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THE EFFECT OF SIMULATED NODULES ON VOCAL FOLD MOVEMENT  
IN A TWO-LAYER SYNTHETIC MODEL

by

Rachelle Nevitt Rauma

A thesis submitted to the faculty of  
Brigham Young University  
in partial fulfillment of the requirements for the degree of

Master of Science

Department of Communication Disorders  
Brigham Young University

April 2009

BRIGHAM YOUNG UNIVERSITY

GRADUATE COMMITTEE APPROVAL

of a thesis submitted by

Rachelle Nevitt Rauma

This thesis has been read by each member of the following graduate committee  
and by majority vote has been found to be satisfactory.

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Christopher Dromey, Chair

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Date

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Shawn Nissen

BRIGHAM YOUNG UNIVERSITY

As chair of the candidate's graduate committee, I have read the thesis of Rachelle Nevitt Rauma in its final form and have found that (1) its format, citations, and bibliographical style are consistent and acceptable and fulfill university and department style requirements; (2) its illustrative materials including figures, tables, and charts are in place; and (3) the final manuscript is satisfactory to the graduate committee and is ready for submission to the university library.

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Date

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Christopher Dromey  
Chair, Graduate Committee

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Graduate Coordinator

Accepted for the College

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Date

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K. Richard Young  
Dean, David O. McKay School of Education

## ABSTRACT

### THE EFFECT OF SIMULATED NODULES ON VOCAL FOLD MOVEMENT IN A TWO-LAYER SYNTHETIC MODEL

Rachelle Nevitt Rauma

Department of Communication Disorders

Master of Science

#### Abstract

This study examined the differences between normal vocal fold vibration and the movement patterns of vocal folds with mass lesions by means of a synthetic model. The experimenter molded and cast three sets of vocal folds, representing normal structure, small nodules, and larger nodules. Acoustic, aerodynamic, and digital video signals were recorded and analyzed in order to quantify air flow and pressure, measure vibratory stability, and visually assess closure patterns across the three structural conditions. Statistical analysis revealed that the presence of vocal nodules resulted in a significantly higher onset pressure, fundamental frequency, airflow at onset, and offset pressure. However, the results were inconclusive with regard to vocal stability, and it remains unclear whether the current models of nodules are sufficiently similar to the human system to adequately model the type of mass lesions typically seen in a clinical context.

## ACKNOWLEDGMENTS

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## Introduction

Voice production is affected by forces acting on the vocal folds, and their patterns of movement contribute to the changes in voice quality perceived by listeners (Gunter, 2002). Numerous models of phonation have been described in the literature and used in research in order to investigate vocal fold movement in relation to airflow. These models have been developed because of difficulty with imaging and measuring vocal fold movement *in vivo* (Alipour, Scherer, & Finnegan, 1997; Berry, Zhang, & Neubauer, 2006; Dollinger & Berry, 2006; Drechsel, 2007; Drechsel & Thomson, 2008; Hsiao, Liu, Luschei, & Titze, 2001; Thomson, Mongeau, & Frankel, 2005; Zhang, Neubauer, & Berry, 2006; Zhang, Neubauer, & Berry, 2007).

One kind of phonation underrepresented in modeling research, however, is a type that can occur as a result of collision forces on the vocal folds (Titze, 1994), which is often referred to as vocal abuse, or phonotrauma, where the voice is used too loudly for too long. Mass lesions can arise as a result of tissue damage (Colton, Casper, & Leonard, 2006). With the many modeling studies that have been undertaken in an attempt to quantify vocal fold movement patterns, there have been a limited number providing measurements of the oscillation patterns that can occur in a disordered system. Information about the interaction of airflow, pressure, and tissue resistance in relation to vocal fold movement will be useful in understanding the differences between normal and pathological voices and will contribute to furthering the current understanding of vocal fold vibration. Having a more detailed understanding about the characteristics of disordered vocal fold movement may also prove to be a clinical aid for the early detection of benign vocal fold lesions. The purpose of the current study is to compare normal and pathological vocal fold movement in a self-sustained oscillatory model of the vocal folds.

## Review of Literature

Scientists have studied vocal fold oscillation in detail for several decades. Various techniques have been used in this line of research, many of which have revealed valuable details of vocal fold physiology. One technique used more recently in research is physical modeling, which uses synthetic materials shaped to a similar geometry as human vocal folds. Models of human vocal folds are useful research tools which can overcome the significant imaging challenges of *in vivo* vocal fold studies. Synthetic models have the potential to provide important data that would otherwise be unavailable.

### *Vocal Fold Morphology*

The vocal folds are located within the larynx at the narrowest portion of the airway. This system of folds seals off the laryngeal airway completely and rapidly when the appropriate muscles are activated. There are several layers of the vocal folds to consider.

The most superficial layer is composed of epithelial tissue. Beneath it is a multilayer system composed of nonmuscular tissue, called the lamina propria. The lamina propria can itself be divided into three layers: superficial, intermediate, and deep. Each layer has a different composition of fibers to allow the vocal fold to oscillate in a complex way when it is set into motion. Lateral to the lamina propria is the thyroarytenoid muscle, which runs along the length of the vocal fold anteriorly to posteriorly. This muscle is the major portion of the vocal fold, comprising the majority of the vocal folds' thickness (Hardcastle, 1976).

The intermediate and deep layers of the lamina propria comprise what is known as the vocal ligament. The ligament (intermediate and deep layers) is thicker at the end points where larger mechanical stresses occur in the fibers (Titze, 1994).

It seems that the vocal fold is designed to protect itself at its end-point, where larger mechanical stresses occur in its fibers. The mucosa is thicker in the middle of the vocal fold where most collision forces occur. It has been suggested that the superficial tissue may be well equipped to withstand a certain amount of direct impact, which then acts as a sort of shock absorber for the ligament (Titze, 1994).

### *Mechanics of Phonation*

The vocal folds are able to sustain oscillation (repeated back and forth movement) over an extended period of time (Titze, 1994). The characteristic back and forth movement of the vocal folds is what causes phonation to occur in humans and animals alike. In the larynx, this phenomenon is called flow-induced oscillation because a stream of air through the glottis promotes vibration in the system. Movement of the vocal folds is not “neurochronaxic,” with individual impulses from the nervous system leading to repeated movement of the folds. Rather, voicing is an aeromechanical event (Holmberg, Hillman, & Perkell, 1988; Tanaka & Gould, 1983; Titze, 1989; Titze, 1994). This can be illustrated in post-mortem examinations, which show the ability of an excised larynx to phonate when air is pushed through the laryngeal system when there is no neurological input to the muscles of phonation. Laryngeal muscles contribute by positioning and shaping the vocal folds, but not in making the folds move back and forth during each cycle.

Van den Berg (1958) described vocal fold vibration as a function of tissue elasticity and vocal fold collision. His description has been called the myoelastic-aerodynamic theory of vocal fold vibration. Van den Berg discussed the importance of the Bernoulli principle, which describes the reduction in pressure that occurs when particle velocity increases in a fluid system. However, continual energy transfer from the

airflow through the glottis to the tissue is more complex than negative pressure from the Bernoulli effect alone, because these forces cannot account for inward and outward movement of the folds (Titze 1994). If this were the case, a damping effect would diminish the movement of the tissue until the vocal folds came to rest. In addition to the Bernoulli effect, the myoelastic-aerodynamic theory describes two other reasons for sustained vibration of the vocal folds: elastic recoil of the tissue and decreased subglottal pressure (Jiang et al., 2000).

During phonation, when the subglottal pressure becomes great enough to overcome the resistance offered by glottal adduction, the vocal folds are forced apart and air flows through the glottal opening. As the air flows through the narrow glottis, air velocity increases to cause a decrease in intra-glottal pressure. This decrease in pressure, in addition to the elastic forces in the tissue of the vocal folds, essentially pulls the cords together medially (Hardcastle, 1976).

Several theoretical models have been suggested to explain phonatory movement of the vocal folds. The one-mass model, described by Titze, has been used to give a visual representation of vocal fold movement during phonation. Later on, the three-mass model was suggested as a way to account for vertical phase differences between the lower and upper parts of the vocal folds, which was lacking in the description of the one-mass model's movement pattern (Titze, 1994).

Rate of airflow through the glottis depends partly on subglottal pressure and will largely determine the degree to which the vocal folds are pulled toward each other during oscillatory movement (Hardcastle, 1976). Greater airflow through the glottis, leading to increased intensity, is almost always accompanied by a passive increase in fundamental

frequency ( $F_0$ ). This is due to passive changes that occur to the vocal folds themselves as they are displaced during more intense phonation. As the vocal folds are moved further away from the midline in periods of loud phonation, they become displaced to the point that the tissue is stretched, resulting in smaller vocal fold cross-sectional mass and increased stiffness. The decrease in mass and increase in stiffness results in an increase in the number of vibration cycles per second (Hz), thus, a passively-induced increase in  $F_0$ .

#### *Interaction of Aerodynamic Variables*

Knowledge of the interaction of pressure, flow, and resistance in the larynx is important in considering how the vocal folds oscillate during phonation. Normal vibratory movement of the vocal folds depends on an interaction of several variables. Glottal resistance can be regulated by adjusting the level of vocal fold adduction, and the driving pressure from the lungs can also be controlled by the speaker. The combination of these two parameters will determine the level of flow during phonation.

The driving force for voice production has been described as a bellows-like system, with pressure being generated in the trachea as the lungs are deflated (Catford, 1983). From this point, the main source of resistance to initiated airflow is typically the glottis (Titze, 1994). Because of vocal fold adduction during phonation, more resistance is present during voicing than during metabolic breathing. Therefore, additional lung pressure is needed to force air through the constricted larynx during speech (Titze, 1994). Research has shown that vocal intensity is tied to subglottic pressure and airflow rate (Holmberg et al., 1988; Tanaka & Gould, 1983).

Resistance and pressure are modified during speech to provide varying degrees of loudness or breathiness. For example, relaxed vocal folds (mild resistance) with a relatively high amount of upward pressure from the lungs (high pressure) will result in



increased flow through the glottis, and will sound breathy to listeners. Hypofunctional (lax) voices are produced with low resistance from the larynx and hyperfunctional (tense) voices are produced with an excessive degree of laryngeal resistance (Södersten, Lindestad, & Hammarberg, 1991). A loud voice is produced with increased pressure, and a quiet voice is produced with a relatively small amount of pressure. It is easier to sustain a low-pitched phonation than a higher one, in terms of muscular effort. As pitch increases, the vocal folds become stiffer (increased resistance), and more pressure is needed to maintain the same amplitude of sound (Titze, 1994).

Because vocal fold movement patterns vary between speakers as a result of differences in pressure, flow, and resistance, some voices have a higher ratio of acoustic to aerodynamic power during phonation. Several studies have focused on calculating vocal efficiency as a measure of this ratio of acoustic to aerodynamic power (Fulton, 2007; Hiki, 1983; Holmberg et al., 1988). In these studies, measures of flow rate multiplied by pressure, in relation to the sound signal's intensity, have been used to provide a quantitative measure of energy transfer in the vocal folds. For example, a breathy voice would be relatively inefficient, due to the inadequate vocal fold resistance relative to the driving pressure from the lungs. Incomplete vocal fold closure during phonation has been shown to be significantly correlated with a high degree of perceived breathiness (Södersten et al., 1991).

#### *Sources of Phonatory Fluctuation*

In a normal system, the vocal folds oscillate fairly consistently from cycle to cycle, without substantial leakage of air or significant irregularity of movement in either fold. The folds move out of phase from each other, yet show no deviant physical behavior during the cyclic movement, despite changes that may occur as a result of altering pitch

or intensity. There are several ways in which a normal system can be disturbed, however, resulting in irregularity in the movement patterns of the folds. Mass lesions such as nodules, polyps, or cysts may develop in an otherwise normal speaker's system, creating such changes in the basement membrane zone (BMZ) as have been shown by Gray (1991) and Courey, Shohet, Scott, and Ossoff (1996). Thickening of the BMZ, with an increase in fibronectin probably represents tearing forces, followed by consequential wound repair in the subepithelium when the voice is driven too hard by a speaker. Gaps at intercellular junctions, disruption and duplication of the BMZ, and deposits of collagen fibers have also been reported (Kotby, Narrar, Seif, Helal, & Saleh, 1988). This disorganization of the BMZ has been said to leave the vocal fold vulnerable to repeated injury (Gray, Hammond, & Hanson, 1995). Hyperphonation results in several changes to the vocal fold epidermis, including: (a) damage to the microvilli, (b) creation of a cobblestone appearance along the surface, and (c) damage to the surface and underlying cells (Gray, Titze, & Lusk, 1987).

In terms of mechanical stress, it has been suggested by Titze that the tissue of the vocal folds is probably not suited for intense and repetitive impact (1994). There is a general belief that growths on the vocal fold surfaces, such as nodules, polyps, and contact ulcers, are the result of repetitive collision forces over a prolonged period of time (Titze, 1994). The following discussion examines mass lesions in greater detail.

*Vocal nodules.* Nodules are localized benign growths on the vocal folds, thought to be the result of prolonged loud phonation, often referred to as vocal abuse (Colton, et al., 2006). It is a common belief among researchers that vocal nodules result from prolonged and repeated collision of the vocal folds during vibration (Titze, 1994).

Research has shown that vocal load (the number of oscillated cycles over time, energy dissipated, and acceleration and deceleration of vocal fold tissue during vocalization) is greater in loud speech than for normal or monotone speech, showing that repetitive and intensive use of the voice may cause enough collision force to pose a threat of injury (Titze, Svec, & Popolo, 2003). Higher-pitched voices, which can be found in most women, children, and tenors, are more susceptible to nodules than lower-pitched voices, suggesting that collision frequency plays a role in the formation of nodules. This has been documented clinically as a cumulative result of the number of collisions per unit of time (Greene, 1980; Van Riper & Irwin, 1958). Effortful vocal production has also been documented as a contributor to the formation of vocal nodules (Boone, 1983). It is therefore likely that nodules result from the reaction of the tissue to the constant stress induced by frequent, hard oppositional movement of the vocal folds.

Early or acute nodules are fairly soft and pliable, may be reddish, and are mostly vascular and fluid-filled. With continued trauma, the tissue undergoes hyalinization and fibrosis, making the nodule and surrounding area much more firm. Nodules that have been present for an extended amount of time will become hard, white, thick, and fibrosed. When nodules are chronic, they are usually bilateral, but they may not be entirely symmetrical (Titze, 1994).

Vocal fold nodules arise at the junction of the anterior and middle third of the vocal fold and in their more advanced stage, tend to appear white, opaque and firm (Dikkers, 1994; Titze, 1994). They typically result in an hourglass glottal closure configuration, and will affect the vocal fold mucosal wave and vibration in different ways, depending on their size and degree of associated edema (Johns, 2003).

Stresses to the vocal folds that are likely to contribute to the formation of vocal nodules include compressive stress and shear stress. Compressive stress perpendicular to the plane of contact may cause cellular rupture over time. Shear stress parallel to the plane of contact has been suggested to cause separation of tissue elements. Damage may also be caused by cellular alteration in function in response to the stress environment (Gunter, 2003).

*Vocal polyps.* Polyps have also been attributed to repeated mechanical stress, in addition to some other type of irritation of the tissue lining of the epithelial layer. Polyps can be localized in one area, or they can be distributed over the surface of the vocal fold. These lesions are fluid-filled masses that can be either sessile (broadly attached at the base) or pedunculated (thinly attached and bulb-like) in appearance (Colton, et al., 2006; Dikkers, 1994; Titze, 1994).

Pathological changes of the vocal fold extracellular matrices alter vocal quality secondary to the loss of normal vibratory function and alteration of tissue viscosity and thereby create mild to debilitating levels of dysphonia when the polyps are present on the folds (Colton, et al., 2006).

Airflow in a patient with nodules or polyps may be equal to or slightly higher than that found in a patient without mass lesions (Colton, et al., 2006). Tanaka and Gould (1985) reported a mean value of 275 mL/s in their two patients with nodules, whereas normal male speakers produce flows of approximately 125 mL/s. Woo, Colton, and Shangold (1987) reported a mean flow rate of 265 mL/s for their combined polyp and nodule group across 14 male and 18 female speakers. In this study, normal speakers produced a mean flow rate of 144 mL/s. The magnitude of the increase of airflow rates

appears to depend on the severity of the lesion (Colton, et al., 2006). Electroglottograms show decreased closing times of the vocal folds and an irregular pattern, which is the most likely cause for the increase in airflow that accompanies mass lesions on the vocal folds.

### *Techniques for Measuring Phonation*

Analysis of vocal fold movement is physically difficult because of their location within the neck. Typical medical imaging approaches, such as x-ray and ultrasound, are ineffective for the vocal folds because the surrounding ring of cartilage forms a barrier, distorting the resulting picture. In spite of these physical limitations, there are several ways to indirectly measure phonation. Measurement procedures such as electroglottography, acoustic measurements, and endoscopy are available for clinicians to gain a more accurate picture of vocal fold movement. Each procedure yields a different piece of information about what occurs inside the larynx during phonation. A combination of measurement techniques will give researchers a more accurate assessment of vocal fold movement.

Electroglottography (EGG) measures change in electrical impedance when two electrodes are placed on opposite sides of the neck, close to the vocal folds. Changes in conductivity across the larynx can provide information on how large a portion of the vocal folds is touching at a given moment in time. This measurement can take place because tissue conducts electricity better than air, so impedance increases when the vocal folds separate and decreases as they come in contact with each other (Titze, 1994).

Acoustic measurements can be made to calculate cycle-to-cycle changes in frequency and amplitude that take place in response to the movement patterns of the vocal folds. In other words, measurements of acoustic output provide a numerical

representation of how the vocal folds are behaving during phonation. With the use of a microphone and voice analysis software, measurements of frequency and amplitude perturbation can be obtained.

Various types of imaging are available to researchers to provide a visual assessment of vocal fold activity. One tool which shows the basic movement of the vocal folds over time is videolaryngoscopy. A dynamic superior view of the vocal folds is provided by a rigid fiberscope, which is positioned at the back of the mouth in the oropharynx. One limitation of videolaryngoscopy is that the images seen by a clinician are not in real time. The strobe light gives the impression of slowed movement, but cannot show the sequential opening and closing phases of the vocal folds. Another imaging tool is the high-speed digital camera, which is capable of yielding thousands of frames per second. High-speed digital images are taken with a constant light source, so the consecutive still images allow for more precise understanding of the nature of movement of the vocal folds. Although novel methods of assessing vocal fold vibration, such as high-speed photography, videokymography, and photoglottography have emerged over the past two decades, laryngeal videostroboscopy remains the most practical and clinically useful tool in assessing vocal fold vibratory characteristics and glottal configuration (Johns, 2003).

### *Laryngeal Modeling Studies*

Historically, models have been developed to simulate vocal fold vibration in order to represent the movement of real vocal folds. The value of models is centered in the idea that they allow us to make predictions beyond what can be, or what has been measured. Measurements can tell us what is, and models can tell us what is possible in unexplored situations (Titze, in Stevens & Hirano, 1981).

*Excised larynx studies.* The canine larynx has been frequently used as a model of the human larynx (Berke et al., 1987; Durham, Titze, & Scherer, 1987; Kakita, Hirano, & Ohmaru, 1981; Slavit & McCaffrey, 1991; Yanagi, Slavit, & McCaffrey, 1991). However, when the canine laryngeal tissues are compared with human tissues, one difference becomes apparent; the canine does not have a well-established vocal ligament (Hirano, 1975). Other differences include the superficial layer, which is thicker in canines, and the lack of intermediate and deep layers in canine vocal fold tissue. It has been established that the canine larynx has no vocal ligament and a relatively thick mucosa. Furthermore, the vocalis muscle in the excised canine larynx does not contract (Titze, 1981). Titze surmised that the ligament in humans is crucial for sustaining high pitches. Canine species can initiate phonation, but are less able to sustain it, particularly at high pitches. These differences in canine and human vocal fold tissue suggest a need for better modeling techniques because sustained phonation is so difficult to produce for quality research.

Excised human larynges have also been used in research, but have been shown to have two distinct disadvantages. First, they are only able to phonate for a short amount of time. Second, they are not suited for parametric studies involving tissue geometry and stiffness.

*Synthetic vibrating vocal fold models.* Physical replicas of the human vocal system have been constructed more recently to simulate human phonation in order to overcome the challenges associated with direct *in vivo* measurements. Physical models are able to phonate for a long period of time, overcoming a major limitation presented by excised human larynges, which can only oscillate for approximately 30 minutes. Replicas

of the human vocal folds have given researchers increased opportunity to study oscillation characteristics. Physical models of the vocal folds have been used in research to study the vibratory characteristics of phonation (Berry, 2006; Berry et al., 2006; Drechsel & Thomson, 2008; Riede, Tokuda, Munger, & Thomson, 2008; Thomson, Mongeau, & Frankel, 2005; Zhang et al., 2006). These models have been refined in the past several years to allow more realistic movement patterns of the artificial vocal folds. One such improvement to this type of modeling is the development of a two-layer (body and cover; Hirano & Kakita, 1985) self-oscillating system (Drechsel, 2007; Riede et al., 2008; Drechsel & Thomson, 2008). Vocal fold modeling has also improved to allow researchers to quantify several parameters of normal vocal fold oscillation. Thomson, et al. (2005) studied the aerodynamic transfer of energy to the vocal folds in a self-sustained oscillatory model of the vocal folds. Riede et al. (2008), Dreschel (2007), and Dreschel & Thomson (2008) extended this research to include a more realistic body and cover model of the vocal folds.

In spite of the advances that have been made in the past several years, however, there are several characteristics of synthetic models that limit the degree to which they can accurately simulate human vocal fold movement. One limitation lies in the morphology of the vocal folds. Synthetic models have no vocal ligament and fewer layers than the tissue structure of human folds. Also, the geometry of the synthetic models is not completely realistic. Without the differentiated layers and equivalent shape of the human structures, the movement of the models may not reflect the exact movement patterns and the phasing subtleties of the normal vibratory cycle.

Another disadvantage to using synthetic models is that the silicone which



comprises the synthetic folds has a different composition than human tissue. Studies have shown a nearly linear stress-strain curve in tensile testing of synthetic folds (Riede et al., 2008); human tissue, on the other hand, reveals a non-linear response on the stress-strain plot as its tissue is stretched (Titze, 1994). Human tissue is essentially stronger, and shows more resilience to elongation.

A third issue that has yet to be resolved in synthetic vocal fold research is the lack of a mucosal wave, which is a key characteristic of healthy phonation in humans. This difference is likely due to the lack of lamina propria layers in synthetic models. Until a mucosal wave similar to that observed in human phonation can be realistically simulated in vocal fold models, it may be difficult to engage the interest of researchers who work in the area of human vocal pathology.

Even with the current differences between human and synthetic vocal folds, however, there are enough similarities between the two to give comparable results on a number of measures. For example, the use of vocal fold models can give valuable information about flow dynamics and the basic movement patterns of vocal folds. Observing the changes that take place when vocal fold geometry and composition are altered will increase our understanding of the parameters which lead to disordered vocal fold movement patterns. Measurements of velocity, airflow, and fundamental frequency in the context of normal and pathological vocal fold movement will improve our understanding of vocal fold oscillation and laryngeal function during phonation. The current study compared normal and pathological vocal fold movement, with the use of a self-sustained oscillatory model with added masses to simulate vocal nodules. With the information from this study, researchers may be better able to draw inferences about the

way changes in the makeup of vocal folds can contribute to pathological differences in the voice.

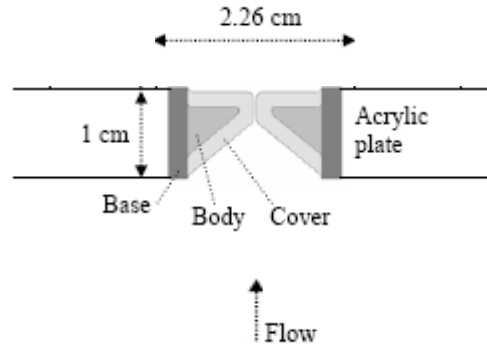
## Method

### *Materials*

A physical model of the human vocal folds was made to replicate the adult male larynx using a three-part addition-cure silicone material (single-part Silicone Thinner and two-part EcoFlex 0030, Smooth-On, Inc.). The vocal folds were a two-layer (body and cover) version of the Thomson et al. (2005) one-layer model, as discussed in Riede et al. (2008) and Drechsel & Thomson (2008). The construction and fabrication of the model has been discussed at length in Riede et al. (2008), and is summarized here.

The vocal folds were constructed with two layers of differing material, as illustrated in Figure 1. Silicone compound (Ecoflex 0030) was used to construct both the body and cover portions of the vocal folds. Varying ratios of Ecoflex 0030 part A, Ecoflex 0030 part B, and silicone thinner (see Table 1) were combined to achieve the differing silicone consistencies for the body and cover portions of the vocal folds, as the modulus of the cured silicone could be adjusted by varying the amount of silicone thinner used. The cover layer was approximately 2 mm thick, with a composition ratio of 1:1:4, and the body layer was made with a ratio of 1:1:2. A base layer was constructed with thick silicone compound (Dragon Skin Q) to serve as a foundation at the lateral portion of the folds (see Figure 2). The stiffness of this base was deemed to be sufficiently high, so as to not vibrate with the vocal folds themselves (Riede et al., 2008).

Fabrication of the molds for all experimental conditions involved computer-aided design (CAD) models, generated using Pro/Engineer and displayed in Figure 3. The body and cover portions of the vocal folds were then made in series, using these molds. The body of the vocal folds was created first, by pouring a 1:1:2 ratio silicone mixture into the body mold, and allowing approximately 6 hours to cure. After the body portion



*Figure 1.*

Schematic showing the approximate dimensions of the synthetic, two-layer vocal folds used in this study. The medial surfaces of the folds were rounded as shown, with a radius of approximately 0.33 cm. Adapted from Drechsel & Thomson (2008).

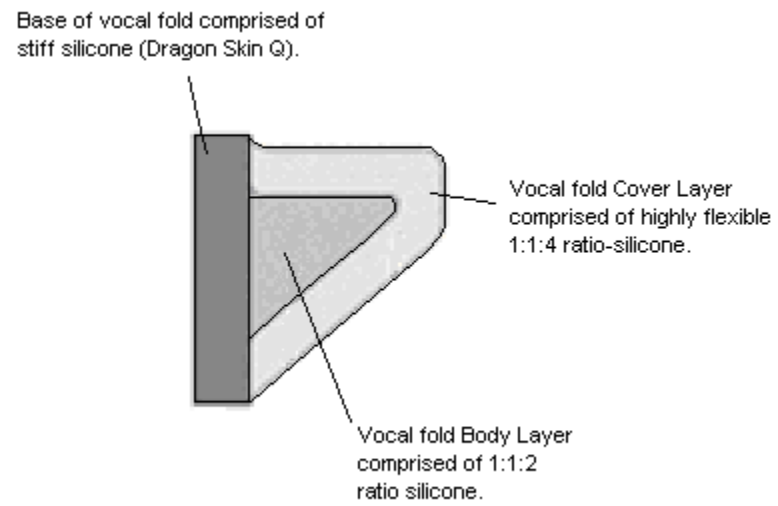
Table 1

*Silicone Ratios Used for Vocal Fold Construction*

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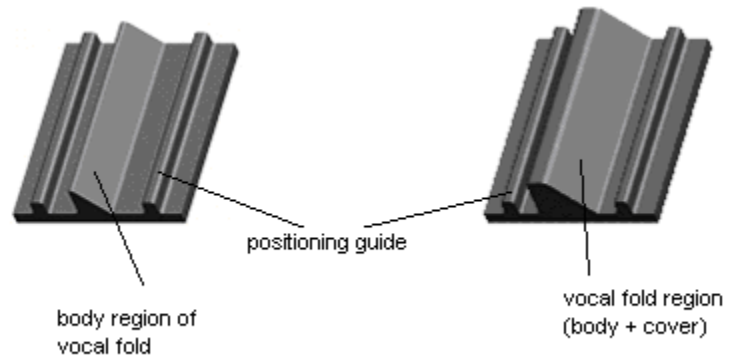
Item	Part B	Part A	Silicone Thinner
Body	1	1	2
Cover	1	1	4
Base	1	1	1
Nodules	1	1	0

---



*Figure 2.*

Single-fold coronal cross-section of Condition 1, with corresponding silicone composition ratios. The dark grey base layer forms the lateral anchor for the vocal folds.



*Figure 3.*

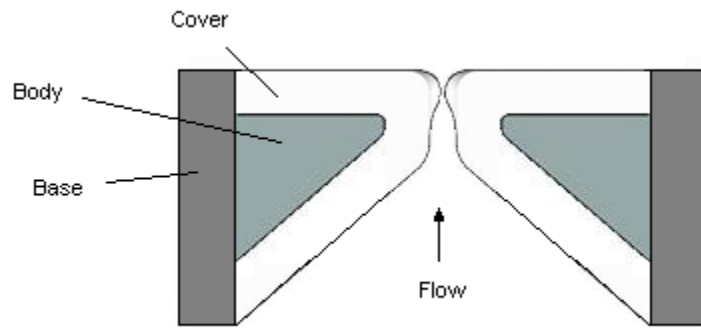
Computer-aided design models used for rapid prototyping and casting of the different layers of the vocal folds for Condition 1.

cured, stiff silicone mixture (Dragon Skin Q) was poured into the mold to create the base of the vocal folds and was allowed 1-2 hours to cure. A 1:1:4 ratio silicone mixture was then poured into the cover mold. The body and base portion were set into the cover mold to bond the body portion with the cover portion. These were allowed to cure for 24 hours. Once this combination body/cover model had bonded and cured, it was removed from the mold and the model was cut into two 1.7 cm lengths to act as symmetric vocal folds. Lateral and dorso-ventral surfaces of the vocal fold models were attached to an acrylic plate using liquid silicone adhesive (Pro Bond®, Elmer's Products, Inc.).

Mass lesions similar to vocal fold nodules were cast using a ratio of 1:1:0 silicone mixture. No silicone thinner was used for nodule conditions in order to simulate a tougher, more fibrous nodule on each vocal fold. The nodule-like protrusions were given a broad base, and were assigned a diameter size of 2 mm, with a thickness equal to that of the lamina propria, based on measurements of adult nodules as provided by Dikkers (1994). The nodules were placed halfway between the anterior and posterior surfaces of the synthetic model, corresponding to the center of the membranous vocal fold where maximum impact from vibration takes place (Titze 1994). The nodules were not placed on the anterior-third boundary of the vocal folds for the purposes of this project, because the point of maximum impact in the synthetic model was in the center of the vocal folds.

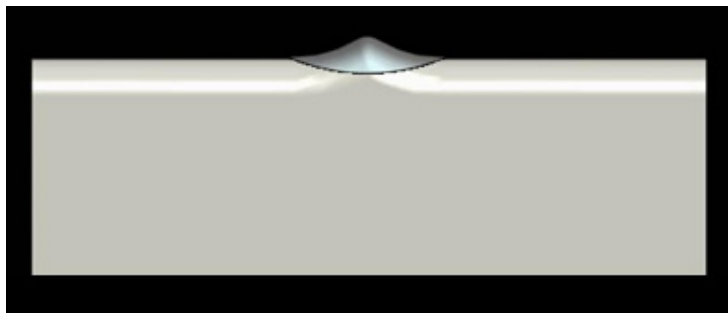
Three experimental conditions were simulated and tested. In Condition 1, “healthy” vocal folds were simulated, in which the cross-section was uniform in the dorso-ventral direction (see Figure 2). In Condition 2, young nodules were simulated by using a small mass of fibrous material with a broad base on the surface of the cover layer (Figures 4 and 5). In Condition 3, older nodules were simulated by using a larger base of





*Figure 4.*

Coronal cross-section of Conditions 2 and 3, with nodule masses protruding from the upper border of the vocal fold cover.



*Figure 5.*

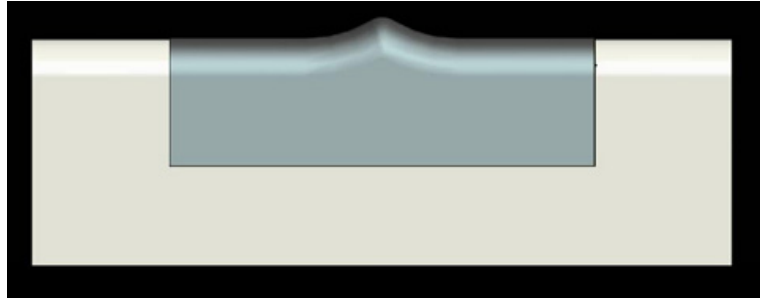
Superior view of Condition 2, in which the nodule mass (dark grey area) is restricted to the immediate area around the swelling on the vocal fold.

fibrous material that extended through the 2 mm cover layer (see Figure 6). Both nodule conditions (Conditions 2 and 3) were assigned the same nodule diameter on the surface of the fold; however, Condition 3 had more mass beneath the surface to simulate extensive vocal abuse characterized by fibrosis, as is present with persistent nodules.

For each experimental condition, the medial surfaces of the vocal folds were positioned to simulate a semi-closed glottis when no airflow was being applied to the model, by assigning a pre-phonatory width of 0.5 mm. This light approximation of the folds allowed room for nodules to lightly touch each other without creating excessive medial tension to skew the measurements. A 2.5 cm uniform polyvinylchloride (PVC) tube was connected to an expansion chamber to simulate the subglottal system. This tube was then connected to an air supply, with shop air used as a flow source (see Figure 7). A pressure regulator (Pneufine 26129-1C-19, CKD Corp) was used to reduce pressure instability from the air supply, and a flow meter (Omega FL4611) was attached to calculate the rate of airflow from the source. Subglottal pressure was monitored using a differential pressure transducer (Omega PX138-001D5V) placed inside the PVC pipe directly below the vocal folds, and displayed on a process meter (Omega DP24-E).

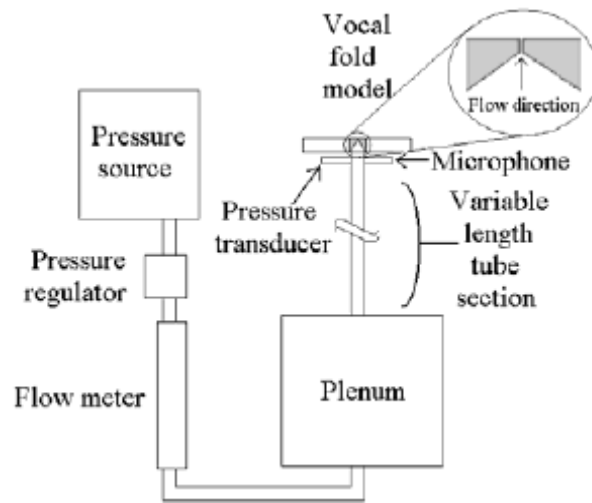
For all experimental conditions the pressure was gradually increased until the model began oscillating. This pressure level was recorded, along with the model's  $F_0$  and air flow rate at onset. Pressure was then increased to allow for acoustic testing at the same pressure for each model in a condition. Once these data were obtained, pressure was incrementally reduced until the model discontinued self-oscillation.

A high speed camera (Photron APX-RS) was used to record images of the physical model during experimentation at a rate of 4,000 frames per second with a 1024 x



*Figure 6.*

Superior view of Condition 3, in which the nodule mass extends dramatically along the length of the fold and into the cover layer



*Figure 7.*

Test setup used in this study. From Drechsel & Thomson (2008).

768 pixel resolution. A microphone was attached above the vocal folds to record changes in  $F_0$  during oscillation (see Figure 7). A Particle Image Velocimetry (PIV) system (LaVision) was used to obtain velocity measurements from the experimental conditions.

### *Testing Procedure*

First, phonation onset pressure (onset), fundamental frequency ( $F_0$ ), and flow were measured for the three separate models in Condition 1 (each model was tested individually). Next, the pressure was increased to 0.90 for microphone signal recording and high-speed camera recording. Acoustic data were captured in a .WAV format, with approximately 6 seconds of recording time per model. The high-speed camera recorded approximately 10 cycles of oscillation for visual perceptual analysis of vocal fold movement. The pressure was then reduced incrementally, until phonation offset pressure (offset) was obtained. The stiffness and length of the vocal folds were kept constant, as these factors are considered to be major causes in changing  $F_0$ . All models in Condition 1 were brought to a pressure of 0.90 kPa for microphone signal recordings and high-speed camera recording.

Each model in Condition 2 ( $N = 4$ ) was then recorded in succession, with the same setup and order of experiments used for Condition 1 testing. Once onset was established, pressure was brought to 0.90 kPa for acoustic and high-speed recording. Pressure was decreased until offset was recorded.

Once Condition 2 experimentation was complete, the models from Condition 3 were tested in succession ( $N = 3$ ). Similar testing procedures were followed; however, the pressure required for oscillation in Condition 3 was so high that the 0.90 kPa pressure used in Conditions 1 and 2 was inadequate for vibration. Therefore, acoustic and high-

speed camera data were recorded at a pressure of 1.90. Pressure was then decreased slowly until offset was reached.

### *Data Analysis*

Onset pressure was obtained and recorded for the normal and nodule conditions. Air pressure was kept at onset, initially, in order to compare frequency and vibratory characteristics between the three conditions at onset. Once phonation began, pressure and airflow data were recorded. Pressure was then raised to 0.90 kPa for Conditions 1 and 2, and 1.90 kPa for Condition 3, for the remainder of testing. A microphone recorded the acoustic output for each model, and this signal was used to obtain information about the frequency of oscillation, harmonic spectrum, and perturbation of amplitude and frequency. The microphone signal from each model was analyzed using TF32, a time-frequency analysis software program. The final measure taken of each model was offset, which was made by turning down the pressure until the point at which the model discontinued oscillation, and recording the corresponding pressure level. All measurements were taken in a laboratory with ambient noise levels below 70 dB SPL (C-weighted) during experimentation. Measurements were taken with the normal model (Condition 1), and were compared with measurements taken from the two disordered systems (Condition 2 and Condition 3).

## Results

To determine whether there was a significant difference across the three conditions, a one-way ANOVA was performed on the dependent measures. A series of Bonferroni post hoc tests was subsequently used to determine which conditions were different from each other. Descriptive statistics (mean and standard deviation) for the dependent variables of phonation onset pressure, phonation offset pressure, jitter, shimmer, signal-to-noise ratio, air flow, and frequency can be found in Table 2 for Condition 1, Table 3 for Condition 2, and Table 4 for Condition 3. Results from the one-way ANOVA ( $F$ -ratios,  $p$ -values, and effect sizes for significant main effects) for the variables that changed significantly across conditions can be found in Table 5. The means and standard deviations for these changes can be seen in Figures 8, 9, 10, and 11.

### *Condition 1*

Phonatory onset pressure, or the pressure required for oscillation to start, was calculated first. The models in Condition 1 required an average of 0.76 kPa to begin oscillation. Flow rate had a mean of 1.24 liters per second (L/s).

The  $F_0$  of Condition 1 models ranged from 123.3 to 126.7 Hz at onset. This  $F_0$  is comparable to the average  $F_0$  of an adult male. When pressure was increased, all models demonstrated a slight increase in  $F_0$ .

The acoustic signal yielded measures of frequency and amplitude perturbation (jitter and shimmer, respectively), which are commonly calculated in the field of speech pathology as a means of objectively characterizing cycle-to-cycle instabilities in the voice. Three jitter and shimmer measurements were taken from each model's acoustic signal. These measurements were compared in each model to ensure the model did not



Table 2

*Descriptive Statistics for Condition 1*

---

Item	M	SD	Range
Jitter %	1.01	0.62	1.24
Shimmer %	5.13	0.53	1.03
SNR	17.97	0.60	1.20
Onset (kPa)	0.76	0.10	0.20
Offset (kPa)	0.60	0.14	0.27
Frequency (Hz)	124.77	1.75	3.40
Flow (L/s)	1.24	0.02	0.04

---

Table 3

*Descriptive Statistics for Condition 2*

---

Item	M	SD	Range
Jitter %	1.03	0.27	0.57
Shimmer %	5.33	1.71	4.12
SNR	18.55	2.63	5.90
Onset (kPa)	0.77	0.09	0.23
Offset (kPa)	0.58	0.13	0.30
Frequency (Hz)	125.63	1.39	3.30
Flow (L/s)	1.53	0.22	0.47

---

Table 4

*Descriptive Statistics for Condition 3*

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Item	M	SD	Range
Jitter %	0.80	0.29	0.39
Shimmer %	3.96	1.08	2.16
SNR	18.43	3.01	5.40
Onset (kPa)	1.69	0.34	0.60
Offset (kPa)	1.23	0.33	0.65
Frequency (Hz)	134.03	3.26	6.40
Flow (L/s)	2.26	0.37	0.66

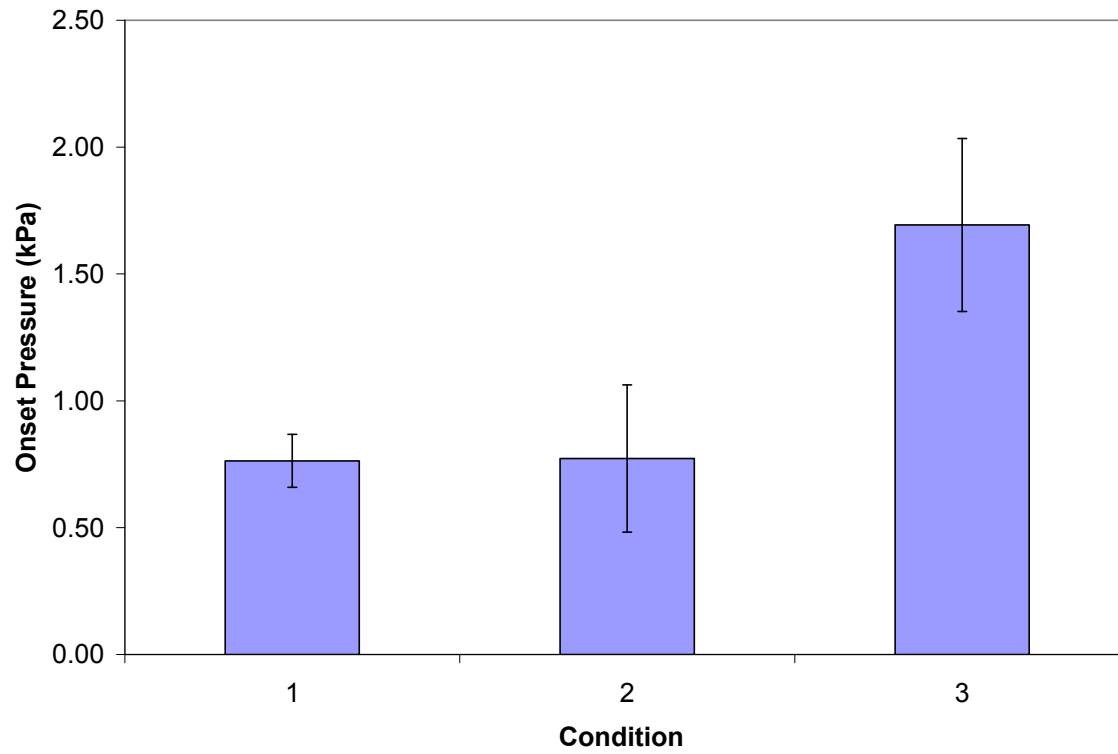
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Table 5

*Repeated Measures ANOVA and Bonferroni Post Hoc Contrasts for Variables Which Changed Significantly Across Condition.*

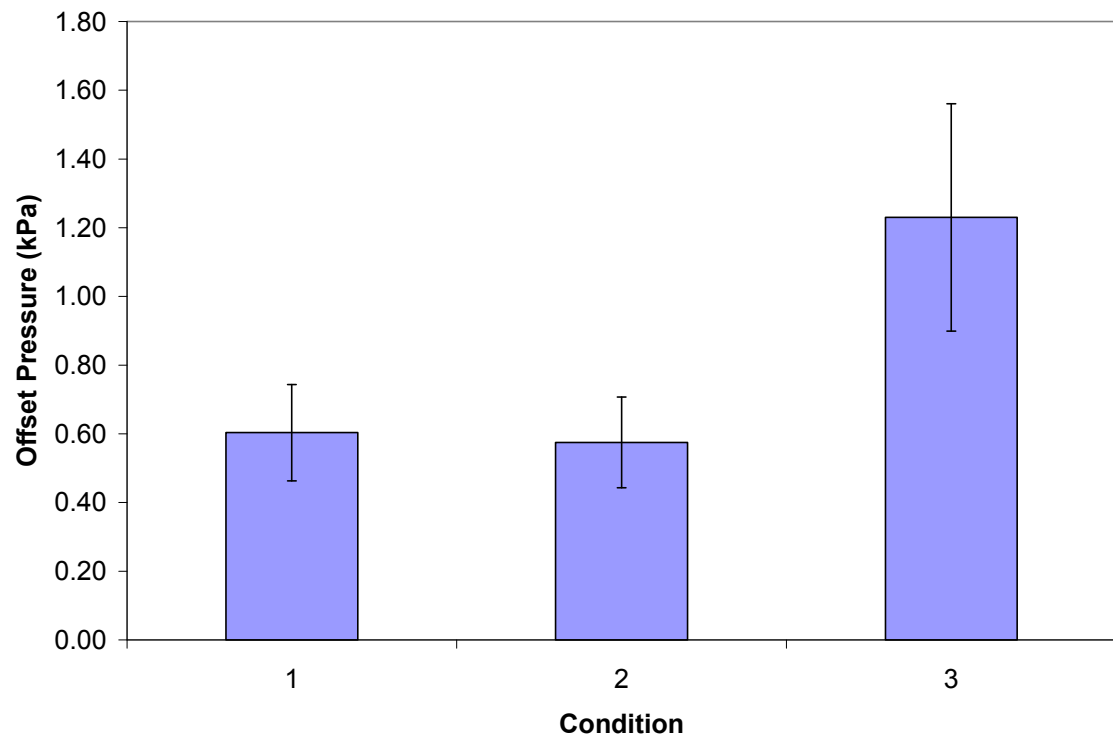
Condition	Overall ANOVA		1 vs. 2	1 vs. 3	2 vs. 3
Variable	<i>F</i> -ratio	<i>p</i> -value	<i>p</i> -value	<i>p</i> -value	<i>p</i> -value
Onset Pressure	22.402	0.001**	1.000	0.002**	0.002**
Offset Pressure	9.809	0.009**	1.000	0.025*	0.014*
Onset Frequency	17.174	0.002**	1.000	0.004**	0.004**
Onset Flow	13.813	0.004**	0.500	0.004**	0.018*

*Note.* Degrees of freedom are 2, 7. \* $p < .05$ . \*\* $p < .01$ .



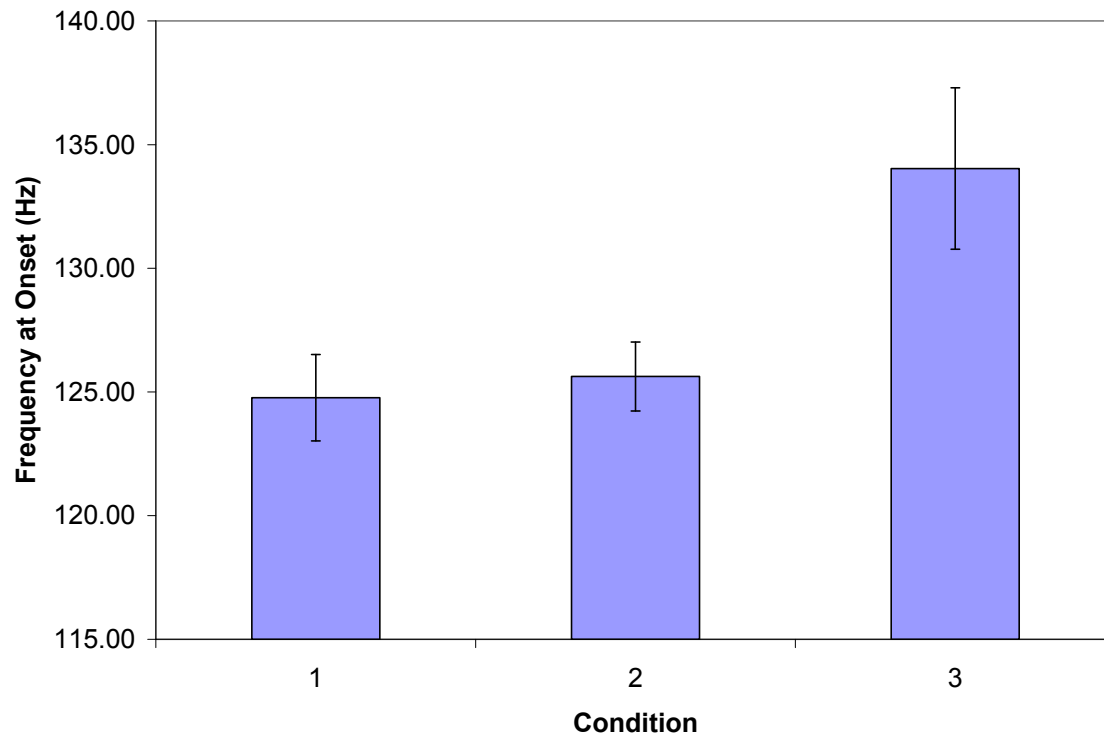
*Figure 8.*

Mean (and standard deviation) onset pressure for each of the three conditions.



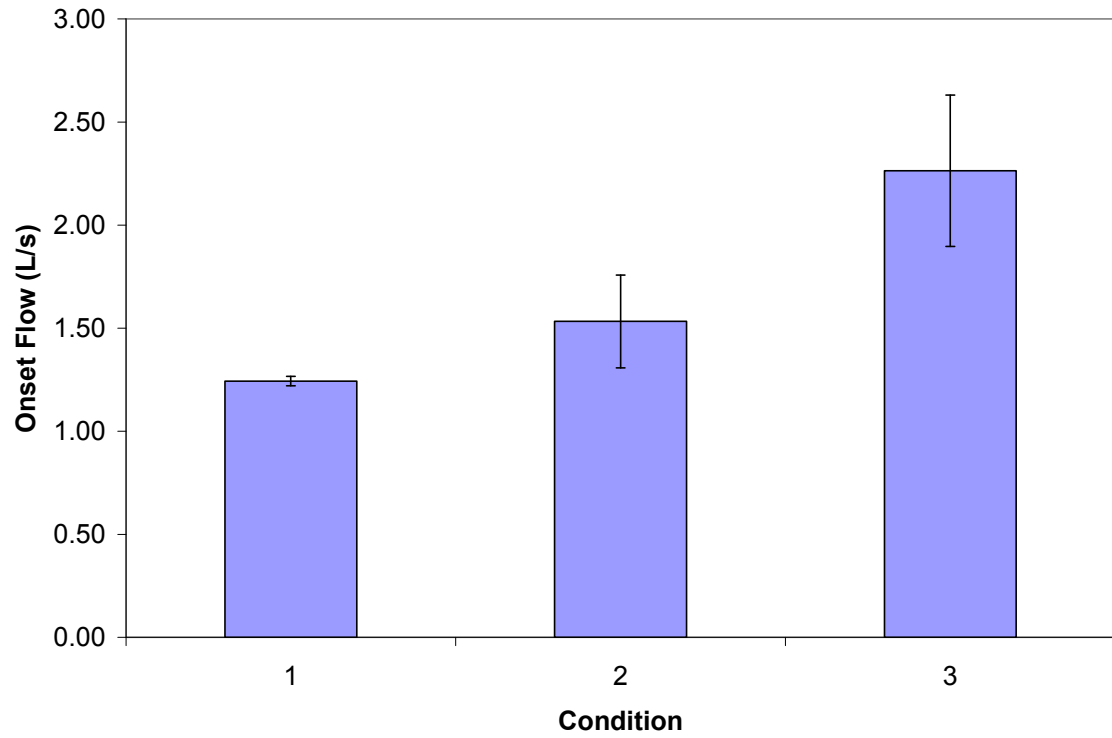
*Figure 9.*

Mean (and standard deviation) offset pressure of each of the three conditions.



*Figure 10.*

Mean (and standard deviation) onset frequency of each of the three conditions.



*Figure 11.*

Mean (and standard deviation) onset flow of each of the three conditions.



exhibit excessive variability in perturbation over time. Average jitter for all models in Condition 1 was 1.01 %. Average shimmer for models in Condition 1 was 5.13%.

Signal-to-noise ratio (SNR) was also calculated, as an additional measure of the model's periodicity. The average SNR for models in Condition 1 was 17.97.

Offset pressure (the pressure at which models ceased vibrating after being set in motion) was calculated at the conclusion of testing for each model and had a mean of 0.60 kPa.

Closure patterns and regularity of oscillation were reviewed with images from the high-speed camera. Models in Condition 1 demonstrated a complete closure pattern, with nearly-symmetric movement of the vocal folds (see Figure 12). Excursion was measured at the point when the folds were farthest away from midline. Excursion for Condition 1 was approximately 4 mm.

### *Condition 2*

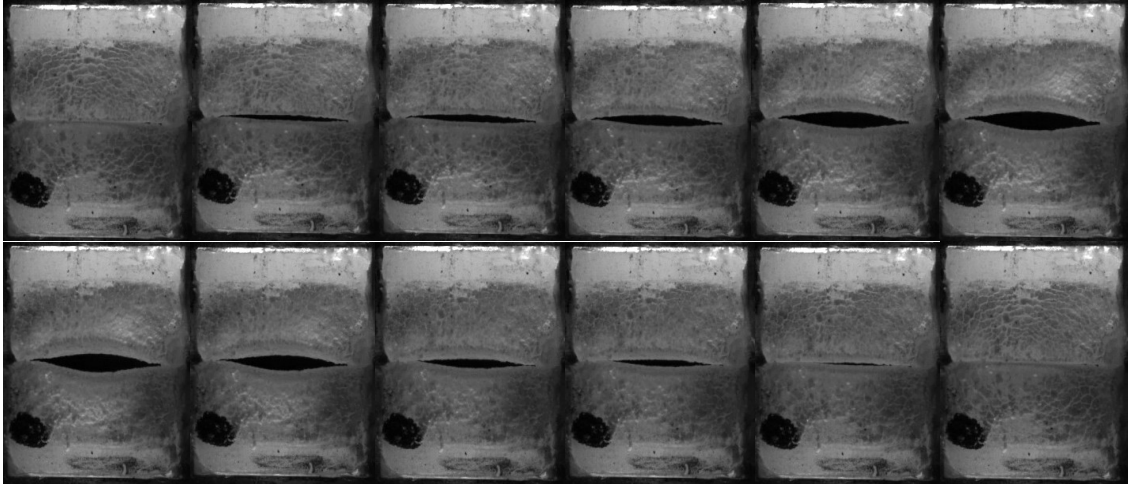
Onset pressure for models in Condition 2 ranged from 0.66 to 0.89 kPa, with an average onset of 0.77. Flow at onset for these models had a mean of 1.53 L/s.

The  $F_0$  of Condition 2 models ranged from 123.7 to 127.0 Hz at onset, with a mean of 125.6 Hz.

The models in Condition 2 had mean jitter of 1.03%, shimmer of 5.33%, and SNR measures of 18.55.

At offset, models ceased vibrating at an average pressure of 0.58 kPa.

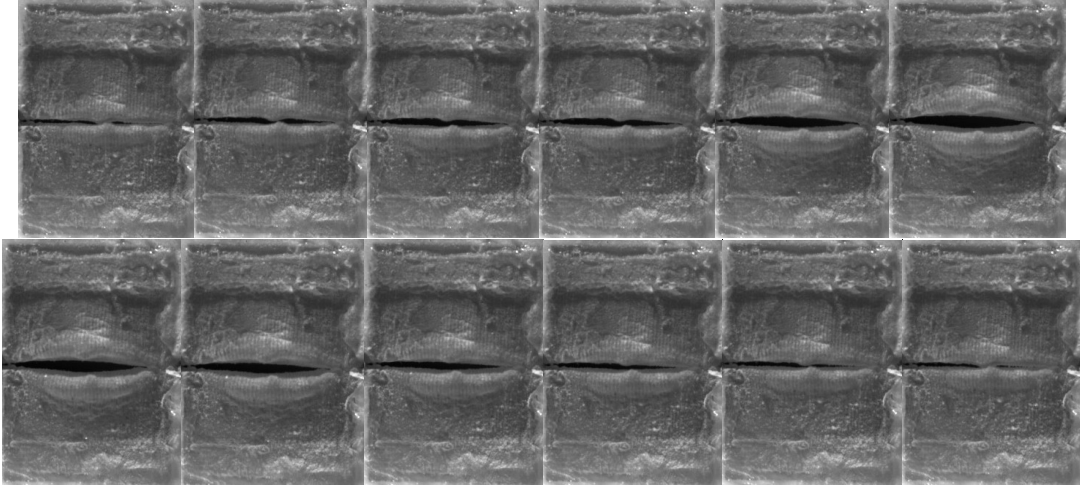
The vocal folds in Condition 2, which can be seen in Figure 13, have a smaller degree of excursion from midline than what is present in the models in Condition 1, with approximately 2.5 mm of space between the folds at excursion. Closure pattern is noted



*Figure 12.*

Still images taken from a high-speed camera recording of a model in Condition 1.

Maximum excursion is approximately 4 mm. A complete closure pattern can be seen, along with the nearly-symmetric movement of the folds during oscillation.



*Figure 13.*

Still images taken from a high-speed camera recording of a model in Condition 2. There is a small degree of excursion from midline, with approximately 2.5 mm between the folds at the most open point. Masses representing early-stage bilateral nodules are present. The closure pattern is incomplete at one end. Movement of the folds during oscillation is slightly asymmetric.

to be incomplete at one end. Movement of the folds during oscillation is fairly regular, with little asymmetry noted.

### *Condition 3*

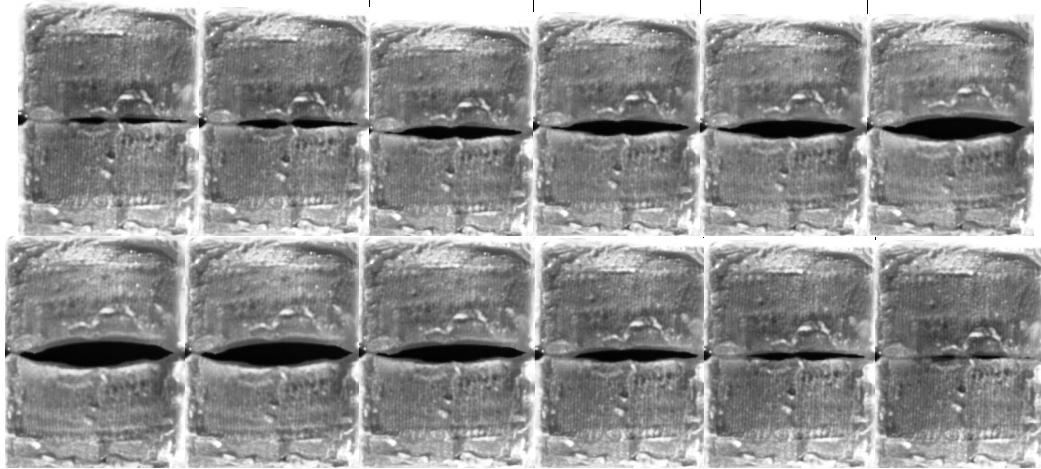
Onset pressure for models in Condition 3 ranged from 1.30 to 1.90 kPa, with an average onset of 1.69. Flow at onset for these models had a mean of 2.27 L/s. Mean onset pressure for this condition was found to differ significantly from the average performance of models in Condition 1 ( $p = .002$ ) and Condition 2 ( $p = .002$ ).

The  $F_0$  of Condition 3 models ranged from 131.2 to 137.6 Hz at onset, with a mean of 134.0 Hz, which was significantly higher than the onset frequency of Condition 1 ( $p = .004$ ) and Condition 2 ( $p = .004$ ).

The models in Condition 3 had mean jitter of 0.80%, shimmer of 3.96%, and SNR of 18.43, when measured at a pressure of 1.90 kPa. These results were not significantly different from means obtained in Conditions 1 or 2.

Upon offset, models ceased vibrating at an average pressure of 1.23 kPa. Mean offset pressure for models in Condition 3 differed significantly from that of Condition 1 ( $p = .025$ ) and Condition 2 ( $p = .014$ ).

The still images taken from high-speed recording of a model in Condition 3 show several irregularities (see Figure 14). A great deal of excursion from midline can be seen, with approximately 6 mm of space between the folds at maximum excursion. The closure pattern of these folds is noted to be relatively complete at both ends of the vocal folds. Movement of the folds is irregular, with asymmetries present during opening and closing phases of oscillation.



*Figure 14.*

Still images taken from a high-speed camera recording of a model in Condition 3.

Chronic bilateral nodules are simulated, with a broad base of stiffened material along the edges of the vocal folds. A large excursion from midline is noted, with approximately 6 mm of space between the folds at the point of maximum excursion. The closure pattern is relatively complete at both ends of the folds. Movement of the folds is fairly regular, without a great deal of asymmetry present during opening and closing phases of oscillation.

## Discussion

The purpose of the current study was to investigate differences between normal and disordered vocal fold activity in a synthetic model. This involved assessment of several vocal measures.

### *Airflow*

Colton, et al., (2006) reported that airflow rates in the disordered larynx appear to depend on the severity of the lesion on the vocal fold. Several studies, including the current investigation, have supported this idea. Tanaka and Gould (1985) reported a mean value of 275 mL/s in two patients with nodules, whereas normal male speakers produce flows of approximately 125 mL/s. Woo, Colton, and Shangold (1987) reported a mean flow rate of 265 mL/s for their combined polyp and nodule group across 14 male and 18 female speakers. Normal speakers produced a mean flow rate of 144 mL/s. The current study yielded a mean flow rate of 1.24 L/s for vocal folds in Condition 1. Models in Condition 3 demonstrated a much higher mean flow rate (2.27 L/s).

Several factors may contribute to higher flow rates when lesions are present on the vocal folds. One such factor is the tendency of vocal folds with nodules to show an incomplete closure pattern. Extra air leaks through the larynx during phonation when the vocal folds do not fully approximate. It is well documented that patients with nodules frequently have incomplete closure patterns during phonation, such as a posterior gap or hourglass closure pattern (Park & Mongeau, 2008). Air leakage through any gaps in the folds occurs during the closed phase of the phonatory cycle and creates higher rates of airflow.

Nodules also rarely match each other in terms of mass and size and therefore contribute to some degree of aperiodicity, as each fold vibrates in a slightly different

phase (Case, 2002). Phase differences can result in an increase in airflow during phonation.

Another contributor to elevated airflow rates in patients with nodules is the degree of stiffness present in the folds when nodules are present. With a set of stiffened folds, pressure from the lungs must be higher to achieve onset of phonation. Airflow levels are then much higher during phonation, because of the need for so much pressure to sustain phonatory movement in the larynx.

### *Onset Pressure*

In the current study, models in Condition 1 had a lower onset pressure than models with nodules in Conditions 2 and 3. Onset pressure appears to be positively associated with variables of stiffness and mass, since an increase in either property led to an increase in the pressure needed for vibration to commence in this study.

Scherer (1991) reviewed other factors that increase onset pressure for human vocal folds. The degree of inferior convergence of the folds has been documented as one feature that impacts onset pressure. If vocal folds are closer to each other at their lower borders, onset pressure is decreased. The current study controlled for this variable by setting vocal fold models the same distance from each other throughout the experiments.

Another feature that can contribute to onset pressure variability is the vertical height of the folds. If the height of the vocal folds increases, less pressure may be needed to allow the vocal folds to oscillate. However, this variable was also controlled in the current study and did not likely influence the onset pressure, either within or between conditions.

The third factor that can affect onset pressure is tissue damping, which accounts for friction in the folds. Scherer (1991) suggested that if the vocal fold tissue has a low

level of viscosity or friction, it has more freedom to move, which leads to a lower subglottal pressure needed to set the folds in motion. This factor is one that can be influenced by the differences in tissue that were present in the three conditions tested in the current study. Vocal folds in Condition 1 had a low viscosity with the composition of the vocal fold cover layer that allowed a great deal of free movement. The folds in Condition 2 had a slight increase in viscosity when the small volume of stiffened mass was added to them. This same principle applies to the folds in Condition 3. They contained a larger stiffened mass along the medial edges of the folds, and thus greater friction; this increased the pressure needed to achieve the onset of vibration.

### *Frequency*

Pitch, the perceptual correlate of frequency, has been shown to change as a function of vocal fold length and tension, subglottal pressure, amplitude of motion of the vocal folds, and activity of the thyroarytenoid muscle (Scherer, 1991).

The fundamental frequency of an object in motion is dependent on multiple factors. Mass and length of the vocal folds can greatly contribute to changes in vibratory frequency. Larger vocal fold mass generally yields a perceptually lower voice with its low fundamental frequency. An increase in airflow through the glottis is usually accompanied by an increase in fundamental frequency ( $F_0$ ). This is due to changes that occur to the vocal folds themselves as they are displaced during more intense phonation. As the vocal folds are moved further away from the midline during periods of loud phonation, their degree of displacement is increased, and the folds are stretched, resulting in smaller vocal fold cross-sectional mass and increased stiffness; thus, there is an increase in the frequency of vibration (Titze, 1989).



The current study revealed an increase in  $F_0$  when stiffened mass lesions were added to the vocal folds. Even though an increase in mass is generally associated with a lower  $F_0$ , the models in Condition 3 of this study were displaced sufficiently to create an increase in  $F_0$  despite the addition of the mass lesions. Because the models in Condition 3 had such a great deal of stiffness along the cover layer of the vocal folds, subglottal pressure had to be increased substantially to induce oscillation. The dramatic increase in air pressure needed to sustain oscillation in Condition 3 created a greater degree of displacement than that noted in Conditions 1 and 2, and therefore, a higher  $F_0$ .

Several previous studies have shown that the speech signal's fundamental frequency increases when nodules are present (Niedzielska et al., 2001; Niedzielska, 2005). This is most likely due to the modification of the airflow through the larynx that results from the pathology (Scalassara et al., 2007).

### *Perturbation*

Previous studies have not reported vocal perturbation data for physical models of the vocal folds. In studies of human phonation, jitter and shimmer are often computed to reveal instability in the voice, which can be a clinical indicator of possible vocal fold pathology. In the human voice, the likelihood of pathology increases when jitter and shimmer percentages increase. In general auditory perceptual terms, moderate amounts of jitter and shimmer are associated with "roughness" of the voice, whereas in physical terms, perturbation is associated with vocal fold vibration instability (Murphy, 2000).

Jitter and shimmer in the current study are rather interesting to note because of their difference from what one would expect from human voice samples. A typical speaker without laryngeal disorder should be able to generate a vowel prolongation with very little jitter, usually less than 1%. As jitter values increase beyond this 1% level, the

voice is perceptually more dysphonic or rough. In human speakers with no laryngeal pathology, average jitter measures are typically below 1% (Case, 2002). Shimmer is another acoustic measure giving information about cycle-to-cycle perturbation of intensity in a voice. Typical speakers, holding a vowel sound as steady as possible, should have little variation in intensity with each cycle of vibration.

Voices of persons with vocal nodules usually show above normal levels of jitter and shimmer (Case, 2002). Increases in nodule size and mass typically lead to elevated jitter and shimmer measures. However, in the current study, the mean jitter values did not statistically differ from one condition to the next, and were actually slightly lower in Condition 3, in which the nodule mass was much higher. Mean shimmer followed a similar pattern; there were no significant differences across conditions, and values were also somewhat lower in Condition 3. These findings might be explained by the structural symmetry of the synthetic folds. Tissue asymmetry may be present in human vocal folds with nodules or polyps, and this may lead to the increased perturbation measures found for such speakers. Typical vocal nodules, while bilateral, will not be as symmetric in composition as the simulated mass lesions in this study. The models in Condition 3 had larger masses, but since they essentially mirrored each other in their mechanical properties, the folds moved in relative symmetry. The identical simulated nodules allowed a relatively uniform movement pattern for each fold during oscillation, which likely led to modest perturbation measures. The regularity of movement from models in the current study is one explanation for the disparity in these findings relative to human speakers. A potentially valuable area for future study would be to investigate asymmetrical vocal fold movement in physical models by deliberately altering the

physical properties of one fold, in order to learn whether such a model would behave in a way that is similar to phonation in human speakers with vocal fold pathology.

### *Signal-to-Noise Ratio*

One voice characteristic of those with vocal nodules is a lowered harmonic-to-noise ratio, or SNR (Case, 2002). In general auditory perceptual terms, high levels of noise are associated with breathiness, and in physical terms, noise is associated with turbulent flow at the glottis (Murphy, 2000). Typically, perceptually breathy voices are associated with vocal nodules or some other form of pathology that interferes with full approximation of the folds during the closed phase of the vibratory cycle. Previous studies have shown that the normal range of SNR for the human voice is from 9-30 dB (Klingholz & Martin, 1985); however, it is widely accepted in voice research that SNR is typically significantly lower for individuals with vocal fold pathology. For example, Zhang and Jiang (2006) reported SNR measures from 7.98 to 15.4 in speakers with laryngeal pathology, and measures from 21.9 to 26.8 in speakers with no laryngeal pathology.

The results from the current study are in contrast to data obtained from human speakers, in that they do not show significant differences in SNR values between conditions. Condition 1 models had a mean ratio of 17.97, and Condition 3 models had a slightly higher mean SNR of 18.43. Several factors may have contributed to these results. The symmetry of the synthetic folds during oscillation may have prevented any difference in SNR between conditions. As discussed previously, the nodules in a human system are often asymmetrical in terms of mass and size. The nodules in the current study were symmetrically cast, and may have introduced less variability in the model system than would be expected in an actual pathological vocal fold condition. Although an

increase in mass was sufficient to create significant differences in onset pressure, frequency, flow, and offset pressure between conditions, the symmetry of the movement patterns in all 3 conditions was enough to ensure stability of SNR, jitter, and shimmer.

Another factor to consider is the stiff silicone-filled area from Condition 3. Its dimensions were unlike anything that would be anticipated in a human larynx. The physical properties of these models, created to help visualize movement patterns of a disordered human system, may vary significantly from those of human speakers who have nodules or polyps. The “nodule area” created for the models in Condition 3 was geometrically unrealistic due to the rectangular shape that the nodule-stiffened area takes up on these models (see Figure 6). Therefore, the SNR observed in this study may not easily generalize to the results that might be observed in studies including speakers who have mass lesions on their vocal folds.

Future studies in vocal fold modeling with mass lesions should test samples from the population of people with nodules and polyps, and compare their performance on SNR and other acoustic measures to samples of people from the population without vocal pathology. These data could then be compared with vocal fold models made to simulate each of these conditions. It would also be beneficial to refine the current model to experiment with asymmetries of size, shape, and stiffness of the simulated nodules, in order to more accurately reflect the disorder data from human voice research.

Other pathological conditions could also be simulated as models become more refined in the future. For example, models could be created to simulate conditions such as Reinke’s edema, unilateral vocal fold paralysis, and vocal polyps. Further research might also focus on measurement variables that were beyond the scope of the current study,

such as particle image velocimetry, Young's modulus, and more in-depth use of high-speed imaging techniques in movement analysis. Different views of the vocal folds could also be recorded, giving a thorough look at vocal fold movement from inferior and transverse angles.

Another direction to follow in future research would be to make modifications to Condition 1 to allow for a more realistic representation of the individual layers of the lamina propria and epithelial tissue. As mechanical models are refined and show more realistic movement patterns, we come closer to being able to use these models in a clinical context and potentially even improve diagnostic practices in the voice clinic.

### *Conclusion*

While the present work demonstrates the basic feasibility of using physical models to better understand disordered human vocal fold behavior, it is clear that further research is needed to make refinements to the models. This preliminary study of the potential to model voice disorders suggests that modeling may be extended to a broader range of phonatory conditions and pathologies in the future. Because modeling research continues to become more refined, a long-term goal for similar research would be to develop a comprehensive laryngeal system that yields new information about glottal aerodynamics and structure. Hopefully, progress in synthetic models along with human subject studies will provide this type of definitive data in the near future. In this way, insight into specific physiological patterns of vocal folds with numerous pathological conditions can be gained to further our understanding of the complexities of the human voice.

Despite some inherent limitations of this study, the results provide a foundation for further research regarding the effect that mass lesions might have on typical patterns of voice production.

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