Muscular control of vocal fold adduction and eigenfrequencies:

Interaction between the TA and LCA muscles

Jun Yin and Zhaoyan Zhang

School of Medicine, UCLA, USA

April 10, 2014
ICVPB 2014 Conference, Salt Lake City

Acknowledgment: Research supported by NIH/NIDCD

- What we want to know: how different vocal fold adduction patterns affect the stiffness/stress conditions in the vocal folds

- Why:
  - stiffness conditions of the different layers of the vocal folds critically determine the resulting vibration and acoustics
  - How humans produce different voice types

Difficulties with experimental approach

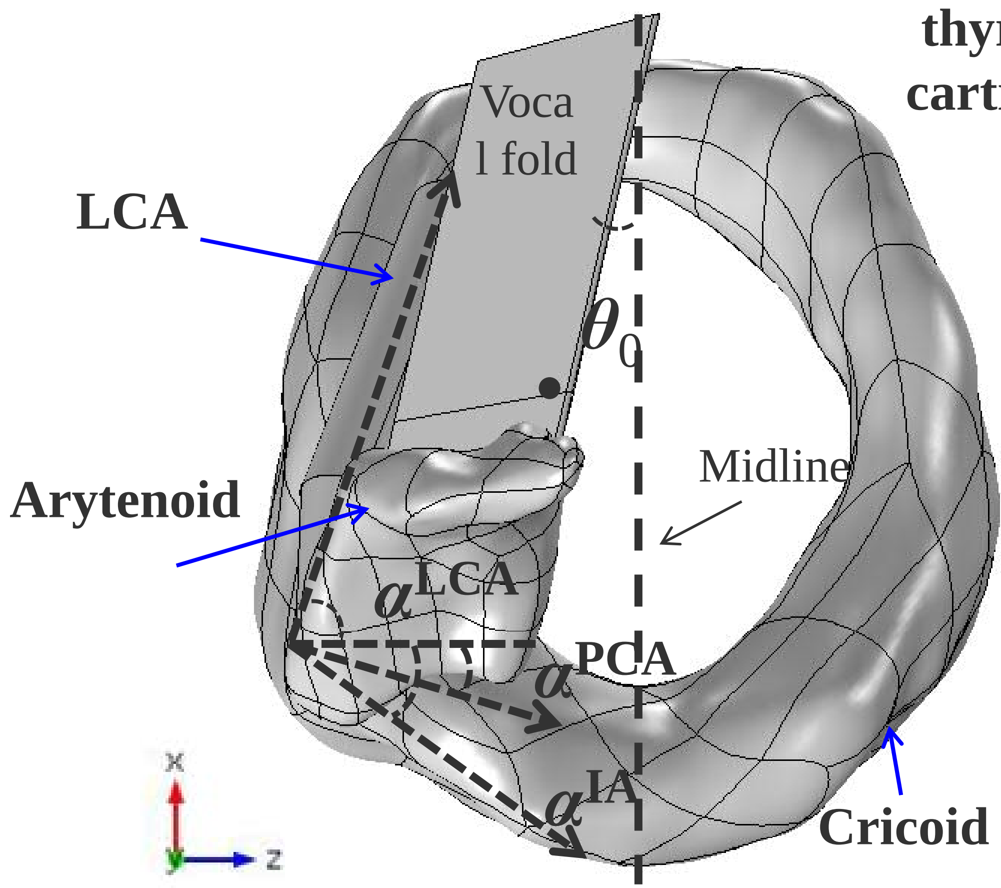
- Able to stimulate laryngeal muscles
  - Need human subjects or in vivo larynx model
- No reliable methods for in vivo measurement of the following during muscle stimulation:
  - Three-dimensional vocal fold geometry, including the inner layers
    - Endoscopic observation limited to a superior view
  - Stiffness within the vocal folds, nonlinear and anisotropic
- Alternative approach: Numerical modeling of muscular control of posturing

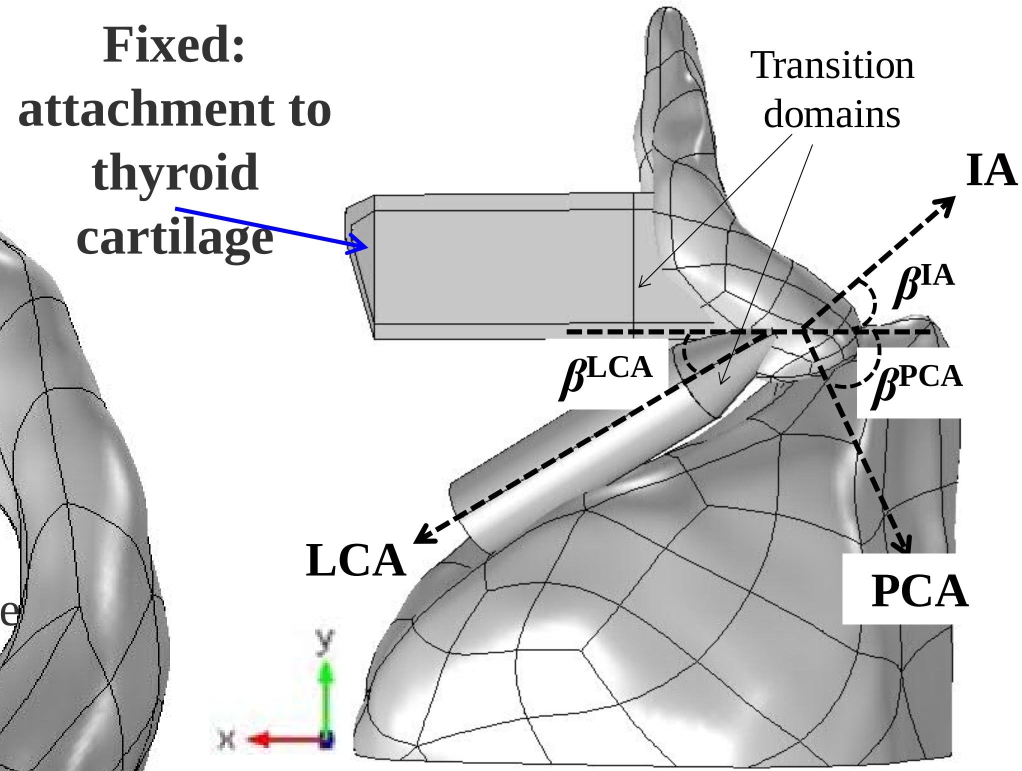
This study

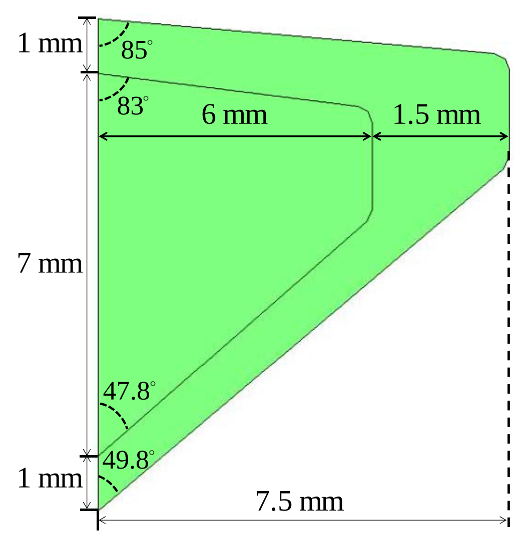
- Focus on the LCA/TA interaction

- Control of:
  - Glottal gap
  - Vocal fold deformation
  - Vocal fold eigenfrequencies
    - Indications of changes in stiffness/stress in the vocal folds

Our Model







MRI data from: Selbie et al. (1998; 2002) and Hunter and Thomson (2012).

Model details

- PCA and IA muscles were not activated in this study, but their passive response to arytenoid motion was modeled.

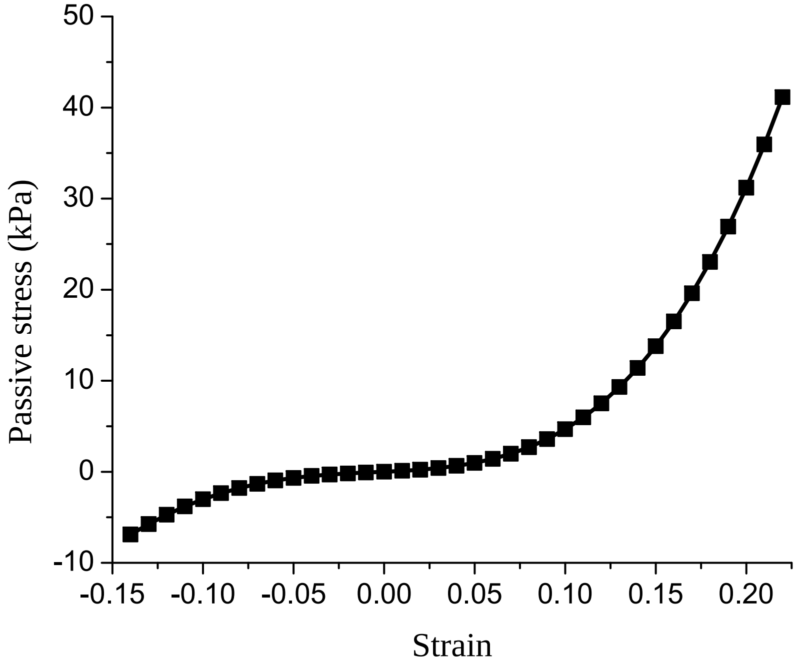
- The interface between the cricoid and arytenoid cartilages was modeled as a contact-sliding interface, which allowed relative sliding motion between the two cartilages in the tangential direction along the interface.

- A virtual film layer was added in between the cricoid and the arytenoid cartilages to prevent separation of the two cartilages

Constitutive model: passive component

- Passive material: vocal fold cover layer and all muscles
  - Isotropic
  - Hyperelastic
  - Nearly incompressible
- Hyperelastic strain energy function

$$W^{passive} = c_{10}\left(\bar{I}_1 - 3\right) + c_{01}\left(\bar{I}_2 - 3\right) + c_{20}\left(\bar{I}_1 - 3\right)^2 + c_{11}\left(\bar{I}_1 - 3\right)\left(\bar{I}_2 - 3\right) + c_{02}\left(\bar{I}_2 - 3\right)^2 + \frac{\kappa}{2}(J - 1)^2$$



Parameters curve fitted from the uniaxial testing data from human larynges in Zhang, Siegmund, and Chan (2007)

