Participants

Six White healthy adults, consisting of three men and three women, between the ages of 21 and 29 years

($M = 24.5$ years, $SD = 3.3$ years) with a body mass index of less than 27, participated in this study. All participants were native English speakers, and none reported history of musculoskeletal disorders, sleep apnea, or neurological disorders that could affect the study's region of interest. Additionally, each participant was judged by the first author to have normal resonance. No subjects had any contraindications for MRI scans. Informed consent was obtained for each participant, and this study was approved by the institutional review board of the University of Virginia.

MRI

All participants were scanned using a Siemens Avanto 1.5-T magnetic resonance scanner with head and neck coil arrays. After localizer scans, 2 three-dimensional (3D) anatomic scans were performed while each participant was at rest. Each subject was instructed to breathe normally through the nose and swallow as infrequently as possible to minimize velum motion and image blurring. A high-resolution, T2-weighted turbo spin echo scan with the optimized SPACE (sampling perfection with application-optimized contrasts using different flip angle evolutions) protocol (Mugler, 2014) was used to acquire images in both static scans (details in Table 1). Sagittal images acquired from the first 3D scan were used to identify an oblique-coronal plane containing the length of the LVP. Images from the second 3D scan were parallel to this oblique-coronal plane. Scan time for each static scan was slightly more than 8 min.

Images of the VP mechanism during speech were acquired using a real-time spiral gradient echo sequence (Feng et al., 2018). This sequence allows for simultaneous acquisition of two nonparallel slices. For most effective evaluation of velar and LVP motion during speech, a mid-sagittal slice and an oblique-coronal slice along the length of the LVP (parallel to the static oblique-coronal plane) were chosen. Images from the static oblique-coronal scan were used to determine the optimal location for the dynamic oblique-coronal slice. Each dynamic slice has a temporal resolution of 18.2 frames per second (fps) and a spatial resolution of $1.2 \times 1.2 \text{ mm}^2$, with a $156 \times 156 \text{ mm}^2$ field of view and a slice thickness of 8 mm. Each participant completed eight dynamic scans while producing a different English syllable for 6.6-s real-time scan. Participants were asked to produce two plosive consonant syllables (/bʌ/, /kʌ/), two fricative consonant syllables (/sʌ/, /fʌ/), two nasal consonant syllables (/mʌ/, /nʌ/), and two vowel syllables (/æ/, /i/). Participants repeated the chosen syllables three times at a self-selected comfortable pace during the dynamic scan. Duration of each vowel syllable production was no more than 0.5 s for any subject.

Image Analyses

Both the high-resolution static images and real-time oblique-coronal dynamic images were analyzed to determine

Table 1. Static magnetic resonance imaging (MRI) protocol, 1.5 T.

Static 3D MRI parameters	
Pulse sequence	SPACE: T2 turbo spin echo, flip angle: 150
Field of view	256 × 256 × 159/119 (sag/ob-cor) mm ³
Repetition time	1,000 ms
Echo time	121/122 (sag/ob-cor) ms
Echo train length	59 ms
Averages	2
Resolution	1.0 mm, isotropic
Length of scan	8 min 22 s for 1 static volume

Note. 3D = three-dimensional; SPACE = sampling perfection with application-optimized contrasts using different flip angle evolutions; sag/ob-cor = sagittal/oblique-coronal.

LVP muscle lengths during speech (see Figure 1). In the static images, in which the LVP body can be easily identified, we defined reference lines and quantified the relationship between these reference lines and the LVP path. Then, in the lower resolution dynamic images that capture velum movement, once the reference lines were placed, we used the previously quantified relationship to determine the LVP path in each dynamic frame. All image analyses were completed in OsiriX (Rosset, Spadola, & Ratib, 2004). The high-resolution static oblique-coronal image corresponding to the location of the dynamic oblique-coronal slice was determined by location recorded by the scanner and manually confirmed. On the chosen static image, we placed two inferior–superior (vertical in 2D image) reference lines on each lateral edge of the VP port (right lateral line [RLL] and left lateral line [LLL]; see Figure 1). Additionally, we placed two lateral reference lines (horizontal in 2D image) at the superior and inferior boundaries of the velum (superior velum line [SVL] and inferior velum line [IVL]; see Figure 1). These reference lines were chosen specifically because their locations are easily identifiable in both the high-resolution static image and the lower resolution dynamic images. All references for image analysis are detailed in Table 2.

