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Contributions of the musculus uvulae to velopharyngeal closure quantified with a 3D multi-muscle computational model

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Abstract

The convexity of the dorsal surface of the velum is critical for normal velopharyngeal (VP) function and is largely attributed to the levator veli palatini (LVP) and musculus uvulae (MU). Studies have correlated a concave or flat nasal velar surface to symptoms of VP dysfunction including hypernasality and nasal air emission. In the context of surgical repair of cleft palates, the MU has been given relatively little attention in the literature compared with the larger LVP. A greater understanding of the mechanics of the MU will provide insight into understanding the influence of a dysmorphic MU, as seen in cleft palate, as it relates to VP function. The purpose of this study was to quantify the contributions of the MU to VP closure in a computational model. We created a novel 3D finite element model of the VP mechanism from MRI data collected from an individual with healthy non-cleft VP anatomy. The model components included the velum, posterior pharyngeal wall (PPW), LVP, and MU. Simulations were based on the muscle and soft tissue mechanical properties from the literature. We found that, similar to previous hypotheses, the MU acts as i) a space-occupying structure and ii) a velar extensor. As a space-occupying structure, the MU helps to nearly triple the midline VP contact length. As a velar extensor, the MU acting alone without the LVP decreases the VP distance 62%. Furthermore, activation of the MU decreases the LVP activation required for closure almost three-fold, from 20% (without MU) to 8% (with MU). Our study suggests that any possible salvaging and anatomical reconstruction of viable MU tissue in a cleft patient may improve VP closure due to its mechanical function. In the absence or dysfunction of MU tissue, implantation of autologous or engineered tissues at the velar midline, as a possible substitute for the MU, may produce a geometric convexity more favorable to VP closure. In the future, more complex models will provide further insight into optimal surgical reconstruction of the VP musculature in normal and cleft palate populations.

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Introduction

Although the biomechanical properties are less understood, the significance of the musculus uvulae (MU) for normal velopharyngeal (VP) function during speech has been well established.[1,2]. The levator veli palatini (LVP) is the primary muscle responsible for velar retraction and elevation during speech. The dorsal convexity of the velum is attributed to the MU and is important in VP closure. The MU functions to add stiffness to the velum and to rotate the velum toward the posterior pharyngeal wall.[3] The noncleft MU has been examined in dissection[2,4–6], histological[7–11], electromyography (EMG)[3], and MRI[12,13] studies. Studies in adults and infants with cleft palate[1,14] and submucous cleft palate[15,16] have demonstrated consistent abnormality in the nasal surface of the velum. Studies have correlated a concave or flat nasal velar surface to symptoms of VP dysfunction including hypernasality and nasal air emission[1,15,17]. Pigott and colleagues[1] proposed this lack of nasal surface bulge during speech to be attributed to MU atrophy, possibly due to surgical denervation while others have suggested a void of muscle fibers. Huang et al. (1997)[4] suggested the variations in the nasal surface of the velum observed by others[15,17] may be attributed to dysmorphology in the LVP muscle in addition to variations in the MU. The authors propose the need for interoperative documentation and studies to demonstrate variations in the MU in cleft palate. The MU and LVP collectively contribute to adequate midline nasal velar bulk. Studies have suggested individuals with repaired cleft palate present with significantly thinner LVP muscle bundles.[18] Therefore, in addition to the possible dysmorphology or absence of the MU in cleft patients, it is important that consideration be given the functional significance of the MU as it relates to the effect on the nasal velar surface.

While its significance in normal VP function is well established, the significance of the MU in cleft palate surgical repair has been controversial[8], and it has been described as the most enigmatic muscle in the human speech apparatus[4]. The MU is quite small compared to the LVP muscle, the primary muscle for elevating the velum. In contrast, the LVP is given the most attention in muscle dissection during cleft palate surgical repair. Although the function of the MU is secondary to the LVP in VP closure[9], its mechanical action is relevant to normal velar function in speech and swallowing. A greater understanding of the function of the MU may engender the development of surgical techniques to properly reconstruct the MU or help to compensate for its absence or hypoplasic nature often observed in individuals with cleft palate[15,17].

