Characterization and Improvement of the Clinical Assessment of Vocal Hyperfunction

by
Cara Elizabeth Stepp

S.B. Engineering Science
Smith College, 2004

S.M. Electrical Engineering and Computer Science
Massachusetts Institute of Technology, 2008

SUBMITTED TO
THE HARVARD-MIT DIVISION OF HEALTH SCIENCES AND TECHNOLOGY
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY IN BIOMEDICAL ENGINEERING

AT THE
MASSACHUSETTS INSTITUTE OF TECHNOLOGY
SEPTEMBER 2009

Signature of Author .....
Harvard-MIT Division of Health Sciences and Technology
July 29, 2009

Certified by .....
James T. Heaton, Ph.D.
Assistant Professor, Harvard Medical School
Thesis Supervisor

Accepted by .....
Ram Sasisekharan, Ph.D.
Director, Harvard-MIT Division of Health Sciences and Technology
Edward Hood Taplin Professor of Health Sciences and Technology and Biological Engineering

Characterization and Improvement of the Clinical Assessment of Vocal Hyperfunction

by

Cara Elizabeth Stepp

Submitted to the Division of Health Sciences and Technology
on July 31, 2009
in Partial Fulfillment of the Requirements
for the Degree of Doctor of Philosophy in Biomedical Engineering

ABSTRACT

Vocal hyperfunction refers to "conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or 'imbalanced' muscular forces" (Hillman, Holmberg, Perkell, Walsh, & Vaughan, 1989), characterized by excessive laryngeal and paralaryngeal tension (Aronson, 1980; M. D. Morrison, Rammage, Belisle, Pullan, & Nichol, 1983; N. Roy, Ford, & Bless, 1996). There is no widely accepted diagnostic measure of the presence and degree of vocal hyperfunction, and currently, assessment during diagnosis is often primarily based on subjective impressions given the patient's history and presentation of symptoms such as auditory-perceptual and visual or tactile discrimination of muscle tension (e.g., laryngeal palpation). Clinical care is hindered by the lack of a "gold standard" objective measure for the assessment of vocal hyperfunction.

The first study in this thesis evaluated a novel experimental design for the study of vocal hyperfunction, making use of the established clinical procedure of injection laryngoplasty. This work found that the use of injection laryngoplasty as a platform for the study of some types of vocal hyperfunction is limited, but may offer a convenient opportunity to study selected associated parameters. Particular promising objective measures were investigated in the remaining four studies: kinematics of the vocal folds, root-mean-squared (RMS) measures of surface electromyography (sEMG), and spectral characteristics of sEMG. Kinematic features of vocal fold abduction and adduction were shown to discriminate between individuals with muscle tension dysphonia and controls. RMS measures of sEMG were investigated through correlation with current clinical neck palpation techniques in voice therapy patients and via a cross-sectional study of individuals with vocal fold nodules. Correlations between RMS neck sEMG and palpation ratings were low, and although some individuals with nodules displayed RMS neck sEMG patterns that were inconsistent with those seen in controls, overall the RMS measures were unable to discriminate between disordered and control groups. Mean coherence between two neck sEMG locations in individuals with vocal nodules was significantly lower in the 15 – 35 Hz band relative to controls, possibly agreeing with past subjective accounts of "imbalanced" muscle activity.

Thesis Supervisor: James T. Heaton, Ph.D.
Title: Assistant Professor, Harvard Medical School

Acknowledgments

First, James T. Heaton, for being the best advisor imaginable – smart, caring, and fun. Thanks for pushing me when I needed to be pushed, backing off when I needed to be left alone, and listening to me when you could have been doing something more important. Dr. Hillman, thank you for the opportunity to work at the Voice Center, for all of your advice on my research, and also my professional growth and career. Your guidance was hugely helpful. Thanks to my committee members Dr. Roy and Dr. Howe for graciously agreeing to be a part of this. I hope you know how much I appreciate your time and expertise. The assistance of Drs. Joseph Perkell, Jim Kobler, Steve Massaquoi, and Neville Hogan was also essential to this endeavor, and I am very grateful for it.