The body of the LVP in the oblique-coronal image was identified similarly to previous studies (Kollara & Perry, 2014; Perry, 2011; Perry, Sutton, Kuehn, & Gamage, 2014). We designated the origin of the LVP to be the LVP origin point (LOP). We estimated the path of the LVP as a two-segment, three-vertex line with each vertex positioned on the LVP body. The vertices were defined such that one vertex is the LOP; the middle vertex, called *velar boundary point* (VBP), lies along the LLL; and the final vertex, termed *mid-velum point* (MVP), is halfway between the RLL and LLL. With the LVP path determined, scaling factors were defined to quantify the relationship between the LVP path vertices and the SVL and IVL. The mid-velum scaling factor, MV_f , is the in-plane vertical distance between MVP and SVL divided by the velar thickness. Similarly, the velar boundary scaling factor, VB_f , is the in-plane vertical distance between VBP and SVL divided by the velar thickness. These factors were determined per subject (see Figure 1C) and led to the

Figure 1. Demonstration of imaging planes, reference lines, and measurements used for analysis. (A) Static, midsagittal view displays oblique-coronal imaging plane along the length of levator veli palatini (LVP), pharyngeal depth (defined as posterior nasal spine to posterior pharyngeal wall along the plane of hard palate), velar length, and sagittal angle (the angle between the line defined by anterior boundaries of the third and fourth cervical vertebrae and the line along the LVP length). (B) Static oblique-coronal view, defined in A, displays the LVP origin point (LOP) and four reference lines used to calculate LVP length, as well as velopharyngeal port depth. In the image plane, two reference lines are horizontal: superior velum line (SVL) and inferior velum line (IVL); two are vertical: right lateral line (RLL) and left lateral line (LLL). (C) Static, oblique-coronal view with boundaries denoted by a white dotted box in B. The two-segment path of LVP is determined such that the end vertex (mid-velum point) lies halfway between RLL and LLL, the middle vertex (velar boundary point) lies on LLL, and the end vertex is the LOP. (D) Once the path is determined, scaling factors are defined to quantify the relationship between mid-velum and velar boundary points and superior and inferior velum lines for dynamic analysis. Velar thickness (VT), mid-velum distance (ΔMV), and velar boundary distance (ΔVB) are defined as the in-plane vertical distances between SVL and IVL, MVP and SVL, and VBP and SVL, respectively. The scaling factor MV_f is ΔMV divided by VT; similarly, the scaling factor VB_f is ΔVB divided by VT. (E) Dynamic, oblique-coronal image with velum at rest displays reference lines and points with LVP path calculation. As the velum moves vertically in plane throughout the image series, SVL and IVL are adjusted in each frame. MVP and VBP are recalculated, and the two-segment path of the LVP is determined for that frame.

following relationship between LVP path vertices and reference lines:

$$\begin{aligned}
 MVP_x &= (LLL_x + RLL_x)/2 \\
 MVP_y &= SVL_y - MV_f \times (SVL_y - IVL_y) \\
 VBP_x &= LLL_x \\
 VBP_y &= SVL_y + VB_f \times (SVL_y - IVL_y).
 \end{aligned} \quad (1)$$

A dynamic image acquired with the velum at rest and the selected static image were manually aligned using

Table 2. Table detailing reference point and lines and measurements taken for image analysis.

Plane	Reference	Definition
Oblique-coronal	Levator origin point	Point located at the LVP origin
	Superior velum line (SVL)	In-plane horizontal line denoting the superior–posterior boundary of velum
	Inferior velum line	In-plane horizontal line denoting the inferior–anterior boundary of velum
	Right lateral line (RLL)	In-plane vertical line at the right lateral boundary of the VP port
	Left lateral line (LLL)	In-plane vertical line at the left lateral boundary of the VP port
	Mid-velum point (MVP)	Point identified on LVP path located in the middle of the velum (equidistant from the RLL and LLL)
	Velar boundary point (VBP)	Point identified at the intersection of LVP path and LLL
	Measure	Definition
Midsagittal	Pharyngeal depth	Posterior nasal spine to the posterior pharyngeal wall along the plane of hard palate
	Sagittal angle	Angle between the line defined by anterior boundaries of the third and fourth cervical vertebrae and the line along LVP length
	Velar length	Length of the curvilinear line from posterior nasal spine to the tip of the uvula through the middle of the velum
Oblique-coronal	Port depth	Linear distance from the anterior, midsagittal point on the surface of the velum to the posterior, midsagittal point in the oblique-coronal plane on the surface of the posterior pharyngeal wall
	Velar thickness	In-plane vertical distance between the superior velum line and the inferior velum line (also defined in midsagittal plane as distance from velar knee to velar dimple)
	ΔMV	In-plane vertical distance between MVP and SVL
	ΔVB	In-plane vertical distance between VBP and SVL

Note. LVP = levator veli palatini; MV = mid-velum distance; VB = velar boundary distance.

anatomical features and landmarks clearly distinguishable in both images, such as head outline, bottom teeth profile, and VP port location. Reference lines and LOP from the higher resolution static images were overlaid on the dynamic image (see Figure 1D). The left and right lateral port reference lines (LLL, RLL) and LOP remained constant throughout each dynamic image series. The SVL and IVL were placed manually in each dynamic image based on current velum location. The intravelar segment of the LVP was estimated as a straight line segment between MVP and VBP, and the extravelar segment was estimated as a straight line segment from VBP to LOP. The total length measurement of the LVP was the sum of the intravelar and extravelar segment lengths. LVP length was calculated for each frame of every dynamic series. LVP resting length was defined as the maximum LVP length calculated across all frames in each dynamic series. For each frame, LVP shortening, in units of millimeters, was calculated as the difference between resting length and LVP length in the current frame, that is, $LVP_{rest} - LVP_{frame}$. The largest shortening value calculated for each sample was defined to be the maximum shortening for the given speech sample. Normalized LVP shortening for each frame was calculated as LVP shortening divided by resting length, and maximum relative LVP shortening was determined for each speech sample. LVP lengths were plotted versus time to create a time course of LVP length throughout each speech sample (normalized example in Figure 2A). We fit a piecewise cubic spline to the LVP length versus time data in MATLAB (The Mathworks, Inc.). We then computed the derivative with respect to time of the cubic spline and

Figure 2. Time course of normalized levator veli palatini (LVP) length (relative to resting length) and muscle velocity while a subject produces /k/ three times during the 6.6-s scan. (A) The length of the LVP shortens for the first utterance and relaxes slightly before shortening for the second and third utterances. Maximum shortening is calculated as the difference between the shortest LVP length and LVP resting length, defined per subject. (B) Muscle velocity is the time derivative of LVP length. Negative velocity indicates muscle shortening, and maximum contraction velocity is defined as the minimum calculated (most negative) muscle velocity.

evaluated the derivative at each time point in the dynamic series to determine LVP muscle velocity. Nonnormalized LVP lengths were used to calculate LVP velocities in units of millimeters per second, and normalized LVP lengths were used to calculate normalized LVP velocities in units of muscle lengths per second (see Figure 2B). Positive velocities correspond to muscle lengthening, and negative velocities correspond to muscle shortening. Therefore, maximum contraction velocity for each speech sample was defined as the minimum calculated velocity (most negative).

Midsagittal dynamic images were used to confirm VP closure during analysis of oblique-coronal images. In each subject's static, oblique-coronal image, acquired while the velum was at rest, we measured VP port depth as the linear distance from the anterior, midsagittal point on the surface of the velum to the posterior, midsagittal point in the oblique-coronal plane on the surface of the posterior pharyngeal wall. In each subject's static, midsagittal image, acquired while the velum was at rest, we made three additional anatomical measurements: pharyngeal depth, defined as linear distance between the posterior nasal spine (PNS) and the posterior pharyngeal wall along the plane of the hard palate; velar length, defined as the curvilinear length from PNS to the tip of the uvula; and sagittal angle, the angle between the line defined by anterior boundaries of the third and fourth cervical vertebrae and the line along the LVP length.

