

BIOMECHANICAL SIMULATION OF LATERAL ASYMMETRY IN TONGUE BRACING

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1 Introduction

Tongue bracing occurs when the lateral edges of the tongue maintain contact with the palate or upper molars. Bracing is pervasive in speech: previous electropalatogram (EPG) data analysis shows lateral contact is consistently maintained during speech [1]. This research has also indicated the presence of *lateral bias* or *asymmetry* in the tongue: a consistent tendency to release contact more on one side than the other, both in unilateral release and in sequential loss of contact during a bilateral release. The present study explores the muscle activations that underlie asymmetry in lateral tongue bracing through biomechanical simulations. Based on biomechanical simulations [2], certain muscles have been previously identified as *bracing agonists* (e.g., posterior and medial genioglossus (GMP, GGM), mylohyoid (MH), verticalis (VERT), and superior longitudinal (SL)) and *antagonists* (anterior genioglossus (GGA), styloglossus (STY), hyoglossus (HG), transverse (TRANS), and inferior longitudinal (IL)). Agonists tend to raise or widen the tongue and therefore increase the likelihood of bilateral bracing, while antagonists tend to lower or narrow the tongue and decrease the likelihood of bilateral bracing. This paper presents a set of biomechanical simulations using a 3D finite-element model of the vocal tract that explore how unilateral bracing can be achieved by asymmetric activation of agonists, antagonists, or a combination of the two. The results of these simulations suggest that unilateral bracing may be instantiated primarily by contra-lateral muscle activation/deactivation from the side on which bracing contact is maintained, providing a starting point for further exploration of lateral tongue dominance.

2 Materials & Method

To explore the relationship between laterally asymmetrical muscle activation and unilateral tongue bracing, we conducted a series of simulations using the Artisynt biomechanical modeling platform [3]. Artisynt uses a combination of dynamic rigid body and finite-element modeling to simulate the hard and soft structures that make up the vocal tract. Artisynt can perform feedforward simulations where time-varying muscle activations are input to the model, and the resulting kinematic and contact behavior can be observed.

2.1 Data Collection and Processing

For the current simulations^a, we used the model of the tongue, jaw, palate and hyoid complex used in [2] (Fig. 1). The model has 96 virtual contact sensors affixed to the hard palate in a similar configuration to the Kay EPG [1]. These sensors can be used to detect tongue-palate contact, including lateral bracing.

The simulations in the present study were based on the simulations from [2], which investigated how laterally symmetrical muscle activation can produce bilateral bracing at different degrees of jaw closure. We used the muscle activations that achieved bilateral bracing with a jaw aperture of 5mm (n=632) as a starting point. For each of these original simulations, we ran four new simulations where we varied the activation of different sets of muscles on the left side of the vocal tract: (1) Agonists and antagonists activated at 100% of the original activation, a replication of the successful bracing simulations in [2]; (2) Agonist activation at 50%; (3) Antagonist activation at 150%; (4) Both 2 and 3.

In all cases, the jaw adductors were activated to reduce midsagittal jaw aperture to 5mm. Lateralized muscle activations were only changed on the left side. Right lateral muscles were kept at the original activations from the simulations in [2]. This approach was based on observations that tongue musculature is symmetrical [4] and so would demonstrate similar behavior if the right side was used instead.

Following [2], we designated GMP, GGM, MH, VERT, SL as bracing agonists, and GGA, STY, HG, TRANS, IL as bracing antagonists. Although SL was also classified as an agonist in [2], it is not lateralized in the Artisynt model, and so its activation level was not varied along with the other agonists.

Each simulation lasted a second. In the first 100 ms, the jaw adductors were linearly activated to produce a 5mm jaw aperture. In the next 700ms, the tongue muscles were linearly activated to their maximum levels. In the final 200ms, the model was left to stabilize and any tongue-palate contact was recorded. The simulations were run using Artisynt's BatchSim tool.

2.2 Data Analysis

Statistical analysis was done using a pair of logistic regression models, which predicted left and right lateral bracing contact respectively. The independent variables in each model were left-side agonist activation (50% or 100%), left-side antagonist activation (100% or 150%) and their interaction. These were normalized to Z scores.

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Simulation code and data: https://github.com/connormayer/asymmetrical_bracing

3 Results

Of the 2528 total simulations, 2083 were successful. The remaining 445 simulations failed due to numerical errors.

Results from the simulation study are displayed in **Fig. 2** below. Here, ‘Agonists: 1 Antagonists: 1’ is the condition with symmetrical muscle activations that reproduces the results from [2], while the other three conditions modify the muscle activations on the left side relative to this condition. These results show that reducing agonist activation on the left side reduces bracing on both sides, but the effect is stronger on the contralateral side. Increasing antagonist muscle activation on the left side has a similar effect, although the asymmetry in bracing contact is not as pronounced.

The model predicting right-side bracing outcomes revealed a significant positive effect of agonist activation ($\beta=2.44$, $Z=13.135$, $p < 0.001$), a significant negative effect of antagonist activation ($\beta=-1.32$, $Z=-6.879$, $p < 0.001$), and a significant negative interaction between the two ($\beta=-1.12$, $Z=-6.082$, $p < 0.001$). The model predicting left-side bracing outcomes showed similar effects of agonist activation ($\beta=1.19$, $Z=11.166$, $p < 0.001$), antagonist activation ($\beta=0.76$, $Z=-6.815$, $p < 0.001$), and their interaction ($\beta=-0.629$, $Z=-5.973$, $p < 0.001$).

These results show that increasing agonist activation on the left side increases the likelihood of a bracing outcome on both sides, while increasing antagonist activation on the left decreases the likelihood of a bracing outcome on both sides. However, the effect is stronger on the contralateral side: increased activation of agonists on the left side causes a greater increase in bracing outcomes on the right side relative to the left, and increasing antagonist activation on the left causes a greater decrease in bracing outcomes on the right side than on the left. The interaction terms suggest a small but significant negative superadditive effect of activating both agonists and antagonists.

4 Discussion & Conclusion

The outcomes of the simulation study have provided insight into the relationship between unilateral bracing and muscle activation patterns. Both reducing agonist activation or increasing antagonist activation on the left while keeping activations constant on the right results in lower tongue-palate contact on the right side. Hence, unilateral release appears to be affected by muscle activations on the opposite side. This can be explained by considering the muscular-hydrostatic properties of the tongue. Activating a muscle unilaterally means contracting it, squeezing that side of the tongue and thus expanding the other side due to the volume-preserving nature of muscular hydrostats [5]. This would cause the opposite side to make more contact with the palate, not the same side, as shown in the tongue diagram in **Fig. 3**. One intriguing inconsistency arises when considering the case where increasing antagonist activation reduces contact on the opposite side. Hydrostatic properties would predict contact to increase, but in this case it may be attributed more to the way in which extrinsic antagonists (e.g. STY, HG) tend to pull the tongue down or move it to the left side rather than affecting volume.

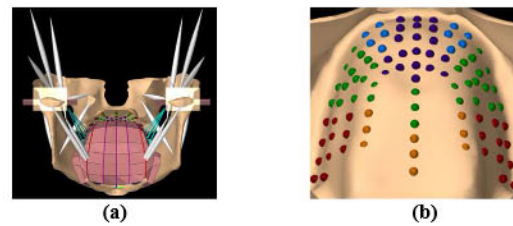


Figure 1: (a) The jaw-tongue-hyoid model in Artisynth; (b) the contact sensors locations on the palate

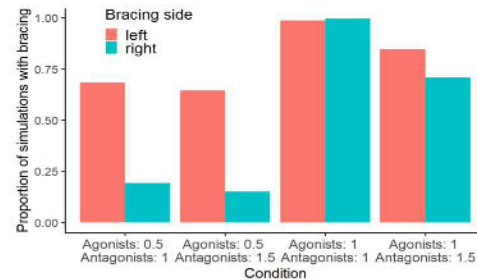


Figure 2: Lateral bracing outcomes for varied agonist/antagonist activation levels on the left side.

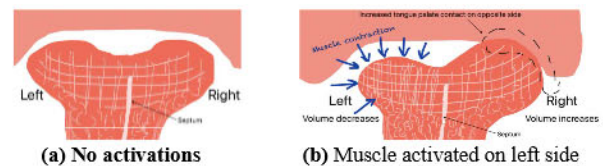


Figure 3: This diagram depicts how simultaneously activating VERT and TRANS displaces tongue volume to the opposite side.

However, further research is necessary to confirm or refute this hypothesis.

Acknowledgments

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