This study seeks to examine the functional descriptions of the MU during speech using a finite-element model. Landes et al. (2011) suggested that the proportional impact of the LVP and MU may be impossible to differentiate[9]. While this may be true using experimental approaches (e.g., histology, dissection, or MRI), computational modeling provides a unique and powerful method to isolate the effects of individual muscles – like the MU – on VP function. Our objectives in this study were to i) quantify VP closure variables with different morphological and muscle activation scenarios, ii) compare these variables with experimental data, iii) quantify the effect of the MU in isolation, and iv) quantify the effect of a midline palate defect. Accomplishment of these objectives provides insights into the

potential benefits of a proper MU reconstruction or other surgical methods for compensating for an absent or hypoplastic MU.

Methods

Finite element model

We created a 3D finite element model of the VP mechanism based on static MRI scans of a 20-year-old Japanese male subject with healthy VP anatomy and compared velum deformations with dynamic MRI scans[19–21]. The model components included the velum, the PPW, the LVP, and the MU (Fig. 1). Simulations were based on the muscle and soft tissue mechanical properties from the literature[22,23]. Muscle material properties from a previous study[22] were implemented, containing both passive (related to passive muscle stretch) and active (related to muscle activation and muscle stretch) nonlinear tensile components. The active tensile component incorporates the known force-length behavior of skeletal muscle[24], where the peak active muscle force occurs when the muscle reaches its optimal length. The velum was modeled as an isotropic, hyperelastic Mooney-Rivlin material with elastic modulus of 25kPa and muscle was modeled as a transversely isotropic material[23]. Muscle fiber trajectories for the muscle material were determined using computational fluid dynamics. We used the Sefea^{TM} (Strain-Enriched FEA, AMPS Technologies) finite element solver with explicit strain energy function specification and automatic meshing of 4-node enhanced tetrahedral elements.

Simulations

We created four unique models representing variations in the velum and MU (Fig. 2). Specifically, the four morphologic categories were i) noncleft single subject-specific model (“Long MU model”), ii) noncleft single subject-specific model with an MU that is modified to a shorter length (“Short MU model”), iii) noncleft single subject-specific model with the MU removed (“No MU model”), and iv) noncleft single subject-specific model with the MU removed and a midline velar defected added (“Midline defect model”). In the healthy noncleft single subject-specific model, MU was 3cm long, based on previous histological studies, where the MU extends down to the uvular base[5,7,11]. The diameter of the MU at the middle was 2.5mm, based on a previous study[3], and the thickness tapered down as it coursed distally (to simulate that the fibers become more dispersed distally[5]). The short MU model was created by effectively trimming the MU both proximally and distally so that it was placed above the LVP; its length was 60% less than the normal MU. Representation of a short MU to be 60% was based on MRI data obtained from five adults with repaired cleft subject (example images shown in Fig. 2A–C). Of these subjects, some presented with a relatively longer MU (Fig. 2A), some with a shorter MU (Fig. 2B), and some with no MU (Fig. 3B). A decrease of 60% represented the average across the subjects. We also simulated an absent MU with and without a midline defect in the velum, designed to depress the velum at the midline a distance equal to the thickness of the MU if present (2.5mm).

We performed three series of simulations. The first series of simulations were designed to investigate the effect of the MU in the context of maximal LVP activation. We maximally activated the LVP, with and without activation of the MU, with the “Long MU” and “Short

MU" models. Our second series of simulations were designed to investigate the effect of an absent MU, with and without a nasal velar defect. In these simulations, we maximally activated the LVP with the "No MU" and the "Midline defect" models. The last series of simulations were designed to investigate the extent to which the MU could assist in closure when the LVP is not maximally activated. In these simulations, we maximally activated the MU, and then determined the LVP activation required to achieve closure. These simulations were performed with both the "Long MU" and "Short MU" models.

Measurement of simulation variables

We measured variables of velar elevation, minimum VP distance, and velar to PPW contact length (Fig. 3) for each simulation at muscle activation levels ranging from inactive to fully active, smoothing the variables to remove numerical noise. Minimum VP distance was defined as the minimum distance from the velum to the PPW at the velar midline (Fig. 3A). Velar elevation was defined as the vertical distance of the center of the LVP belly at the velar midline between rest and elevated velar positions (Fig. 3B). Velar contact length was determined as the length of contact between the velum and the PPW (Fig. 3B).

Results

In the absence of a defect, the simulations demonstrated that activation of the LVP (Fig. 4), with or without activation of the MU, produces features characteristic of normal velum elevation and contact length observed during speech dynamic MRI data (Fig. 5): a raised velum retracted against the PPW with the velar knee positioned at the location of the LVP sling[25,26]. This result is consistent with the current understanding that the LVP is the primary mover of the velum, in the context of a healthy non-cleft velopharyngeal anatomy.

The presence of the velar defect significantly affected the ability for the LVP to achieve closure. The defected decreased contact length by 62% compared with the other simulations that involve LVP activation (Fig. 5A). For some muscle activations, the defect creates a small opening in the VP port (Fig. 6). This result points to the "space occupying" function of the MU tissue.

Simulations of 100% activation of the MU with low levels of LVP activation demonstrated that the MU has the capacity to assist the LVP in VP closure. The MU activated alone served to move the velum towards the PPW: activation of the MU in the long MU model decreased the VP distance by 75%, while in the short MU decreased the VP distance by 30% (Fig. 5C). When there is no MU activation, 19% activation of the LVP is required to achieve VP closure (Fig. 7A). By contrast, when the MU is activated to 100%, only 3% LVP activation is required to achieve closure in the long MU case (Fig. 7B), and 8% LVP activation is required in the short MU case (Fig. 7D). Notably, the shape of the velum upon closure differs in these cases: when only LVP is used for closure there is a defined velar "knee" (Fig. 7A and C), while when MU activation is used to assist in closure, the velum is extended (Fig. 7B and D). These results suggest that when the LVP function is compromised (as is often the case in VPI), the MU has the capacity to assist in closure, albeit with a different velum shape at closure.

Discussion

In this study, we created a novel 3D computational model f the VP mechanism based MRI data to investigate the functional role of the MU during speech. While the action of the MU is considered secondary to that of the primary contributions provided by the LVP, the results from our model suggest that the consideration of the MU during surgery can potentially lead to better VP functioning. The model illustrated two important potential roles for the MU. First: through its action as a velar extensor, it can assist in VP closure, in particular in the context of low levels of LVP activation, which could be the case following cleft repair in some subjects. Second: through its space-occupying function[1,15,17,3–6,27], presence of MU tissue provides convexity of the velar surface and thus prevents small closure defects that can arise even in the context of a fully functioning LVP.

In the present study, the simulations demonstrated the extensor role of the MU, similar to the flexible beam model explained by Kuehn et al. (1988)[3]. Specifically, that study described the velum as a two-layered flexible beam with the varied compressional forces. In this model, compression of the upper beam (representing the nasal velar surface) causes extension of the moveable end (representing the velum). The study used this modeling system to demonstrate the role of the MU in providing extension of the velum for creating a seal between the velum and the posterior pharyngeal wall. Our model in the present study further confirms the flexible beam model using computational modeling with physiologically relevant geometry.

Our results show that the extensor function of the MU has the potential role of assisting the LVP in closure at lower muscle activation. It has been shown that individuals with repaired cleft palate demonstrate relatively greater physiological effort for LVP contraction during speech compared to noncleft adult subjects[28,29]. The authors proposed several possible explanations for increased muscle effort required for cleft palate speakers. Individuals functioning at the upper boundary of their muscle capabilities are at risk of fatigue that may be demonstrated in hypernasal speech and/or marginal or borderline VP dysfunctional cases[29]. Our findings suggest an added operational disadvantage that increases the risk of overall VP fatigue in individuals with cleft palate. Specifically, the condition of absent or nonfunctional MU and abnormal nasal velar surface together appear to be highly related to VP dysfunction. Surgically decreasing VP distance may compensate for loss or dysfunction of the MU (e.g., inducing LVP muscle overlap and sling tightening[23,30–33]). Furthermore, successful repair of the MU could help compensate for weakness or dysfunction of the LVP. Lastly, it appears that while a longer MU is better than a short or hypoplastic MU, any amount of MU tissue at the midline is beneficial.

The significance of the space-occupying function of the MU is that other tissue may be used to produce a favorable convexity on the nasal velar surface if the MU is absent. This can be done with autologous tissues such as fat and muscle. Another option is to implant artificial or engineered tissues to occupy space at the velar midline. The absence of tissue at the location of the normal MU creates a midline defect, shown to be significantly detrimental to VP closure in our study. Research on surgical outcomes from varied techniques should examine the impact of maintaining MU volume and preventing midline palatal defects, such as a concave or flat nasal velar surface.

The potential benefits of a properly reconstructed MU are sacrificed in some procedures. For instance, the double-opposing Z-plasty[34] does not enable the reconstruction of the MU, and the transposed tissue flaps lead to an irregular MU position that is not within the velar midline[4]. Kuehn et al. (1988) discussed the importance of the MU by emphasizing that it is the only muscle running longitudinally and therefore is primarily responsible for any compression of the velar surface. Surgeries that do not restore the longitudinal direction of the MU fibers may therefore, lose the ability to benefit from this type of compressional force, limiting velar extension.

It has been observed that the MU and LVP are consistently coactivated during speech[3]. While the MU coactivation with full activation of the LVP did not affect variables of velar elevation or closure force to a great extent (Fig. 4), the MU transfered VP contact pressure downward on the PPW due to its role as a velar extensor, resulting in more uniform contact pressure on the PPW along the posterior portion of the velum (Fig. 8). This extension during velar elevation has been hypothesized to enhance midline velopharyngeal contact[4].

While this study has sought to elucidate the function of the MU, several modeling assumptions were used that should be considered. Our model did not include additional VP muscles such as the pharyngeal constrictors, palatoglossus, or palatopharyngeus. We assumed that muscle sarcomere lengths would be optimal at rest; however, the resting sarcomere lengths of the LVP and the MU are currently unknown, particularly in the cleft palate population. Modeling parameters such as specific muscle tension and soft tissue stiffness have influence on the exact quantitative results, but comparatively less influence on the observed qualitative results, similar to a previous finite-element study of VP function[23]. We have not considered dynamics or viscoelasticity in this study; however, these considerations should certainly not contradict the roles of the MU as a space-occupier and velar extensor or the maximum potential to achieve closure. However, these dynamic factors may be influential in the time-varying deformation of the VP complex during speech; this is an area deserving future research.

In spite of these limitations and assumptions, our results strongly support the conclusion that the MU functions as both a space-occupying structure and a velar extensor. This study demonstrated these functions described by previous anatomical studies using computational modeling and by exploring the separate roles of variations in the MU in VP function. Predictive analyses, such as this, cannot be appreciated *in vivo* or through dissection studies. Our modeling approach enables systematic testing of hypotheses about the MU relevant to surgical procedures.

Children born with a cleft palate, either unrepaired or even after repair, clearly have a disadvantageous VP mechanism. Velopharyngeal closure as reflected in speech quality is a continuous variable, rather than an all or none phenomenon. Any and all potential advantages gained during the surgical repair of the cleft palate, especially in cases of borderline VPI, take on critical significance. This study confirms the role of the MU in the overall VP mechanism and gives strong rationale for either repairing or replacing the MU in all cases where cleft palate repair is performed. Future use of computational studies involving more muscles relevant to VP functioning, such as the palatopharyngeus and tensor

veli palatini, will improve the mechanistic understanding of VP muscle function and lead to additional beneficial treatment strategies for patients with a cleft palate or VP dysfunction.

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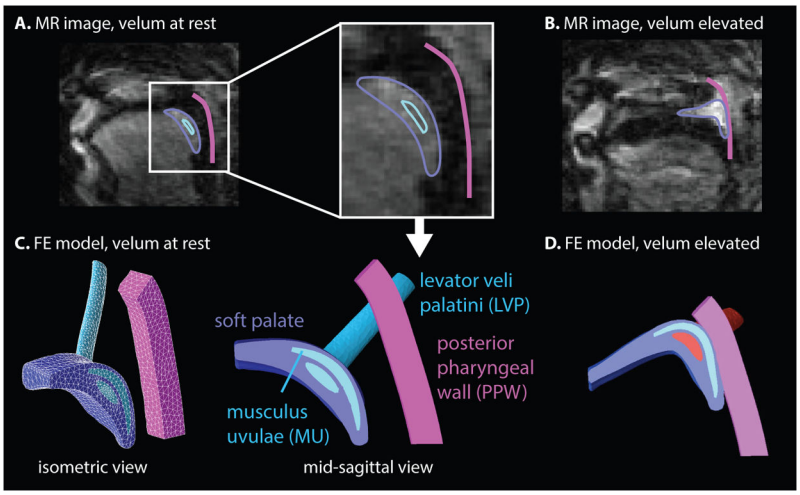

Figure 1. Creation of finite-element models from MRI data

Using both static and dynamic MRI data as a basis (A), we created a 3D finite element model of the velopharyngeal mechanism with two muscles: the levator veli palatine (LVP and the musculus uvulae (MU). We ran simulations that included activation of the LVP and MU muscles (D–shown here is a simulation of activation of only the LVP), and we then compared the deformations predicted by the model with dynamic images in the velum-elevated position (B). The simulations produce features characteristic of normal velum elevation observed during speech dynamic: a raised velum retracted against the PPW with the velar knee positioned at the location of the LVP sling[25,26].

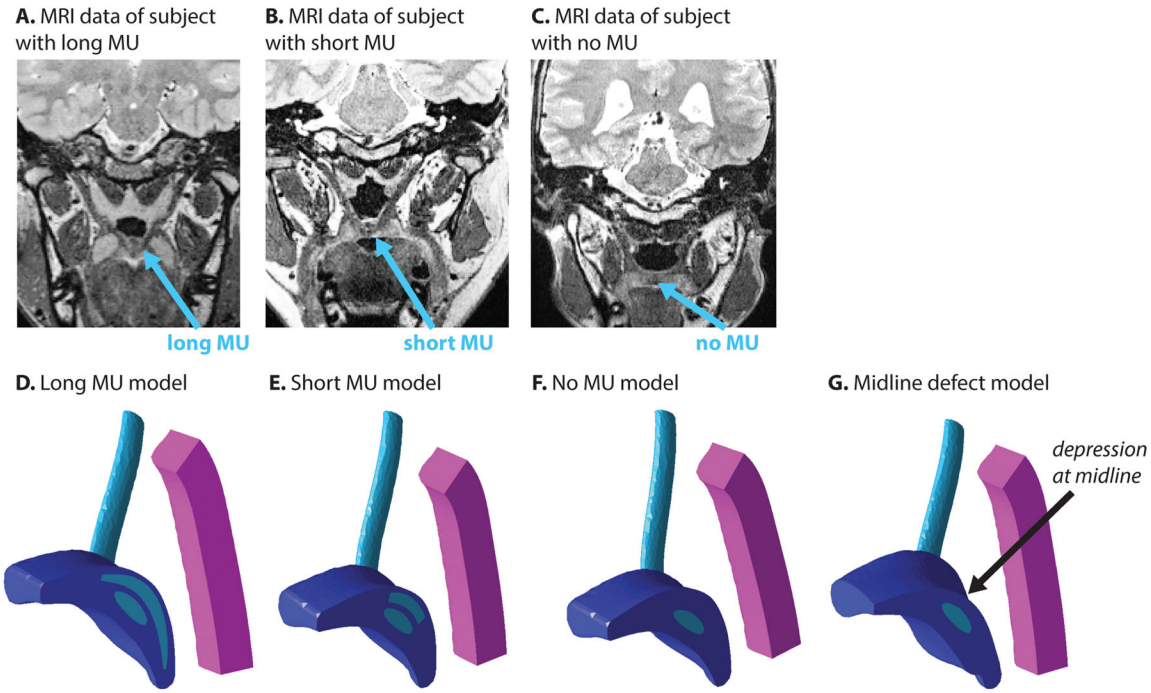

Figure 2. Definition of model variations

MRI data (A–C) demonstrate variations in the length and anatomy of the MU across individuals who have had cleft repair. In order to explore the effect of variations in MU anatomy, we modeled four morphological scenarios for the MU to quantify its contribution to VP function: the first model included a long MU (D), the second model a included short MU (E), the third model had no MU with the space replaced with velum tissue (F), and the last model had no MU with the space not replaced by other tissue resulting in a defect in which the midline of the velum depressed (G).

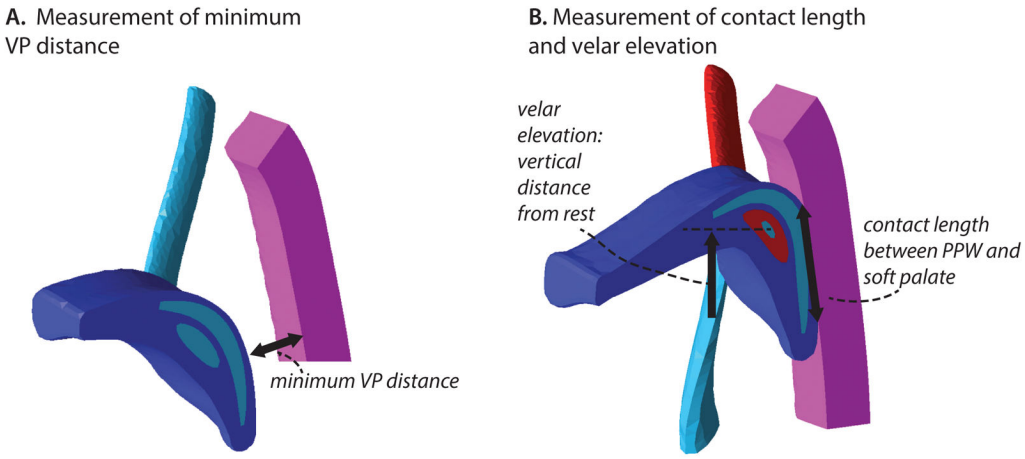

Figure 3. Measurement of closure variables from the models

We measured three VP closure variables for each simulation: minimum VP distance (A), velar elevation (B), and contact length (B).

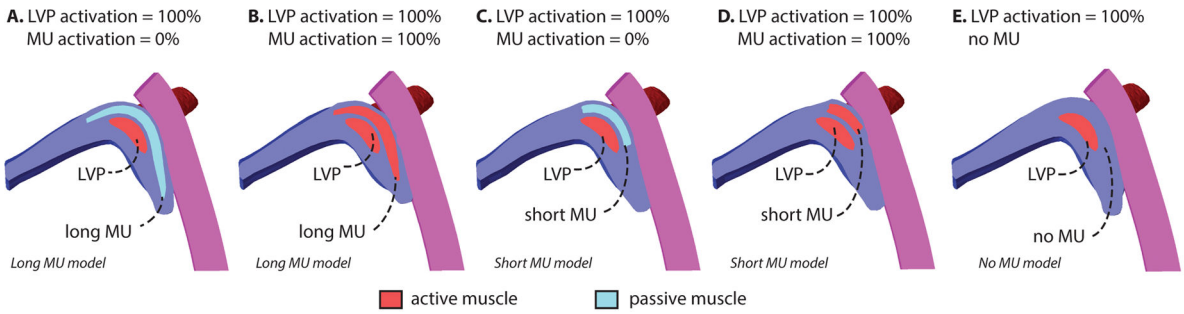

Figure 4. Simulations involving full LVP activation

Multiple combinations of simulations in which the LVP was active, with (B,D) and without (A,C,E) activation of the MU, demonstrate very similar closure characteristics of the velum.

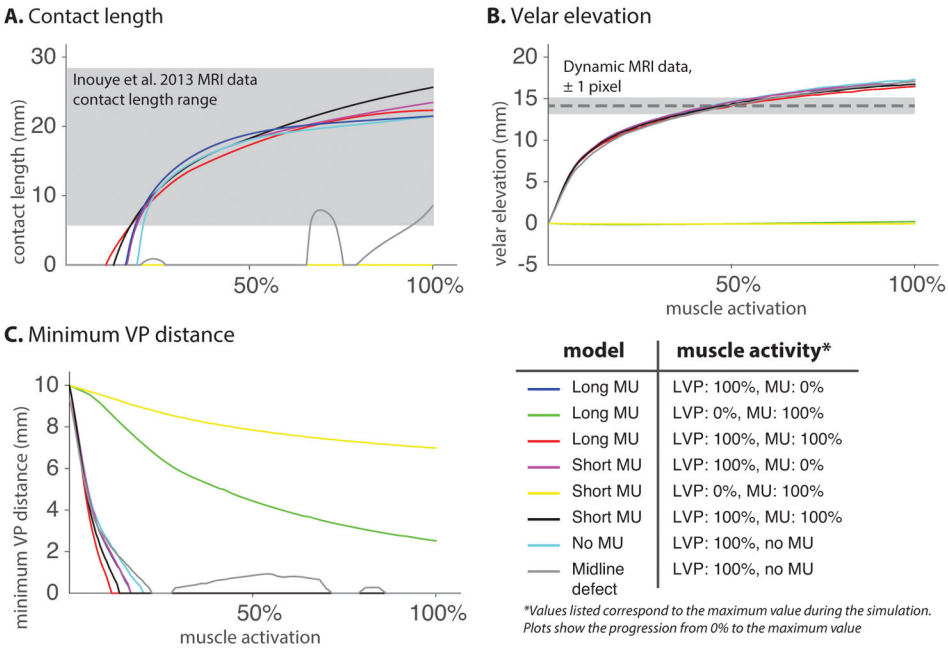

Figure 5. Measurement of closure patterns from the finite-element model as compared to image data

Physiologically normal contact lengths (A) were achieved when the LVP was fully active, with the exception being the case of midline velar defect (gray line). In the midline defect model, the contact length is decreased by 62% compared with other simulations that involve LVP activation. Physiological velar elevation (B) is achieved with all simulations with full LVP activation. The LVP acting alone achieved VP closure (C) at 17% activation, while only 12% activation was necessary when the MU was also activated at the same level.

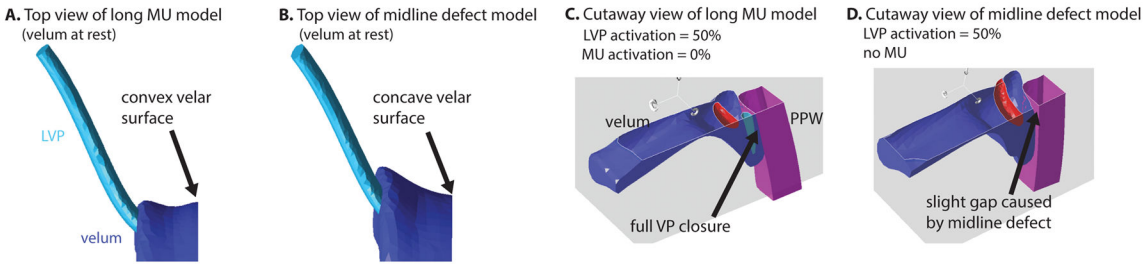

Figure 6. Illustration of closure in the model that included a midline defect

The model with a midline defect had a concave velar surface (B) as compared to a convex velar surface (A) in the no-defect models. As a result, the midline defect model had small closure gaps (D), while the no-defect models had no closure gaps (C).

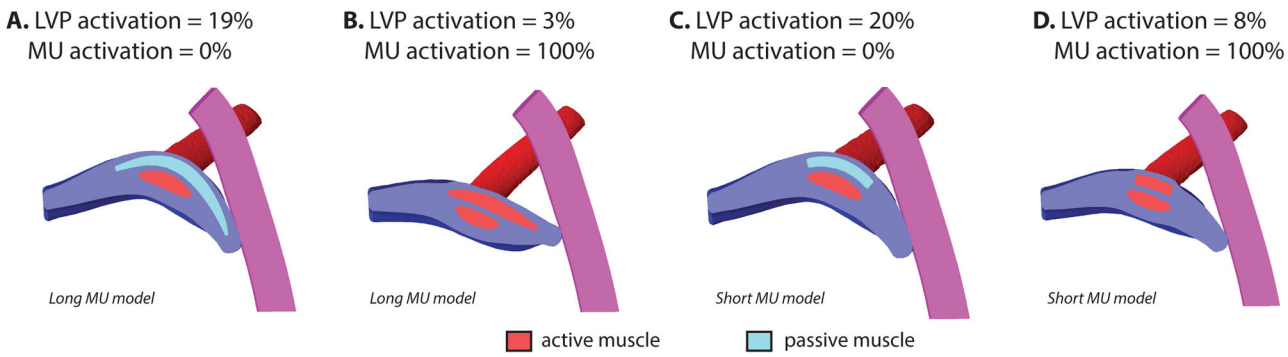

Figure 7. Comparison of closure characteristics when the MU is activated to assist in closure

Without MU activation, the model required 19–20% activation of the LVP (A,C) to achieve closure. However, in the presence of full activation of the MU, the long MU model only required 3% activation (B) to achieve closure and the short MU model only required 8% activation (D) to achieve closure, demonstrating the ability for the MU to assist in closure when the LVP is not fully functional.

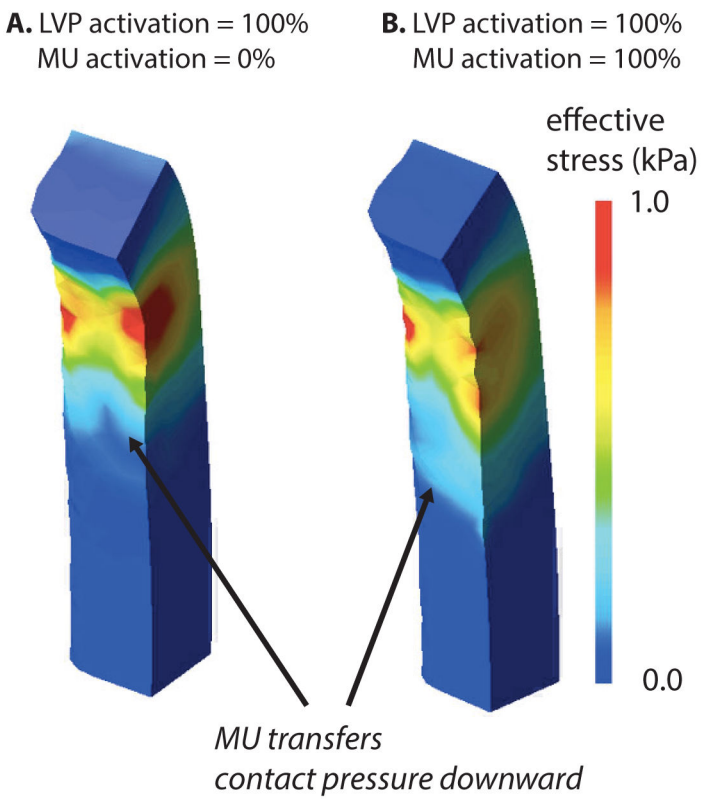

Figure 8. Comparison of contact pressures under full LVP activation, with (B) and without (A) full MU activation

The MU transfers VP contact pressure downward on the PPW due to its role as a velar extensor, resulting in more uniform contact pressure on the PPW along the posterior portion of the velum.