Statistical Analysis

LVP shortening and maximum LVP contraction velocities were tested for normality using the Shapiro–Wilk test. Assumptions of normality for LVP shortening and normalized shortening values were met ($p = .153$ and $p = .267$, respectively) but were violated for LVP contraction velocities and relative contraction velocities ($p < .001$ for both). However, following a logarithmic transformation, assumptions of normality were met for both contraction velocities and relative contraction velocities ($p = .443$ and $p = .604$, respectively). Mauchly's test of sphericity indicated that assumptions of sphericity had not been violated for any LVP function measure: shortening: $\chi^2(5, N = 48) = 3.115, p = .684$; normalized shortening: $\chi^2(5, N = 48) = 2.298, p = .807$; contraction velocity: $\chi^2(5, N = 48) = 1.657, p = .895$; and relative contraction velocity: $\chi^2(5, N = 48) = 1.688, p = .891$.

Differences in LVP shortening and maximum LVP contraction velocities between types of sounds were analyzed using a repeated-measures analysis of variance. Pairwise differences between types of sounds were compared using paired t tests with Holm–Bonferroni corrected critical values to minimize the effect of multiple comparisons and to control the familywise (Type I) error rate. The relationship between VP port depth and each LVP function measure (shortening or maximum contraction velocity) was analyzed using linear regression between each subject's VP port depth measurement and average LVP measure across all sounds. Similarly, the relationships between each of pharyngeal depth, velar length, and sagittal angle and LVP

shortening and maximum contraction velocity were analyzed using linear regression.

Results

LVP shortening relative to resting length varied an average of 1.7% between repeated production of the sample syllable across all speech samples for all subjects. LVP shortening measured during vowel and nasal consonant syllables was consistently lower than shortening during plosive and fricative syllable production for all but one subject. For that subject, shortening during /k/ was similar to shortening during vowel production for all three productions, and shortening was greatest during other plosive and fricative syllable production.

LVP shortening relative to resting length varied significantly between manners of production, as determined by a repeated-measures analysis of variance, $F(3, 33) = 43.86, p < .001$. Subjects demonstrated the greatest LVP shortening during production of fricative, $M = 17.8$, 95% confidence interval (CI) [14.7, 20.9], and plosive, $M = 17.5$, 95% CI [14.4, 20.6], syllables. Maximum LVP shortening values (percent resting length) measured during both plosive and fricative speech samples were significantly greater ($ps < .001$) than those during nasal consonant production, $M = 10.1$, 95% CI [7.5, 12.8], or vowel production, $M = 13.9$, 95% CI [11.2, 16.6] (see Figure 3A). The LVP shortened significantly more ($p < .001$) during vowel production compared to during nasal production. Additionally, maximum LVP shortening (in millimeters) significantly differed between each manner of production, $F(3, 33) = 39.54, p < .001$. The magnitudes of shortening were significantly different ($ps < .001$) between all pairs, except for between plosives and fricatives. Therefore, maximum LVP shortening was significantly greater during high-pressure syllable production (fricatives and plosives) than during low-pressure syllable production (vowels and nasal consonants).

A similar trend held true for LVP maximum contraction velocities; maximum contraction velocities varied significantly across the four manners of production, $F(3, 33) = 16.00, p < .001$. Relative maximum contraction velocity (muscle lengths per second) was highest during fricative syllables, $M = 0.67$, 95% CI [0.50, 0.90], followed by plosive syllables, $M = 0.62$, 95% CI [0.48, 0.81] (see Figure 3B). Maximum contraction velocities for both fricatives and plosives were greater than contraction velocities demonstrated during vowel, $M = 0.38$, 95% CI [0.31, 0.47], or nasal consonant, $M = 0.34$, 95% CI [0.25, 0.48], production. In addition, maximum contraction velocities (in millimeters per second) varied significantly between sound types, $F(3, 33) = 16.09, p < .001$. Similar to LVP shortening, maximum contraction velocity was significantly higher during high-pressure syllable production (fricatives and plosives) than during low-pressure syllable production (vowels and nasal consonants).