Thanks to all of the Voice Center staff for the constant support, including Dr. Zeitels, Meredith, Marie, Tara, Harold, Kathryn, Selena, Ashley, and Matt. In particular, Dr. Burns and Maia really went above and beyond to find me subjects, Jennifer made everything easier, and Janet had the best advice. Thanks to the voice center grad students, especially Asako, Daryush, and Prakash (my guardian tech angel). Every day was easier because of you. Asako, consider this thesis mention as a replacement for the one you should have received. You are absolutely loved and appreciated by me. And much thanks to my two favorite professors, Yoshi and Gerardo. Yoshi, thank you for many morning meetings – I hope you give this thesis an A+. Gerardo, thanks for being there. I like you.

To my dear friends, thanks for many nights across the street: Melanie and Mike, Hannah, Maia and Jason, and KC. Mel, thanks for taking the time to listen to me blow off steam no matter how busy you are! KC, how could I get along without my career guide and my go-to (not back-up) date? Ann, Naomi, Adam, Ronit, and Bennett, thanks for all the fun times. The 2004 SHBT class, in order of their attendance at class outings, with (#) indicating their responsiveness during email discussions: wendy(6), nancy(5), erik(2), thomas(7), ted(4), tom(1), manny(3). We had great times, and I'm thankful for all of you. Tom especially, thanks for being 'my person' when I needed it.

My greatest thanks go to my family. My Mom and Dad have always given me completely unconditional love and support, and the expectation that I should do my best. Mom, you are inspirational – you are such a smart, giving woman. I can only aspire to become what you are. Dad, thanks for the permission to slow down sometimes. You have no idea how much it helps to have someone telling you that you are already great, and that it's ok to take a break. Brandon, thanks for always being there, being the brother I didn't have, forgetting my birthday, and always ringing the doorbell. There's no one I'd rather eat cheese with. To all of my extended family, thanks for all of the love and support and card games. It means more than you know.

Contents

Thesis organization

This thesis contains five studies with a common underlying goal of improving the clinical assessment of vocal hyperfunction. The thesis is organized as five self-contained manuscripts (Chapters 2 – 6), preceded by a common foreword (Chapter 1). Chapter 1 provides the motivation for the current studies and acts as a general primer for understanding the work performed for naïve readers. More familiar readers may prefer to skip this section and begin with Chapter 2.

Each of the five manuscripts (Chapters 2 – 6) is written in preparation for publication in a range of different journals. Given the varied audiences of the associated journals, the chapters vary in the amount and type of background information provided. In some manuscript chapters there is significant overlap with the information provided in the common background, whereas in others there is not.

Specifically:

Chapter 2: C.E. Stepp, J.T. Heaton, M.E. Jetté, J.A. Burns, R.E. Hillman. "Use of injection laryngoplasty as a platform for the study of objective measures of vocal hyperfunction," to be submitted to the Journal of Speech Language and Hearing Research.

Chapter 3: C.E. Stepp, R.E. Hillman, J.T. Heaton. "A virtual trajectory model predicts differences in vocal fold kinematics in individuals with vocal hyperfunction," submitted to the Journal of the Acoustical Society of America (JASA).

Chapter 4: C.E., Stepp, J.T. Heaton, m.N. Braden, M.E. Jetté, T.K. Stadelman-Cohen, R.E. Hillman. "Comparison of neck palpation rating systems with objective measures," submitted to the Journal of Voice.

Chapter 5: C.E. Stepp, J.T. Heaton, T.K. Stadelman-Cohen, M.N. Braden, M.E. Jetté, R.E. Hillman. "Acoustic, aerodynamic, and electromyographic characteristics of phonatory behaviors in individuals with vocal fold nodules," to be submitted to the Journal of Speech Language and Hearing Research.

Chapter 6: C.E. Stepp, R.E. Hillman, J.T. Heaton. "Use of Neck Strap Muscle Intermuscular Coherence as an Indicator of Vocal Hyperfunction," submitted to the IEEE Transactions in Neural Engineering and Rehabilitation Engineering.

Chapter 1: Background

Voice Production

The classic theory of speech production is the source-filter model (Fant, 1960; further described by Stevens, 2000). This model separates functionally and physically the source, or vocal stimulus, from the filter, which consists of the articulatory apparatus. The source, then, of most human speech is created through use of the larynx.

Relevant Anatomy and Physiology

The larynx is a system of suspended cartilages, lined with folds, acting as a valve between the airway and the pharynx (see Figure 1-1). Nearby muscles can alter the position and shape of these folds. The area of the larynx at the level of the vocal folds is referred to as the glottis. The muscles of the larynx can be divided into two main groups: intrinsic and extrinsic muscles. While intrinsic laryngeal muscles connect different parts of the larynx to each other, the extrinsic laryngeal muscles (also referred to as strap muscles) connect the larynx to outside structures (Fink & Demarest, 1977).

The larynx transforms airflow from the lungs into a series of air puffs which constitute the voice, and thus the source for the articulatory filters of the upper airway. Airflow from the lungs drives through the vocal folds, causing them to open; the folds are then pulled back together due to Bernoulli forces and the elastic properties of the folds, cutting off the airflow and creating an air puff. These forces may be manipulated to change the characteristics of phonation. Forces created by airflow from the lungs may be manipulated by using higher driving subglottal pressures, whereas the vocal fold tension and length characteristics are primarily controlled by the intrinsic laryngeal muscles. In addition, the intrinsic laryngeal muscles are also the primary actors in vocal fold adduction, thereby positioning the vocal folds to allow generation of voice. The extrinsic laryngeal muscles also have a role in vocal control, including a part in directing abduction, adduction, and pitch changes (Erickson, Baer, & Harris, 1981; Hirano, Koike, & von Leden, 1967; Hong, Ye, Kim, Kevorkian, & Berke, 1997; Konrad, et al., 1984; Roubeau, Chevrie-Muller, & Lacau Saint Guily, 1997), although their specific mechanical action in vocal control is not well understood.

The human vocal fold (informally referred to as the vocal cord) has a complex structure that allows it to perform not only its respiratory function, but also that as the primary phonatory instrument. The vocal fold has a number of layers which become less pliable as you move deep to the surface of the fold (Carole T. Ferrand, 2001). The topmost layer of the vocal fold is a thin epithelium, under which lies a basement membrane (Carole T. Ferrand, 2001; Hirano, 1988). Underneath these outermost layers is the lamina propria. The lamina propria can be divided into three layers: the superficial layer (Reinke's space), the intermediate layer, and the deep layer. Reinke's space is quite pliable and plainly moves during vocal fold vibration. The intermediate layer is somewhat less

pliable, although it is made up of elastic fibers. The deep layer of the lamina propria is much less pliable and consists of collagenous fibers. Together, the deep and intermediate layers may be called the vocal ligament. Beneath the lamina propria lies the thyroarytenoid (vocalis) muscle, which constitutes the main body of the vocal fold (Hirano, 1988).

There are five intrinsic muscles of the larynx, all of which have both their origin and insertion within the confines of the laryngeal cartilages (C.T. Ferrand, 2001), and are usually defined as abductors, adductors, and tensors (Choi, Berke, Ye, & Kreiman, 1993). Figure 1-2 shows a superior view of the glottis with labeled intrinsic muscles. Tensors are thought to regulate the length and tension of the vocal folds; adductors contract to close the glottis, while the sole abductor opens the glottis (Choi, et al., 1993). The three adductors are the lateral cricoarytenoid (LCA), the thyroarytenoid (TA) and the interarytenoid (IA). The LCA is a paired muscle and adducts the vocal folds by pulling the vocal processes toward each other in an inward and downward movement via its contraction. The thyroarytenoid (TA) forms the muscle mass of the vocal folds; it is a paired muscle and its medial-most region is sometimes referred to as the vocalis. The TA is subject to abduction, adduction, and stretching via the contraction of the surrounding intrinsic muscles; however, it can also exert its own internal tension. This tension is thought to stiffen the TA, contribute to adduction of the vocal folds, and increase the rate of vibration of the vocal folds (C.T. Ferrand, 2001). The IA is unpaired and consists of two bundles of muscle fiber, the transverse and oblique portions. Contraction of the IA moves the arytenoid cartilages medially, closing the posterior portion of the glottis. The posterior cricoarytenoid (PCA) is the only abductor of the vocal folds (C.T. Ferrand, 2001; Tucker, 1987); it is large and fan-shaped. Its contraction causes the vocal processes to be pulled away from each other (C.T. Ferrand, 2001). The most important function of the PCA is to open the glottis during respiration; however, the function of the PCA is not limited to respiration, nor does it contribute with merely passive control of voice as has been previously supposed. The PCA is known to contribute to fine control of subglottic pressure, frequency, and intensity (Choi, et al., 1993) and controls active devoicing. The cricothyroid (CT), considered to be the main actor in pitch change, elongates and stretches the vocal folds with its two sets of muscle fibers, the *pars recta* and the *pars oblique*. Contraction of the CT is known to elongate the vocal folds, decrease their mass per unit area, and increase their tension, thus resulting in a higher frequency of vibration (C.T. Ferrand, 2001). The cricothyroid joint movement is caused by contraction of the two bellies of the CT muscles. The *pars recta* displaces the joint vertically, while the *pars oblique* displaces the joint horizontally (Hong, et al., 1998). The motor innervation of the intrinsic muscles of the larynx is exclusively supplied by the vagus nerve; further, with the exception of the cricothyroid, all of the intrinsic muscles are supplied by the same branch of the vagus nerve, the recurrent laryngeal nerve. The cricothyroid muscle, however, receives innervation via the external division of the superior laryngeal nerve of the vagus (Fink & Demarest, 1978).

The extrinsic muscles of the larynx are those which have only one point of attachment to the larynx; they are also referred to as the strap muscles (see Figure 1-3). These muscles may be subdivided into two classes: infrahyoid and suprahyoid muscles. The infrahyoid

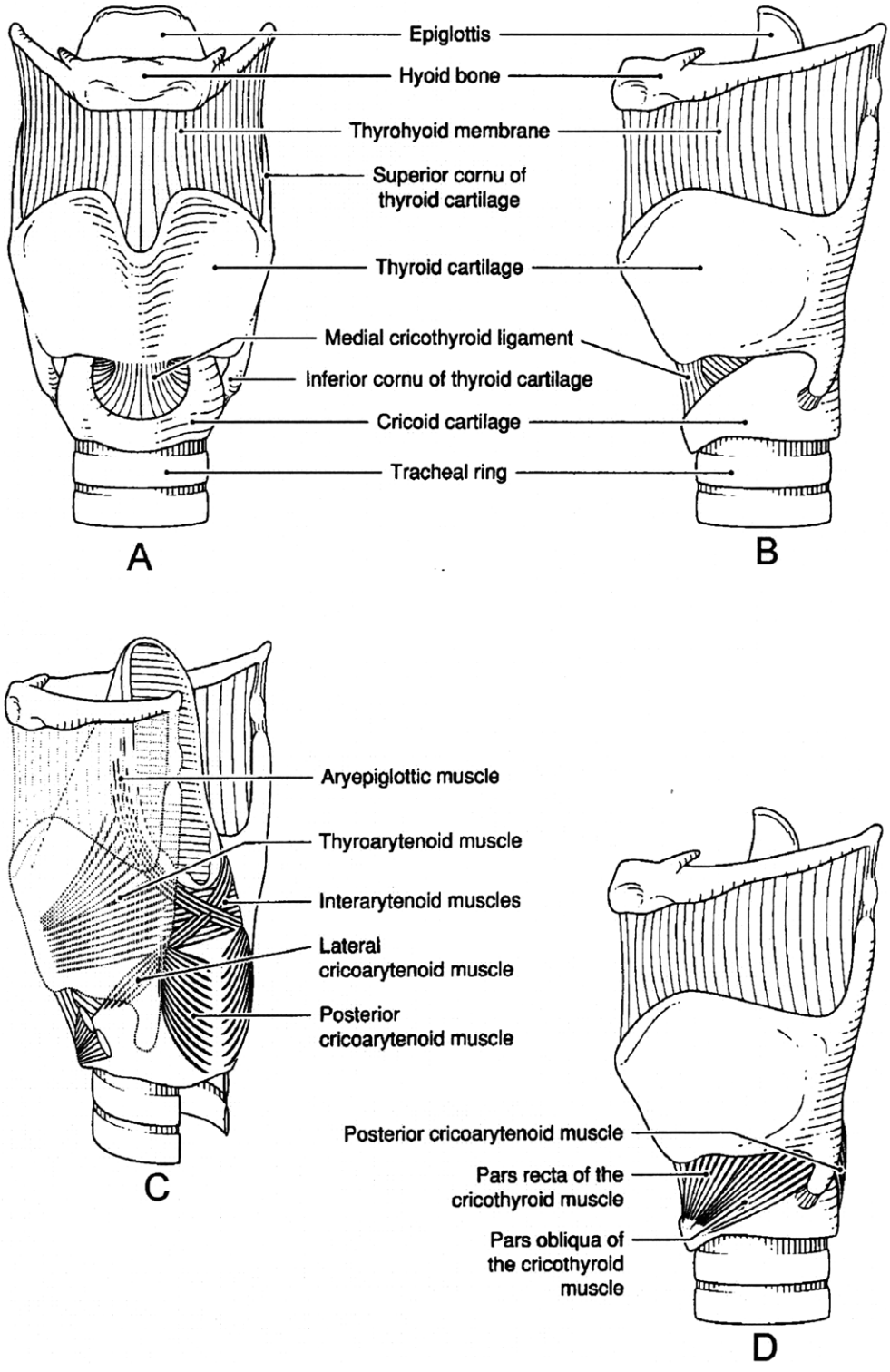


Figure 1-1. Anatomy of the larynx. Anterior and lateral views of the laryngeal cartilages and the hyoid bone are shown in panels A and B, respectively. Posterior-lateral and lateral views of the intrinsic laryngeal muscles are shown in panels C and D, respectively. (Adapted from Titze, 1994b)

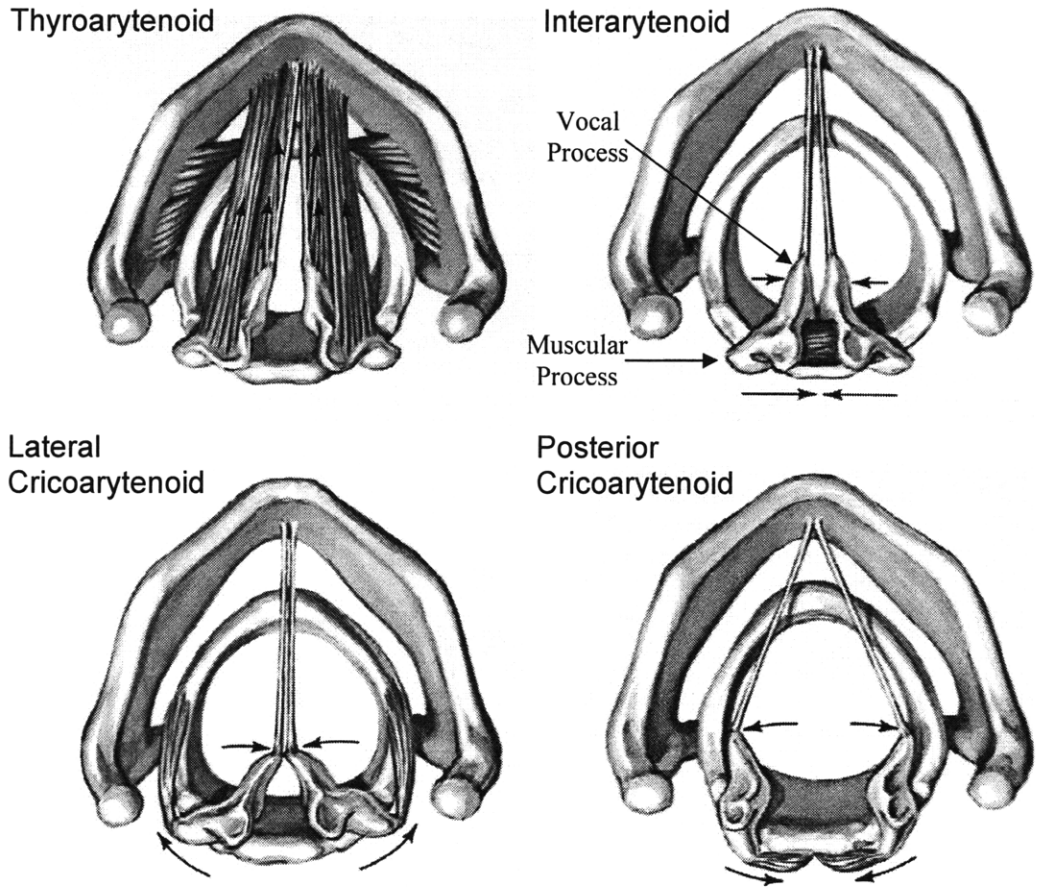


Figure 1-2. Superior view of the glottis. Mode of operation of the four intrinsic laryngeal muscles associated with abduction and adduction are indicated with arrows. The TA, IA, and LCA contribute to adduction, whereas the PCA is responsible for abduction. Blue arrows indicate the vocal and muscular processes of the arytenoid cartilage.

muscles have their point of attachment at structures inferior to the hyoid bone and contract to lower the larynx, while the suprahyoid muscles have their point of attachment to structures superior to the hyoid bone and contract to elevate the larynx. The suprahyoids include the anterior and posterior digastric, stylohyoid, mylohyoid, geniohyoid, and hyoglossus, while the infrahyoids include the sternohyoid, omohyoid, sternothyroid, and thryohyoid (C.T. Ferrand, 2001). The motor innervation of the extrinsic muscles is more diverse and includes the glossopharyngeal nerve, the pharyngeal plexus of the vagus, and the cervical plexus (Fink & Demarest, 1978).

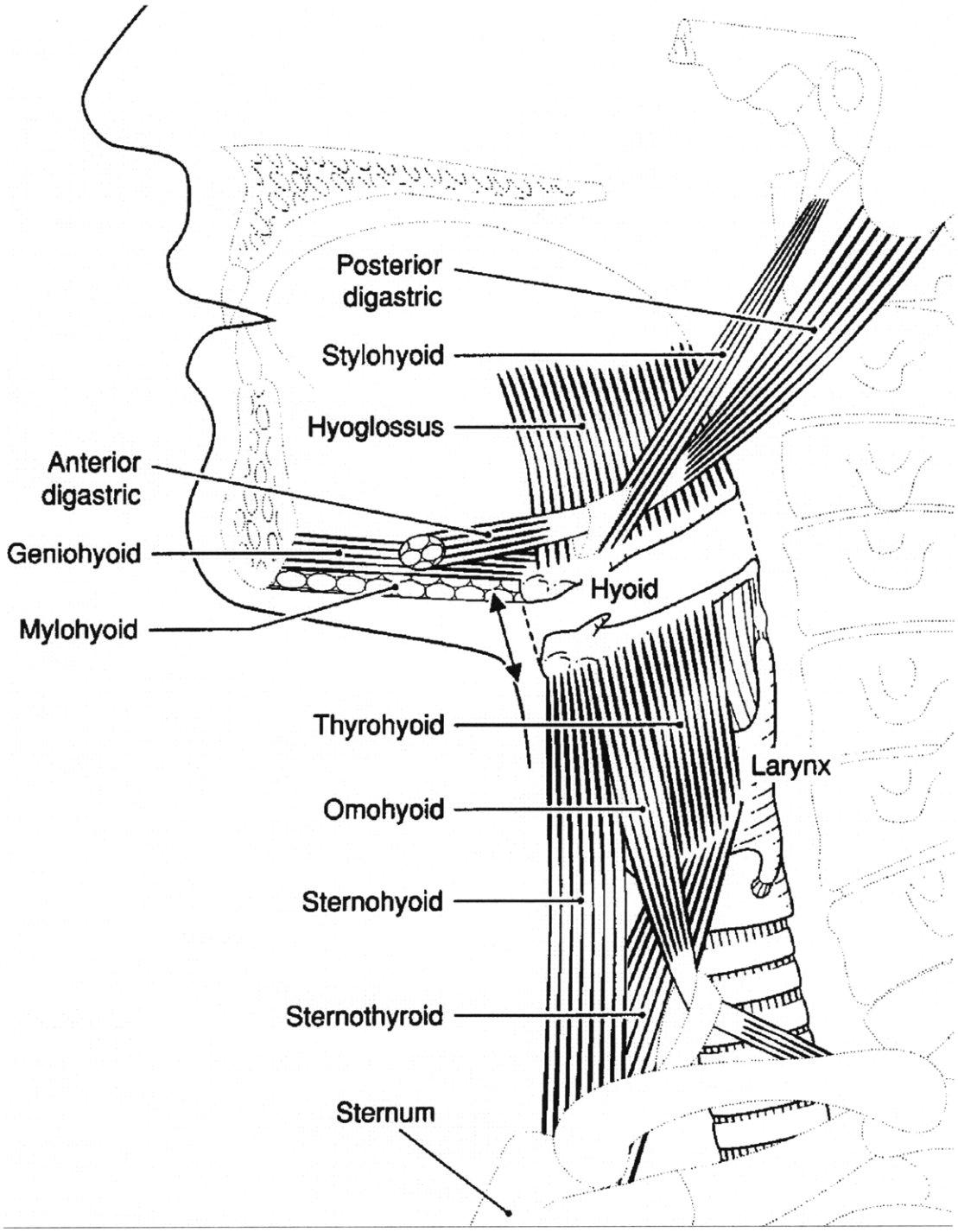


Figure 1-3. The extrinsic laryngeal muscles. (Adapted from Titze, 1994b)

ROLE OF EXTRINSIC LARYNGEAL MUSCULATURE

By the very nature of their anatomical connections, it is apparent that the extrinsic laryngeal muscles are positioned to have some effect on phonation control. For example, in cases of thyroidectomy, the strap muscles are routinely cut and/or damaged, with patients generally reporting transient and in some cases more permanent changes in voice; (Debruyne, Ostyn, Delaere, & Wellens, 1997; Debruyne, Ostyn, Delaere, Wellens, & Decoster, 1997; Hong & Kim, 1997; McIvor, Flint, Gillibrand, & Morton, 2000; Reynere, 1974; Stojadinovic, et al., 2002).

The extrinsic laryngeal muscles have direct control over the vertical position of the larynx, which in turn affects the length and tension of the vocal folds due to rotation of the cricoid cartilage caused by cervical lordosis (Honda, Hirai, Masaki, & Shimada, 1999). Direct forces likely to affect phonation include tracheal pull, and tension from the following muscles: sternothyroid (ST), thyrohyoid (TH), sternohyoid (SH), cricopharyngeal (CP), thyropharyngeal (TP). More indirect influences on voice production include the following suprahyoid and infrahyoid muscles: digastric, mylohyoid, geniohyoid (GH), hyoglossus, genioglossus, and omohyoid. The large number of factors affecting the biomechanics of the larynx creates a redundancy in this system and may play a role in the apparent contradiction across studies of the contributions of various muscles to phonation tasks. Specifically, a 1996 review by Vilkman and colleagues of 15 EMG studies of the contributions of extrinsic laryngeal muscles to phonation in humans found lack of consistency within studies as well as conflicting results between them (Vilkman, Sonninen, Hurme, & Korkko, 1996). Muscles examined in multiple studies were the ST, TH, CP, and SH, none of which yielded conclusive results for pitch lowering or raising. Table 1-1 summarizes the results of this review. Although many models of pitch control via extrinsic factors exist (Kenyon, 1927; Sonninen, 1968; Vilkman, et al., 1996), the conclusion of this review was that current data are too disparate to corroborate any comprehensive model extrinsic laryngeal pitch control.

More recently, Roubeau and colleagues measured the normalized EMG of the TH, ST, and SH in one female and one male with healthy normal voice while performing ascending and descending glissandos (Roubeau, et al., 1997). Their results are reproduced in Figure 1-4. During ascending glissando from 120 Hz to 600 Hz, the SH was found to be generally active throughout, whereas the ST and TH were modestly active (10% of the maximum recorded) between 120 Hz – 150 Hz and then 500 Hz – 600 Hz. During descending glissando, however, the female participant showed most pronounced differences in ST and TH activity across frequency. Here, the ST and TH were again modestly active in the 500 Hz – 600 Hz range, but in the range from 120 Hz - 200 Hz showed more activity (25% of the maximum recorded). The SH again showed moderate activity over all frequencies except at the extremes (0% of the maximum recorded at 600 Hz, 15% of the maximum recorded in the 120 Hz – 200 Hz range). For the ascending glissando, the male participant showed far more variation than the female participant. The TH, ST, and SH all showed high activity during both low and high frequencies (values of 20 % - 50% of the maximum recorded). For the descending glissando, the male participant showed tonic activity in the TH (30% of the maximum

recorded), with maximum activity at the lower frequencies (50% of the maximum recorded). The ST and SH showed fairly stable levels (around 10% of the maximum recorded) at most frequencies, with some increase in activity at the lower frequencies (15% - 25% of the maximum recorded). The differing results between ascending and descending pitch change noted by Roubeau imply that activity of the extrinsic laryngeal muscles is more closely tied to dynamic aspects of pitch than to specific pitch production.

VOICE DISORDERS

The term "voice disorder" has been defined as "a problem in producing voice that is primarily caused by a disturbance or loss of normal laryngeal function" (Hillman, Gress, