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Short communication

Modeling mechanical stresses as a factor in the etiology of benign vocal fold lesions

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Abstract

Vocal fold tissue lesions such as nodules and polyps are thought to develop in response to mechanical stress that occurs during vocal fold collision. Two computational models of vocal fold collision during voice production are used to investigate this hypothesis. A one-dimensional lumped mass model, whose parameters are derived from vocal fold tissue dimensions and material properties, predicts stress perpendicular to the direction of impact (normal stress). A previously published three-dimensional finite element model that incorporates the same dimensions and properties predicts the entire stress tensor. The hypothesis is supported by predictions from the finite element model that three components of normal stress and one component of shear stress are increased during collision in the typical location of lesions (i.e. the center of the superior medial edge of the vocal fold in the middle of the vibrating and contact region). The lumped mass model predicts that mechanical stress is negatively correlated with mucosal thickness (increased by voice warm-up and hydration), is positively correlated with driving force (proportional to voice intensity), and is affected by voice production method. These relationships are consistent with clinical observations of vocal fold lesion risk factors and have implications for improving prevention and treatment of benign vocal fold lesions.

Keywords: Vocal folds; Larynx; Biomechanics; Injury; Finite element analysis

1. Introduction

Vocal nodules and polyps are convex lesions on the midline of the medial surface of the vocal folds associated with excessive voice use, loud voicing, inadequate hydration, and voice misuse (Buckmire and Rosen, 2001). Changes in vocal fold structure, such as vocal nodules and polyps, may increase the effort needed to speak, decrease the quality of sound produced, or eliminate the ability to produce audible voice. These symptoms are especially detrimental to the estimated 23% of the American workforce who have occupations in which voice use is essential (Titze et al., 1997).

Mechanical stress during vocal fold collision is implicated as a cause of benign vocal lesions, such as vocal nodules and vocal polyps, based on observations of high-velocity impact between vocal folds during speech (Titze, 1994) and of structural disruptions of the basement membrane in these lesions (Gray and Titze, 1988). Experimental investigation of this hypothesis is impeded by the difficulty of quantifying spatial and temporal stress variations in the small tissue volume (1–2 cm^3) that vibrates, on average, between 100 and 200 Hz during voice production, and the challenge of measuring and manipulating independent variables such as tissue stiffness. Theoretical models of vocal fold tissue mechanics are an ideal alternative research platform since they can predict spatial and temporal stress variations at appropriate resolutions and permit manipulation and control of geometric and material parameters that may influence these variations.

Simple and complex theoretical models have been used to quantify mechanical stress in vocal fold tissue. Titze (1994) uses impact/momentum principles and empirical relationships between vocal fold structure and function to generate an order of magnitude estimate of collision-induced compressive stress as a function of

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non-structural variables. Jiang et al. (1998) use finite element models to examine the spatial variation of tissue stress levels in the case of natural vibration without collision. This paper uses a novel lumped mass model and a previously published finite element model to investigate the hypothesis that vocal fold tissue lesions such as nodules and polyps develop in response to mechanical stress occurring during vocal fold collision.

2. Methods

2.1. Lumped mass model

A simple spring mass model (Fig. 1c), inspired by the lumped mass models of vocal fold movement that have been useful in the speech synthesis domain (for example, Ishizaka and Flanagan, 1972), generates an approximation for superficial normal stress in vocal fold tissue during collision with the opposing fold. The model approximates a coronal vocal fold cross-section (Fig. 1a) as two layers: a cover composed of the mucosa and underlying connective tissue, and a body composed of the thyroarytenoid muscle. Springs (k_c, k_b) in series define the two layers and a mass (m) at the junction of the springs represents the center of gravity. Interaction with the opposing fold occurs through the cover spring (k_c): When the folds are separated ($x>0$), this spring is not deformed. When the folds are in contact ($x<0$), the mucosal spring is constrained by the opposing fold at the midline plane, and deforms accordingly.

The frequency of vibration (F_0) is the inverse of the average of the non-contact and contact periods.

$$F_0 = \pi^{-1}\left(\sqrt{\frac{m}{k_c+k_b}}+\sqrt{\frac{m}{k_b}}\right)^{-1}. \quad (1)$$

Stress in superficial tissue is the product of the spring constant (k_c) and the displacement of the mass (x) divided by a cross-sectional area (A) during the contact phase. x_0 is the maximum lateral displacement of the mass:

$$\sigma_{xx} = -\frac{k_c x_0}{A}\sqrt{\frac{k_b}{k_c+k_b}}\sin\left[\sqrt{\frac{k_c+k_b}{m}}\left(t-\frac{\pi}{2}\sqrt{\frac{m}{k_b}}\right)\right]. \quad (2)$$

The lumped mass parameters are expressed in terms of physiological variables by comparing the lumped mass model to longitudinal compression of a rectangular prism approximation of a vocal fold (Fig. 1b) that is defined by geometric and material parameters from the literature (Tables 1 and 2). The frequency of vibration (115 Hz for nominal values) and maximum compressive stress (3.1 kPa for nominal values) during collision in terms of physiological variables are given by Eqs. (3) and (4):

$$F_0 = \pi^{-1}\left[\sqrt{\frac{\rho(D_c+D_b)}{E_c/D_c+E_b/D_b}}+\sqrt{\frac{D_b^2}{E_b}}\right]^{-1}, \quad (3)$$

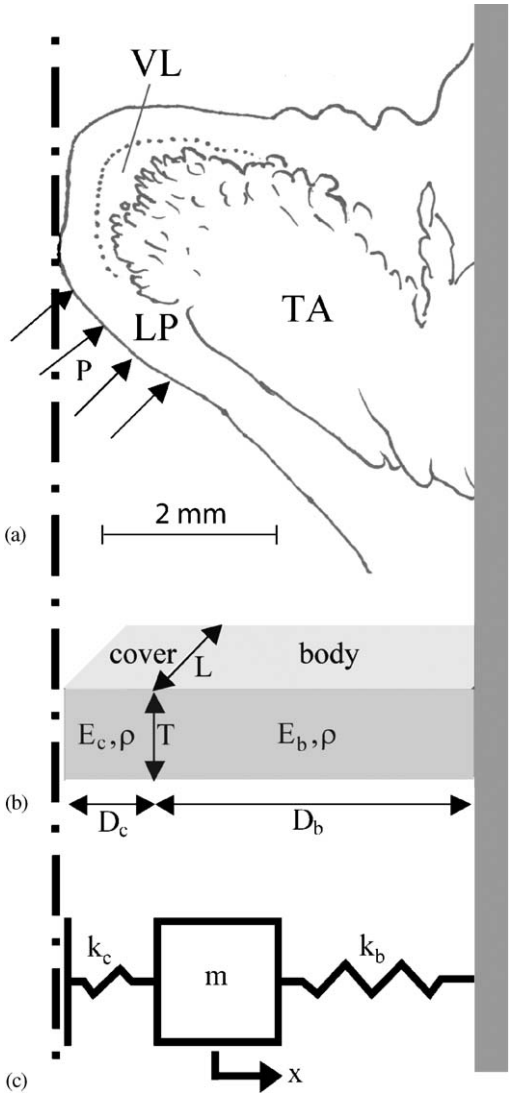

Fig. 1. Development of a lumped mass model of vocal fold collision. The dashed line indicates the plane of collision between opposing vocal folds in the center of the larynx. The gray line on the right represents the fixed boundary formed by the laryngeal cartilages. (a) Sketch of a histological cross-section illustrating the layered structure of the vocal fold—LP: lamina propria, VL: vocal ligament, TM: thyroarytenoid muscle. The arrows indicate the direction that air pressure acts during voice production. (b) Rectangular prism representation of a vocal fold with two layers that serves as a conceptual intermediate between the anatomical structure in (a) and the lumped mass model in (c). Cover represents the mucosa and ligament. Body represents the muscle. Each layer is defined by geometric properties of depth (D), thickness (T) and length (L) and material properties density (ρ) and stiffness (E). Nominal values of all parameters are given in Table 1. (c) One-dimensional lumped mass representation of a vocal fold defined by three parameters (m, k_c, k_b) that are defined by rectangular prism model parameters.

$$\sigma_{xx\ \max} = -\frac{PE_cD_b}{D_cE_b}\sqrt{\frac{E_b/D_b}{E_c/D_c + E_b/D_b}}. \quad (4)$$

2.2. Finite element model

A previously published three-dimensional model of vocal fold closure and collision, implemented using commercial finite element software (Abaqus Standard 5.8-1, Abaqus Inc., Pawtucket, RI), generates spatial and temporal predictions of the stress tensor during a single vocal fold closure (Gunter, 2003). The model consists of 14,242 10-noded tetrahedral linear elastic elements ($E = 36.1$ kPa, $v = 0.3$) that define a single vocal fold that is 14 mm long (Fig. 2a). The initial condition for vocal fold closure is the static solution for deformation by a medial driving pressure (P), which is manipulated in a range that is typical of human voice production (0.4–1.4 kPa) (Fig. 2b). The solution for vocal fold closure is the transient dynamic recoil of the unloaded vocal fold from this initial configuration including movement of the tissue towards its neutral position and collision with a midline plane (Fig. 2c). Details of model development, justification of assumptions of isotropy, linear elasticity and lack of an aerodynamic representation, calculation of a natural frequency of 128 Hz, and validation against experimental measurements of vocal fold collision forces are discussed elsewhere (Gunter, 2003). Stress tensor magnitudes at the centroids of elements along the vocal fold edge are compared for the contact and non-contact states.

Table 1
Typical vocal fold dimensions and material properties from the literature

Parameter	Symbol	Nominal value	Source
Length	L	1.5 cm	Hirano et al. (1988)
Thickness	T	0.25 cm	Perlman and Durham (1987)
Cover depth	D_c	0.11 cm	Hirano et al. (1983)
Body depth	D_b	1 cm	Tayama et al. (2002)
Cover stiffness	E_c	36.1 kPa	Min et al. (1995)
Body stiffness	E_b	20.7 kPa	Alipour-Haghighi and Titze (1991)
Tissue density	ρ	1.0 g/cm^3	
Driving pressure	P	0.8 kPa	

Table 2
Lumped model parameters in terms of biomechanical variables

Parameter	Relationship
A	LT
M	$LT(D_c + D_b)\rho$
k_c	E_cLT/D_c
k_b	E_bLT/D_b
x_0	PD_b/E_b

3. Results

The closed-form nature of the lumped mass model of vocal fold collision facilitates a comprehensive analysis of the effects of independent variables (Fig. 3, Table 3). Stress is independent of vocal fold length and thickness, is linearly related to driving pressure, correlates positively with increases in muscle depth, and cover stiffness, and correlates negatively with increases in muscle stiffness, and cover depth. Frequency is increased by either increase in muscle stiffness or cover stiffness. The

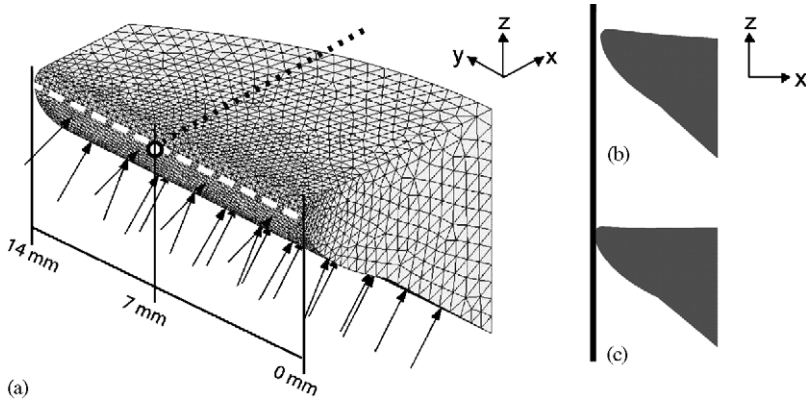

Fig. 2. Finite element model of vocal fold collision. (a) Three-dimensional geometric structure based on measurements of human vocal folds. Divisions indicate the boundaries of elements. Each element is defined by material properties taken from the literature. Arrows indicate driving pressure load. Dashed white line follows vocal fold edge, along which stresses are examined. Numbers indicate reference points for locations on the vocal fold edge. White circle indicates injury prone region at the center of the vocal fold edge. Dashed dark line indicates section cut for right hand images. (b) Cross-section showing initial condition for vocal fold collision model ($t = 0$ s). Deformation is the static solution to the driving pressure load. Solid black line indicates the collision plane. (c) Cross-section of dynamic vocal fold collision model at $t = 0.2$ s illustrating collision between the medial vocal fold surface and the collision plane.

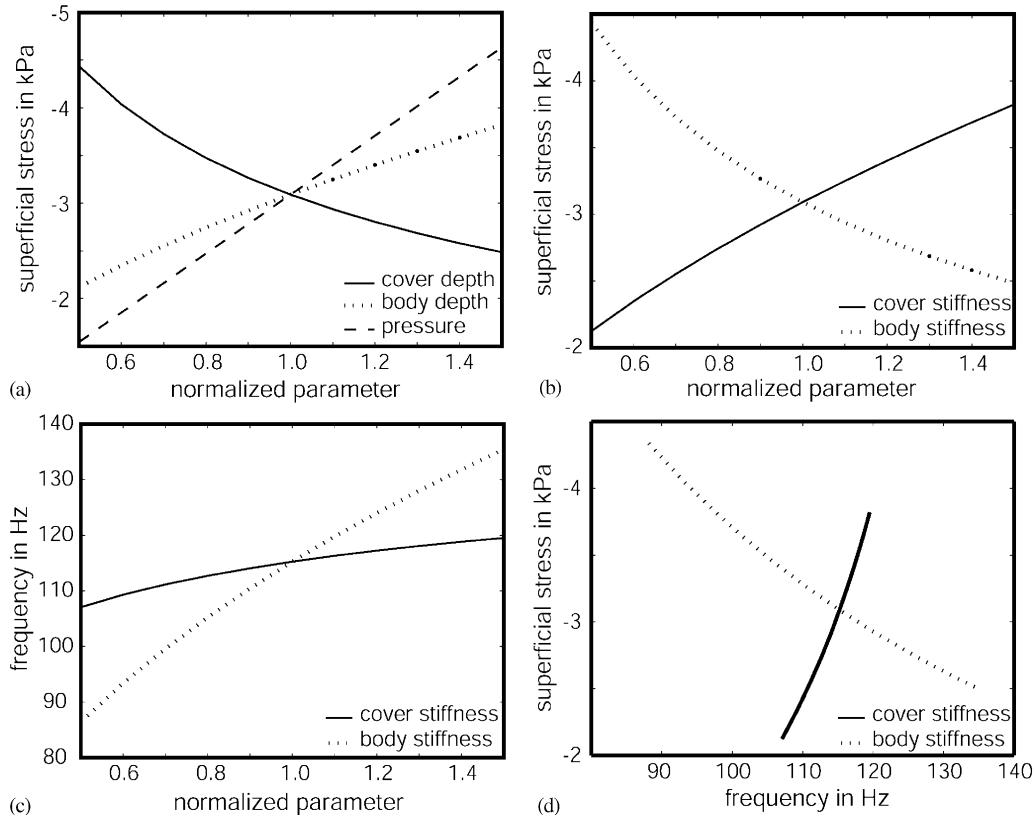

Fig. 3. Dependence of superficial stress in vocal fold tissue during collision and vocal fold vibration frequency (i.e. pitch) on geometric and material parameters as predicted by a lumped mass model of vocal fold collision. Negative stresses are compressive. Stress axes are reversed to illustrate compressive stresses. All manipulated parameters are normalized to the nominal values given in Table 1.

Table 3
Effect of physiological parameters manipulation on compressive stress magnitude

Physiological parameter manipulated	change in compressive stress magnitude
Volume differences	
↑ driving pressure	↑
Anatomical differences	
↑ muscle depth	↑
↑ cover depth	↓
Implementation differences	
↑ frequency by ↑ muscle stiffness	↓
↑ frequency by ↑ cover stiffness	↑

relationship between normal stress and frequency is direct if cover stiffness is manipulated and inverse if muscle stiffness is manipulated to create the change in frequency.

The finite element model permits three-dimensional examination of the entire stress tensor. There are increases in the magnitude of all compressive normal stresses (σ_{xx}, σ_{yy}, $\sigma_{zz} < 0$) and vertical shear stress on the plane tangent to the vocal fold edge (σ_{xz}) following collision in an element in the injury prone region for a driving pressure of 0.8 kPa (Fig. 4). These trends are typical of all superficial elements along the vocal fold edge. There is no discernible relationship between collision and the remaining shear stresses (σ_{xy}, σ_{yz}) or tensile stresses (σ_{xx}, σ_{yy}, $\sigma_{zz} > 0$). All compressive stresses and the vertical shear stress on the collision plane are elevated in the injury prone area (i.e. distance from the anterior boundary = 7 mm) for a driving pressure of 0.8 kPa (Fig. 5). The collision plane ($-\sigma_{xx}$) and vertical ($-\sigma_{zz}$) compressive normal stress maxima at the injury prone region are global maxima for the model. The horizontal compressive normal ($-\sigma_{yy}$) and vertical collision plane shear (σ_{xz}) have higher values on the anterior vocal fold boundary during the pre-collision period. Increased driving pressure increases post-collision compressive and vertical collision plane shear stress magnitudes at the injury prone region. All compressive stresses are linearly related to driving pressure ($r^2 > 0.9$).

4. Discussion

Predictions made by the computational models support and expand the hypothesis that benign vocal fold pathologies such as nodules and polyps are a reaction to mechanical stress. The magnitudes of multi-

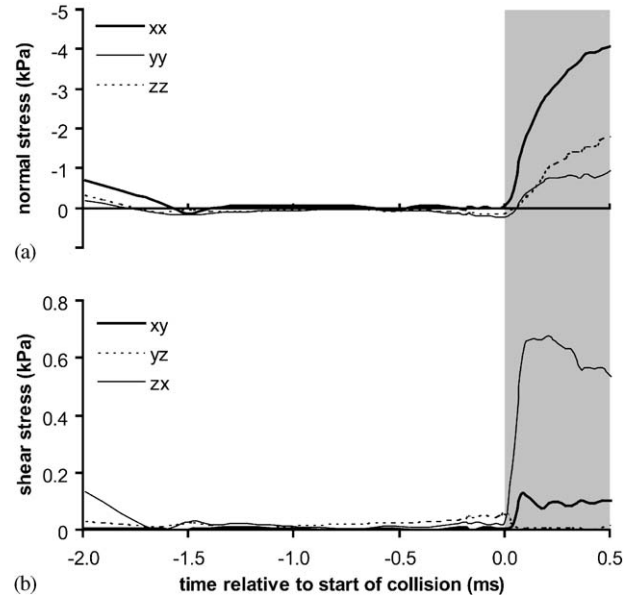

Fig. 4. Effect of vocal fold collision on components of the stress tensor at the centroid of a superficial element in the injury prone region for a driving pressure of 0.8 kPa as predicted with a finite element model. Time = 0 is the beginning of collision. Shaded area indicates post-collision period. (a) Normal stresses during vocal fold closure and collision. Tensile stresses are greater than zero. Compressive stresses are less than zero. Stress axis is reversed to illustrate compressive stress magnitudes. (b) Shear stresses during vocal fold closure and collision.

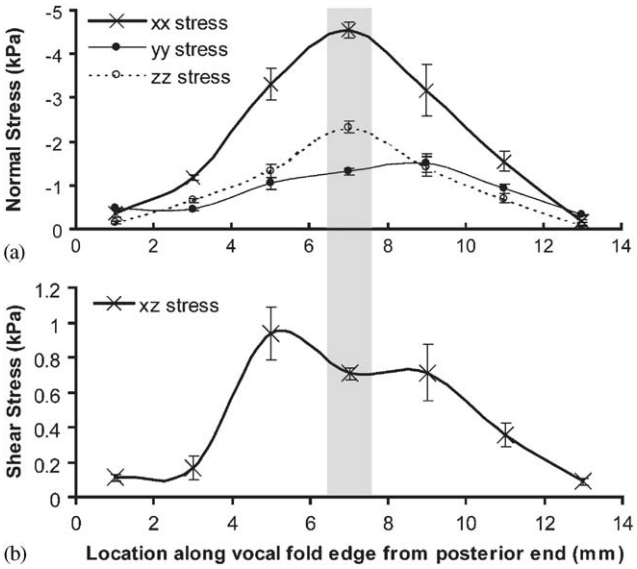

Fig. 5. Spatial variation of stress tensor components in superficial tissue along the vocal fold edge (see Fig. 2) for a driving pressure of 0.8 kPa as predicted by a finite element model. Shaded area is the injury prone region. Each point is a spatial average of seven elements within a 0.25 mm region. Error bars are standard error. (a) Normal stresses along vocal fold edge during collision. Negative stresses are compressive. Stress axis is reversed to illustrate compressive stresses. (b) Shear stress in the vertical direction on a plane parallel to the collision plane. Asymmetry reflects geometric asymmetry of the model.

ple components of the stress tensor increase during collision in the region where pathologies often develop (i.e. center of the vocal fold edge) (Fig. 5). This is also the region that attains the highest velocity immediately prior to contact. Therefore, the stresses reflect the development of elastic energy as contact forces decelerate the tissue. The nature of the increased components suggests possible mechanisms of tissue damage that lead to lesions, such as nodules and polyps, which are characterized by basement membrane disruption (Gray and Titze, 1988) and expanded extracellular matrix (Mossallam et al., 1986). The collision-associated compressive stress maxima in two coordinate directions and elevated compressive stress in the third at the injury prone region (Fig. 5a) are of similar magnitude to those that increase protein synthesis and secretion by tracheal epithelial cells in culture as shown by Swartz et al. (2001) and are therefore likely to lead to an expansion of the extra-cellular matrix. Vertical shear stress on the plane parallel to collision is elevated at the injury prone location (Fig. 5b) and is of the appropriate orientation to cause the disrupted adhesions between epithelial cells and the basement membrane. The lack of tensile stress in the injury prone region during collision (Fig. 4a) suggests that tension is not the cause of tissue failure that leads to vocal nodule and polyp formation. In the absence of data pertaining to the dose response of vocal fold tissue elements to the components of the stress tensor it is difficult to say which magnitudes and directions of stress pose the greatest injury risks. Carefully controlled studies using vocal fold tissue culture models would further guide interpretation of these results and permit study of the role played by frequency of deformation in tissue injury.

The models provide insights into mechanical bases for risk factors associated with development of vocal fold pathologies. Driving pressure is proportional to sound volume (Isshiki, 1964). Therefore, the linear relationship between driving pressure and compressive stress magnitude in the region that is prone to injury provides an explanation for the presumed association between high volume voicing and formation of pathologies. The inverse contribution of cover depth to compressive stress magnitude (Fig. 3a) offers an explanation for the proposed protective nature of hydration and warm up against vocal fold injury since both are likely to increase fluid volume in, and depth of, the cover. The prediction by the lumped mass model that mechanical stress is dependent on the method of frequency manipulation (Fig. 3d) addresses the role played by voicing practice in determining mechanical stress levels and risk of tissue injury. According to these predictions, it is conceivable that two individuals with similar anatomy may produce sounds of similar pitch and volume using different production strategies (i.e. different muscle activation patterns) and therefore have different mechanical stress

levels and injury risk. This lends credibility to anecdotal evidence that lack of training or inappropriate training leads to voice "misuse" and increased risk of lesion formation. It also suggests that information on how voice is generated may prove useful for clinical diagnosis and treatment of these benign lesions.

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