Constitutive model: active component

- Active material: laryngeal muscles (TA and LCA)

$$W = W^{active} + W^{passive}$$

- Active stress-stretch relation

$$\boldsymbol{\sigma}_{active} = \boldsymbol{\alpha}\boldsymbol{\sigma}^{\max} f^{active} \frac{\boldsymbol{\lambda}}{\boldsymbol{\lambda}_{ofl}}$$

$$f^{active} = \begin{cases} 1 - 4\left(1 - \frac{\lambda}{\lambda_{ofl}}\right)^2, & 0.5\lambda_{ofl} \le \lambda \le 1.4\lambda_{ofl} \\ 0, & 0.5\lambda_{ofl} > \lambda \text{ and } \lambda > 1.4\lambda_{ofl} \end{cases}$$

Muscle activation level: $\boldsymbol{\alpha} \in [0, 1]$

- Active stress-strain energy relation

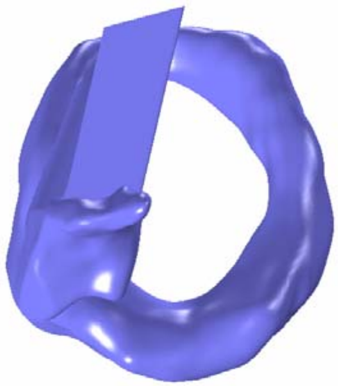
$$\boldsymbol{\sigma}_{active} = \boldsymbol{\lambda} \frac{\partial W^{active}}{\partial \boldsymbol{\lambda}}$$

$$W^{active} = \begin{cases} \frac{\alpha\sigma^{\max}}{3}\left[4\left(1 - \frac{\lambda}{\lambda_{ofl}}\right)^2 - 3\right]\left(1 - \frac{\lambda}{\lambda_{ofl}}\right), & 0.5\lambda_{ofl} \le \lambda \le 1.4\lambda_{ofl} \\ 0, & 0.5\lambda_{ofl} > \lambda \; and \; \lambda > 1.4\lambda_{ofl} \end{cases}$$

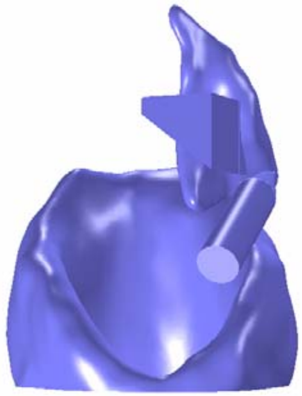
Ref: Hunter et al., 2004; Yin and Zhang, 2013

Vocal fold posturing: LCA activation

Superior view

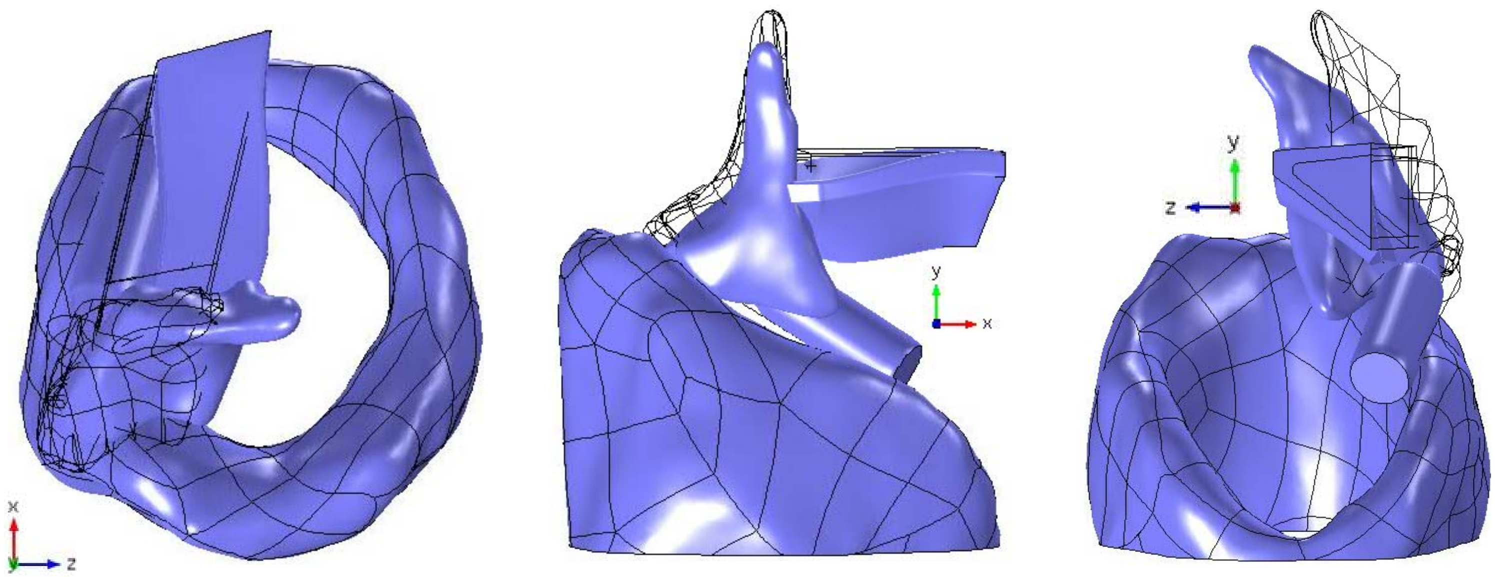


Side view



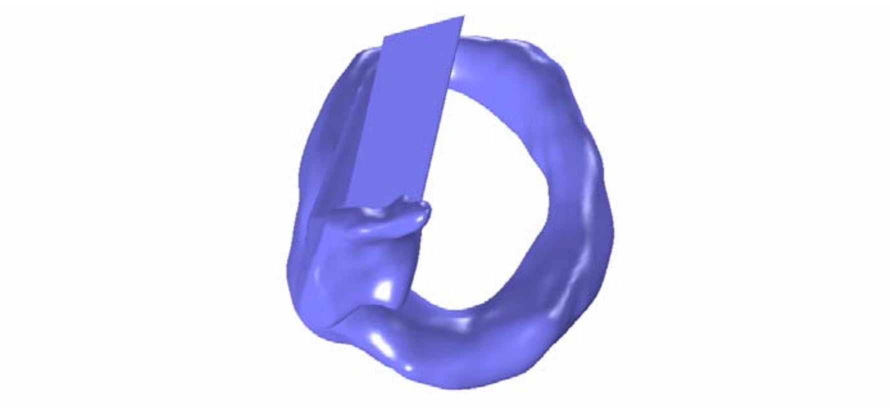
- Rotation of the arytenoid about the cricoid mainly in the coronal plane
- Posterior vocal fold to move medially and downward
- Adduction of the posterior glottis

LCA activation closes the posterior glottis, but not the middle part

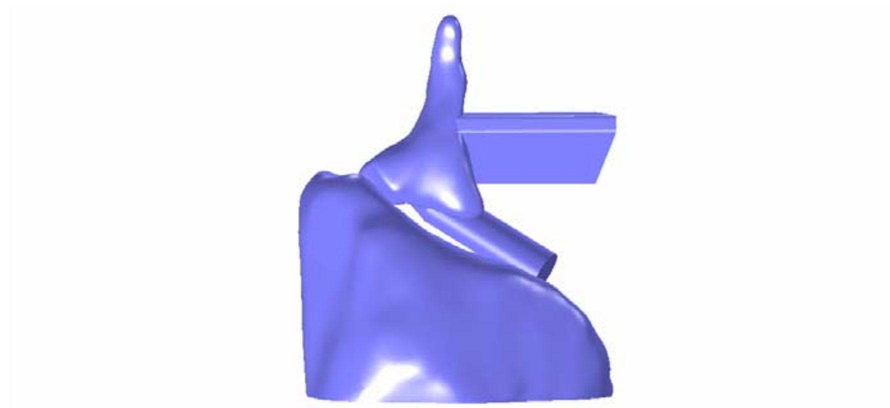


Vocal fold posturing: TA activation

Superior view



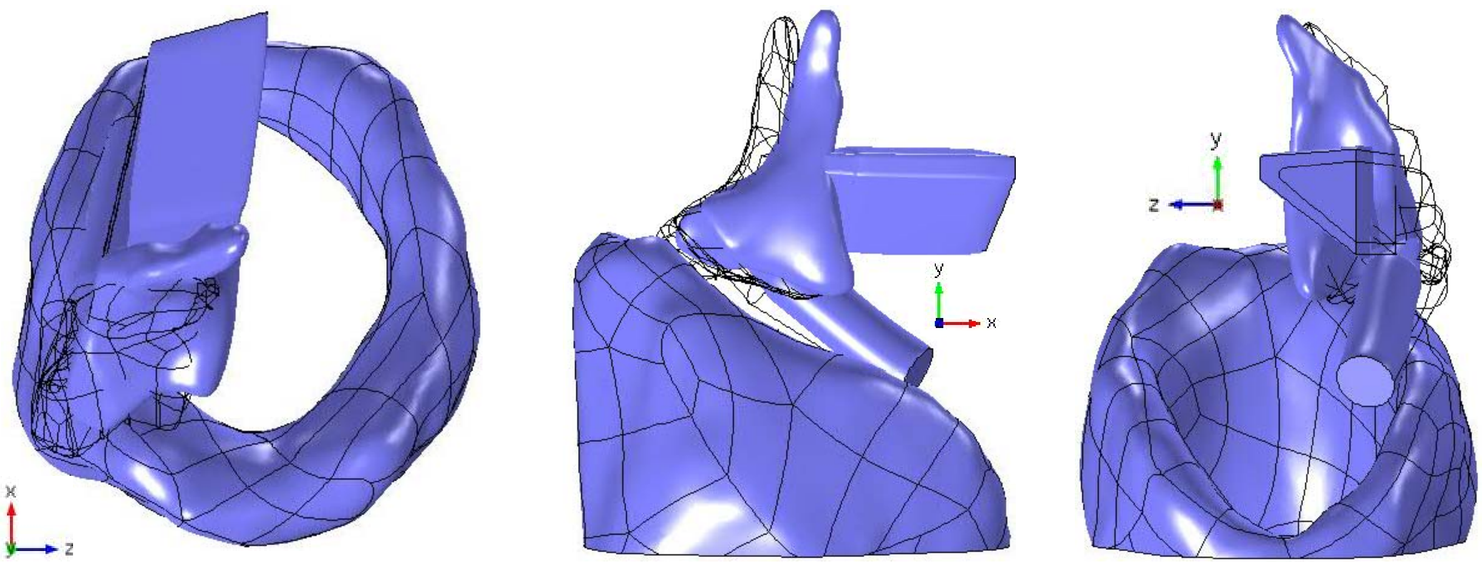
Side view



- Shortening the vocal fold
- Rigid-body-like rotation about the anterior attachment to the thyroid cartilage
- Slightly medial bulging

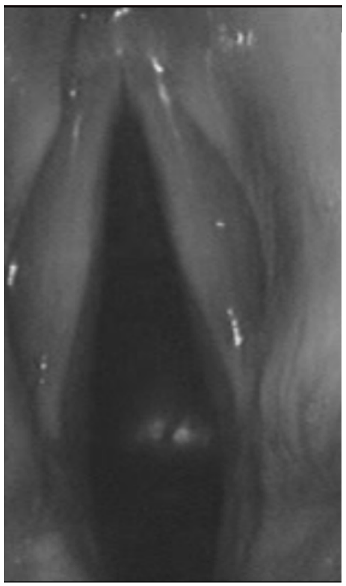
Anterior adduction

TA activation adducts the anterior vocal fold, but not the posterior part

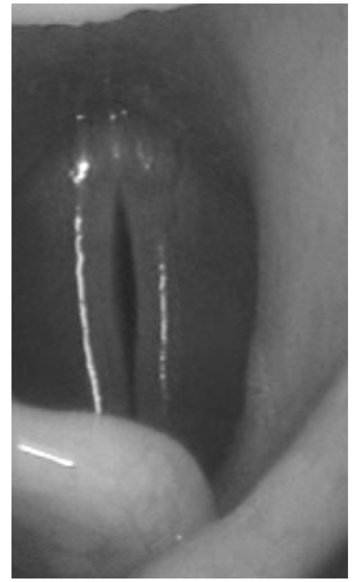


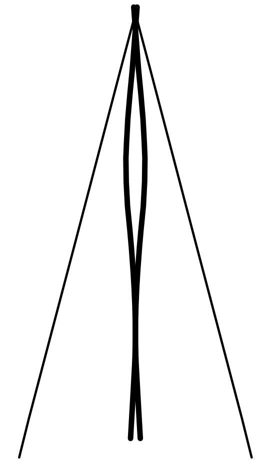
Due to difficulties in experiments, comparison to experiments is limited to a qualitative validation

Resting shape

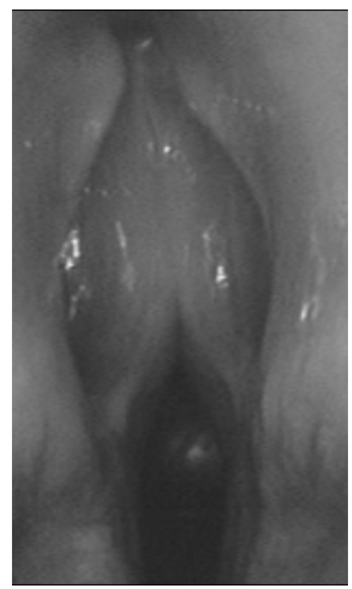


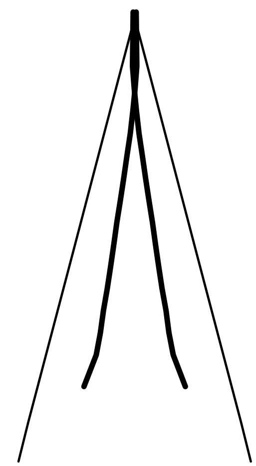
LCA contraction





TA contraction



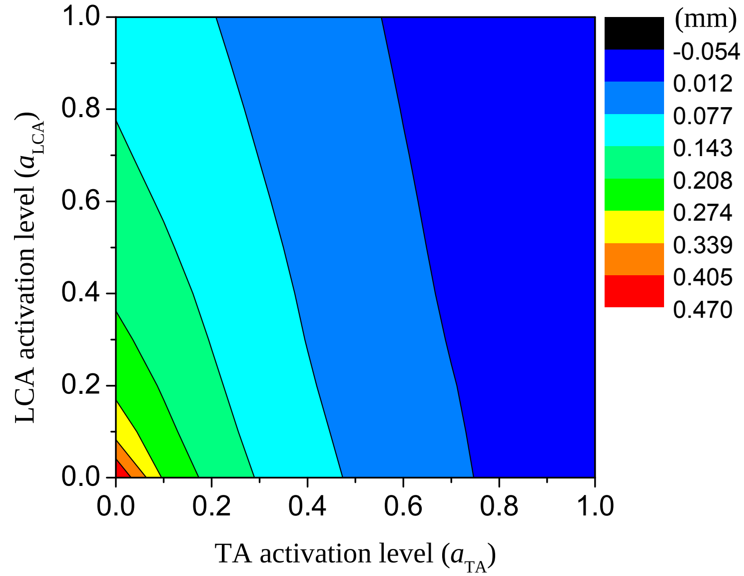


- *Qualitative agreement with in vivo canine experiments in Choi et al. (1993) and Chhetri et al. (2012).*

LCA/TA interaction: control of glottal gap

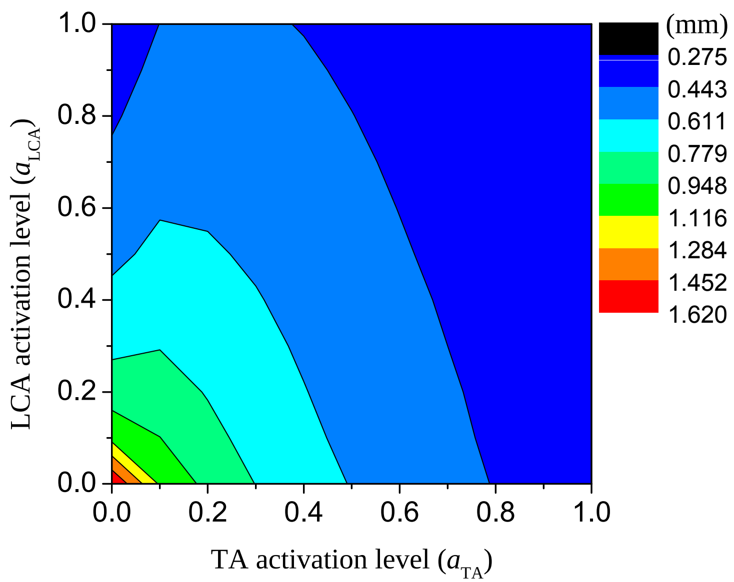
- Anteriorly, both TA and LCA activation reduce glottal gap
- Posteriorly, TA activation first increases then decreases glottal gap

Anterior cross section



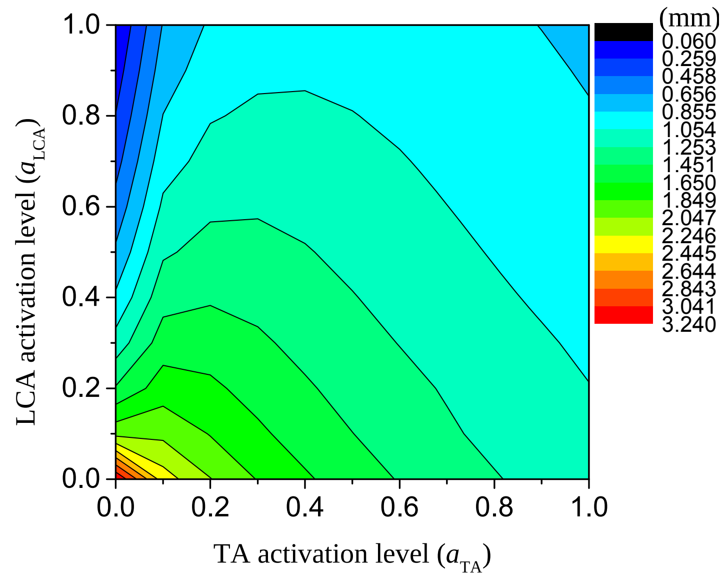
Middle cross section

LCA activation

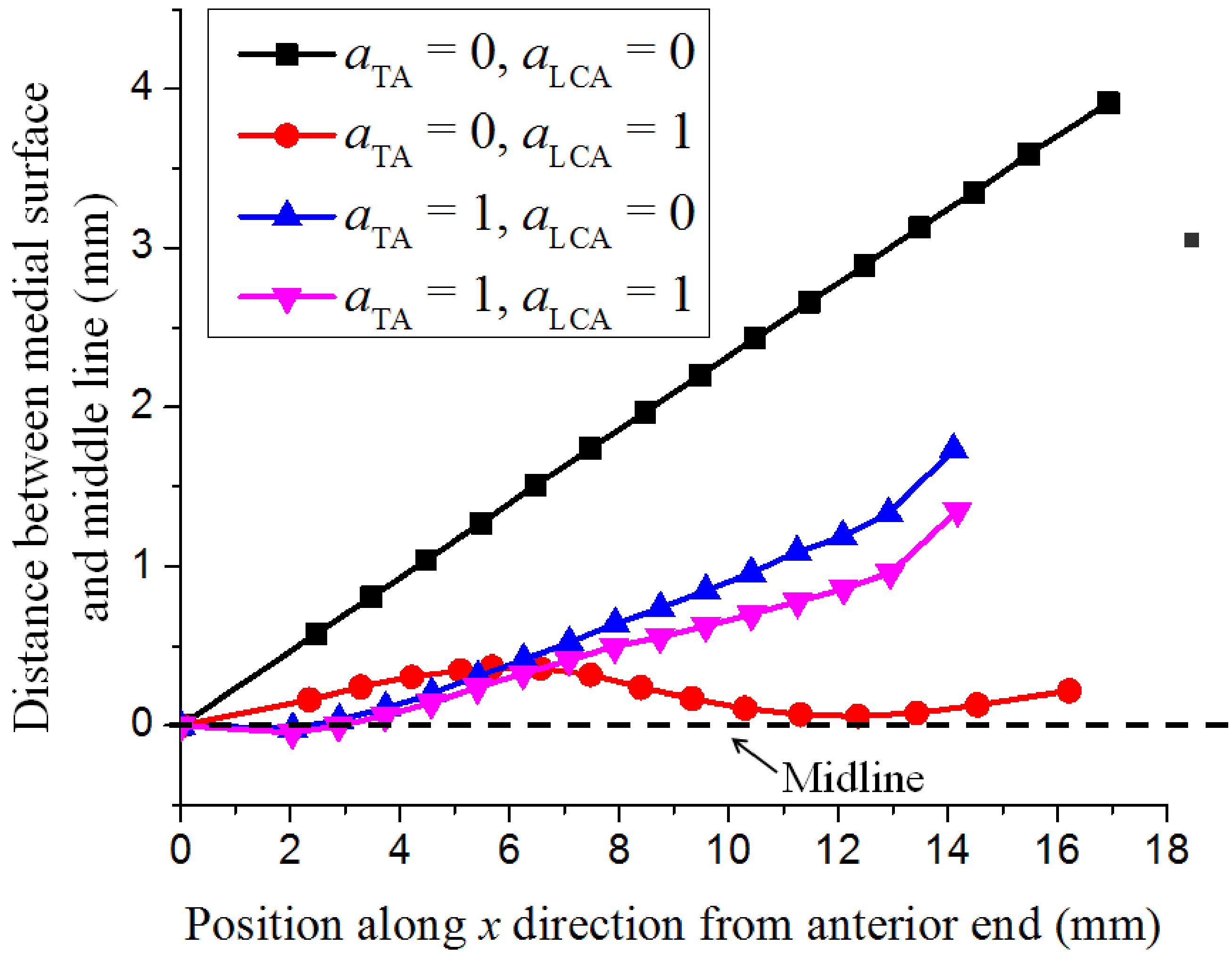


TA activation

Posterior cross section

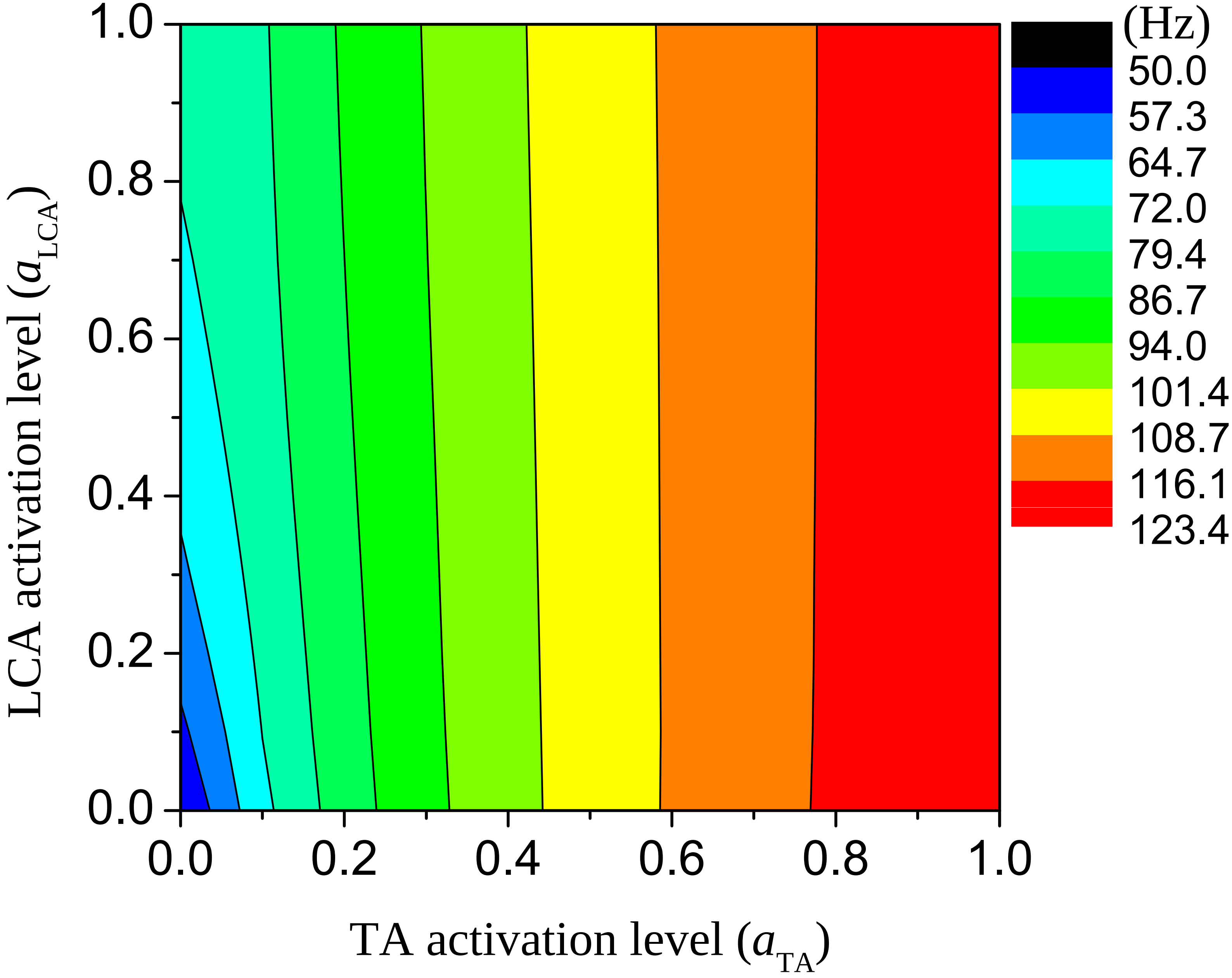


TA activation significantly weakens the adduction effect of LCA activation



- Presumably IA activation is required to close the posterior gap, in the presence of strong TA activation.

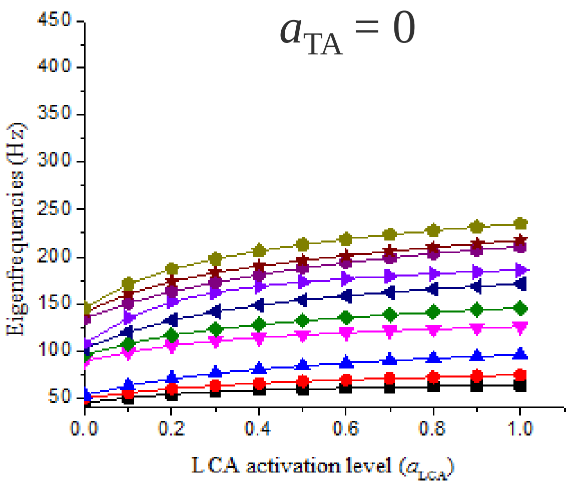
Control of 1st vocal fold eigenfrequency

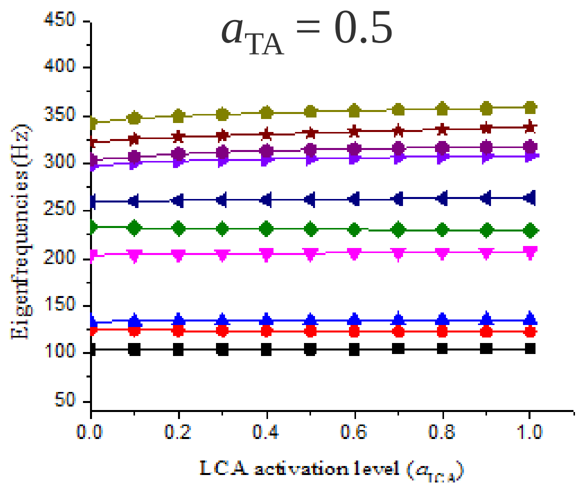


First ten eigenfrequencies

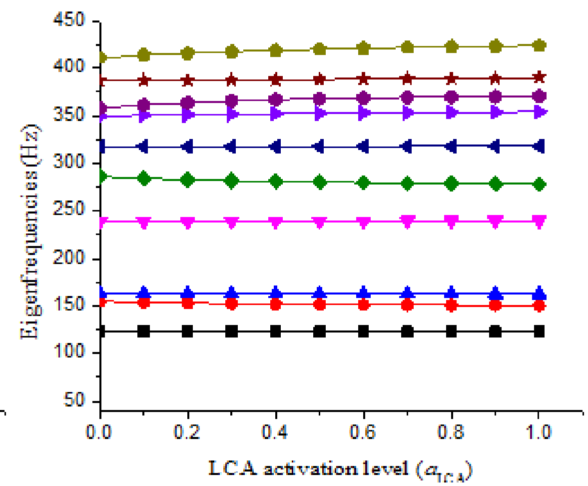
- *LCA activation alone is likely to have only minimum effect on phonation frequency*

➢ LCA activation



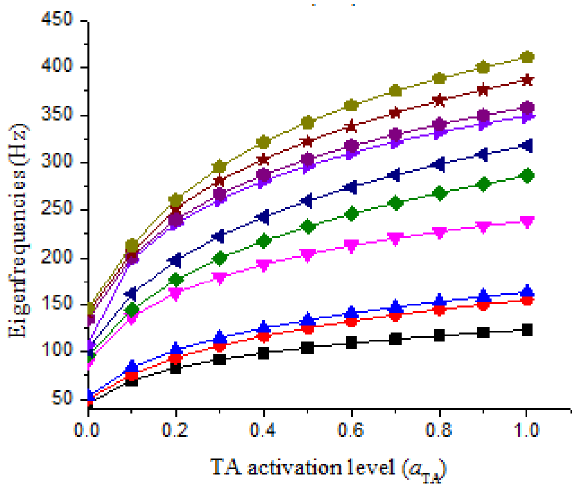


$a_{TA} = 1$

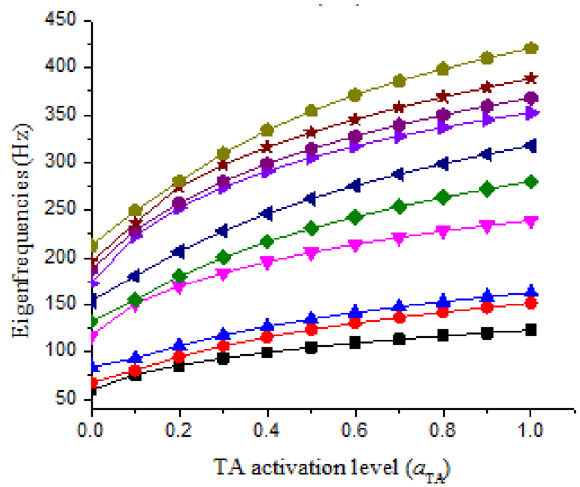


➢ TA activation

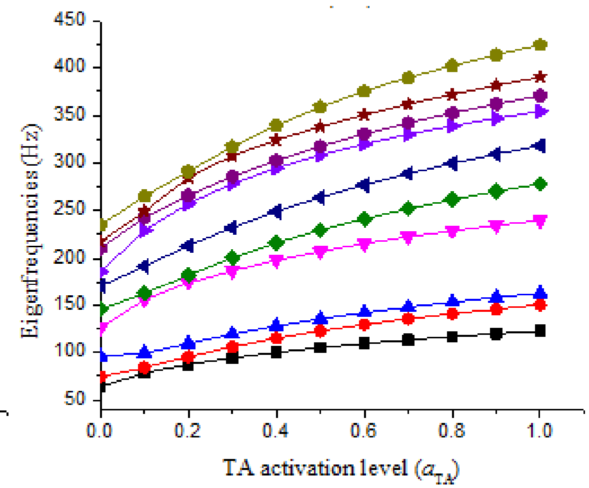
$a_{LCA} = 0$



$a_{LCA} = 0.5$



$a_{LCA} = 1$



What this means …

- LCA activation: a simple geometric adduction
  - Adduction of the posterior portion of the glottis
  - Does not change stiffness/stress much
- TA activation: both adduction (anterior) and stiffening
  - Adduction of the anterior portion of the glottis
  - TA activation stiffens vocal folds and thus reduces the adduction effect of LCA activation
- TA may act as a finer controller of phonation
  - Vibration: Open quotient
  - Voce quality (e.g., normal, breathy, or pressed)