The relationship between each measurement and VP port depth was examined to provide insight into how LVP function relates to subject anatomy. Average maximum

Figure 3. (A) Maximum shortening, relative to resting length, for the four types of sounds (bars indicate mean $\pm$ 95% confidence interval). (B) Maximum contraction velocity in muscle lengths per second across the four types of sounds (bars indicate mean $\pm$ 95% confidence interval). *P* values and significance were determined using paired *t* tests with Holm–Bonferroni corrected critical values. ***p* < .01, ****p* < .001, *****p* < .0001.

shortening (in millimeters) across speech samples positively correlated with VP port depth, $R^2 = .89$, $F(1, 4) = 32.57$, $p = .005$ (see Figure 4A). Similarly, maximum contraction velocities (in millimeters per second) positively correlated with VP port depth, $R^2 = .82$, $F(1, 4) = 18.26$, $p = .005$ (see Figure 5A). Maximum contraction velocities also positively correlated with pharyngeal depth, though to a lesser extent than with port depth, $R^2 = .69$, $F(1, 4) = 9.02$, $p = .040$. Neither LVP shortening nor maximum contraction velocity correlated positively or negatively with sagittal angle or velar length (see Figures 4 and 5). Relative maximum shortening and relative maximum contraction velocity also positively correlated with VP port depth, $R^2 = .86$, $F(1, 4) = 23.60$, $p = .008$, and $R^2 = .71$, $F(1, 4) = 9.99$, $p = .034$, respectively.

Discussion

The purpose of this study was to develop a new method for evaluating LVP muscle behavior during real-time speech using dynamic MRI and demonstrate this method by comparing LVP function between sounds and probing the relationship between VP anatomy and in vivo LVP function. Experimental investigation into how VP anatomical variability affects LVP function was impossible without a method to measure LVP function in vivo. Our described method combines static and real-time dynamic MRI, both acquired in the plane of the LVP, to measure LVP length and velocities during syllable production. Our results demonstrate that our method is capable of determining the variation in LVP function between different manners of production, as well as providing LVP muscle shortening and velocity measures that can be used to investigate the VP structure–function relationship.

Our dynamic LVP shortening results are comparable to LVP shortening values reported in previous MRI studies. Average LVP shortening was reported to be 19% during sustained fricative production (Ettema, Kuehn, Perlman, & Alperin, 2002), similar to our average shortening of 17.8% during dynamic fricative syllable production ($SD = 4.8$).

Ettema et al. reported greatest amounts of LVP shortening during fricative production, followed by vowel and then nasal production. This study found the same progressive decrease in LVP shortening, and the real-time nature of our method enabled us to demonstrate that LVP contraction velocities are greater during fricative and plosive syllable production than during nasal or vowel production. Our shortening results also compare favorably to length changes reported during real-time production of the nonsense word *ansa*. Perry et al. (2014) reported average LVP shortening values across 10 subjects, ranging from 5% during /n/ to 16% during the final /a/ production. In our six subjects, LVP shortening ranged from 10.1% ($SD = 4.2$) during nasal production to 17.8% ($SD = 4.8$) during fricative production.

Relating LVP shortening and velocity measurements to VP port depth provided new insights into the relationship between VP anatomy and in vivo LVP function. We found that both LVP shortening and maximum contraction velocities positively correlate with VP port depth. This suggests that individuals with a shallower VP port require less muscle shortening and shortening at a slower rate than individuals with a deeper VP port do. Additionally, maximum contraction velocities positively correlated with pharyngeal depth, whereas LVP lengths and velocities did not correlate with either sagittal angle or velar length ($ps > .05$; see Figures 4 and 5). VP port depth and pharyngeal depth are both indicative of the physical distance the LVP must contract to achieve VP closure. These correlative relationships imply that a subject's VP port dimensions affect LVP muscle function and provide evidence to support the hypothesis that a range of ratios between VP portal and LVP muscle dimensions exists, outside of which healthy VP closure is not achievable (Inouye, Perry, et al., 2015; Perry, Kuehn, Sutton, Goldwasser, & Jerez, 2011; Satoh, Wada, Tachimura, & Shiba, 2002).

Given the context of the force–length relationship, our dynamic MRI-based method provides new and valuable insights into LVP muscle mechanics during speech. Our method facilitates novel functional insights during speech

Figure 4. Maximum shortening, measured in millimeters, positively correlates with velopharyngeal port depth (A) and moderately positively correlates with pharyngeal depth (B). Maximum shortening does not significantly correlate with sagittal angle (C) or velar length (D).

that are similar to insights gained using ultrasound imaging to investigate lower limb muscle mechanics during walking and other locomotion (Brennan, Cresswell, Farris, & Lichtwark, 2017; Cronin & Lichtwark, 2013; Lichtwark, Bougoulas, & Wilson, 2007). These in vivo imaging methods are required to evaluate typical muscle behavior and its effect on function in healthy and pathological populations. During speech production, when the LVP contracts

Figure 5. Similar to maximum shortening, maximum contraction velocity, measured in millimeters per second, positively correlates with velopharyngeal port depth (A). Maximum contraction velocity also significantly positively correlates with pharyngeal depth (B) but does not significantly correlate with sagittal angle (C) or velar length (D).

and shortens to close the VP port, the muscle moves away from optimal length, reducing force-generating capacity. When less shortening occurs, due to either the type of sound or the speaker's VP anatomy, the LVP is operating near the peak of the force-length curve. The reduction in LVP force-generating capability is minimal for our observed range of shortening (5.6%–26.3% across all subjects; see Figure 6A) if we assume that optimal fiber length occurs at resting

Figure 6. Curves demonstrating two fundamental relationships of skeletal muscle. (A) Force–length relationship. Active muscle force is maximized at optimal fiber length. Active force-generating potential decreases as the muscle shortens or lengthens from optimal fiber length. At 26.3% shortening from optimal fiber length (the maximum demonstrated in our subjects), levator veli palatini (LVP) active force-generating capability is approximately 13% less than maximum active force. (B) Force–velocity–power relationship. A muscle produces maximum muscle force when muscle contraction velocity is zero. As a muscle shortens more quickly, it can produce less force. A muscle can produce no force when contraction velocity reaches maximum velocity ($v_{\max}$). Muscle power, defined as Force $\times$ Velocity, is maximized when contraction velocity is $\sim 0.3v_{\max}$. We estimated LVP $v_{\max}$ from levator fiber type distribution and muscle fiber type $v_{\max}$ s (Larsson & Moss, 1993; Moon, Thompson, Jaeckel, & Canady, 1998). For the demonstrated range of LVP contraction velocities (0.39–0.74 muscle lengths per second), muscle force is decreased between 73% and 88% from maximum. However, at these contraction velocities, the LVP is producing near-maximum power.

length, similar to previous studies (e.g., Inouye, Pelland, Lin, Borowitz, & Blemker, 2015). However, the ascending limb of the force–length curve becomes very steep as fibers continue to shorten from optimal fiber length. As the LVP muscle shortens more substantially, force-generating potential sharply decreases. Further studies are needed to determine how much the LVP shortens in individuals with VPD and if that level of shortening results in a meaningful reduction in muscle force generation.

Our method provides measurements of LVP contraction velocities during speech, which are functionally meaningful due to the force–velocity relationship of muscle. As muscle velocity increases, the force produced decreases until a maximum velocity ($v_{\max}$) is achieved at which no force can be produced. $v_{\max}$ of the LVP is unknown, but using the fiber type distribution (Moon et al., 1998) and known $v_{\max}$ for each fiber type (Larsson & Moss, 1993), we estimate LVP $v_{\max}$ as 1.5 lengths per second. In our limited speech sample, the LVP operated at contraction velocities between 0.39 and 0.74 muscle lengths per second. Contraction velocities of this magnitude lead to substantial

reductions in LVP force-generating potential (see Figure 6B). For effective production of sounds with higher contraction velocities, the LVP would need greater activation to achieve the force required, leading to possible muscle fatigability, especially in individuals with shorter and smaller LVP muscles. There is a trade-off, however, between muscle force generation and power production. Muscle power, that is, the product of force and contraction velocity, is maximized near $0.3 v_{\max}$. In our group of healthy adults, the LVP is operating close to $0.3 v_{\max}$ for many of the sounds included, leading to near-maximal power output. The contraction velocities observed in healthy speakers yield enough force to achieve sufficient closure for plosive and fricative sounds while remaining near peak power. However, LVP contraction velocities and their effects on force generation, power production, and VP function in individuals with VPD are unknown and warrant investigation.

Limitations

A critical limitation of this study is the dynamic imaging frame rate of 18.2 fps. Velar elevation can occur in or under 100 ms (Kuehn, 1976), making the minimum frame rate for effective imaging of the VP mechanism at least 10 fps (Narayanan, Nayak, Lee, Sethy, & Byrd, 2004; Perry et al., 2014; Sutton, Conway, Bae, Seethamraju, & Kuehn, 2010). Our frame rate of 18.2 fps is well above that minimum, but there is the possibility that our images missed the exact instant of maximum LVP shortening. This would result in possible random underestimation of true LVP shortening values and possible addition of noise to our measurements. Despite this possibility, we were able to capture differences between sounds representing different manners of production greater than the potential noise. As stated above, our LVP shortening results compare favorably with shortening values determined from dynamic scans at 30 fps (Perry et al., 2014), giving us confidence that our method is able to capture LVP behavior trends. Additionally, our reported contraction velocity values are average velocities over the 55 ms between frames, rather than instantaneous velocities, so our reported values could underestimate true maximum LVP contraction velocities. Future studies would benefit from a higher frame rate to minimize the time over which the velocity measurement is averaged, moving closer to a true instantaneous velocity measurement. A higher frame rate would be especially important when examining specific phonemes in various contexts, such as differentiating LVP behavior in a connected speech sample.

There are other limitations of this study that should be mentioned. First, the oblique-coronal plane containing the length of the LVP must be chosen appropriately during scanning; otherwise, it is impossible to make accurate LVP muscle length measurements. In healthy anatomies, these planes can be found in a straightforward manner using anatomical landmarks. However, it is unknown whether a plane containing the entirety of the LVP exists in individuals with VPD or how systematically it can be determined during scanning.

Second, the LVP is estimated as the union of two line segments, a simplification of the LVP path in healthy individuals, and possibly an oversimplification in pathological populations. However, our measurements of healthy LVP shortening were comparable with previously reported length measurements. Manual image registration and segmentation require a substantial time commitment for data analysis; future work is necessary to automate these procedures. Therefore, we sought a balance between anatomic accuracy and time burden for analysis. LVP geometry has been simplified to reduce computation time in previous computational studies (Berry, Moon, & Kuehn, 1999; Inouye, Pelland, et al., 2015; Srodon, Miquel, & Birch, 2012), including one with a two-segment representation of the LVP (Inouye, Perry, et al., 2015), and these studies nevertheless provided meaningful insights into the relationship between VP anatomy and function. The other VP muscles could affect the VP structure–function relationship; in particular, the musculus uvulae can act agonistically to the LVP as a velar extensor and reduce the burden of the LVP during VP closure (Inouye, Lin, Perry, & Blemker, 2016; Kuehn, Folkins, & Linville, 1988). Without the musculus uvulae, we found positive correlative relationships between LVP function and VP portal dimensions, but inclusion of the musculus uvulae in our analysis could help explain the variability in our correlations and should be considered in future studies.

Finally, the speech sample in our protocol included only four manners of production and a mix of voiced and voiceless consonants. As with all consonant–vowel productions, there is a co-articulatory effect (Bell-berti, Baer, Harris, & Niimi, 1979), and the results of our consonant–vowel samples could vary with inclusion of different vowels. However, the purpose of our study was to introduce a new method capable of measuring in vivo LVP function, and our results support hypotheses that LVP function differs significantly between manners of production and provided insight into how VP anatomy affects LVP function. Future studies should consider inclusion of multiple vowels in consonant–vowel samples to examine differences in production and collect audio recordings during image acquisition that can be further analyzed, for example, spectrographic analysis, to determine phoneme-specific levator lengths and velocities.

Conclusions

This study introduces a method for measuring in vivo LVP function, which promotes an increased understanding of the relationship between VP anatomy and LVP function. Our method can be applied to a variety of real-time speech samples, from isolated syllable utterances to full sentence production, and our results augment the limited existing literature reporting LVP shortening during speech. We also presented, to our knowledge, the first evaluation of LVP muscle shortening velocity during speech and laid the groundwork for experimental investigation of VP structure–function relationship. Results from our limited samples

and sample population suggest that certain VP anatomical dimensions strongly influence in vivo LVP function and, consequently, achievable force generation and overall VP function. Future studies should explore LVP behavior and its relationship with VP anatomy with a more comprehensive speech sample in both healthy speakers and individuals with VPD. The method presented here could be used to elucidate this relationship, as well as compare LVP function between healthy and clinical populations.

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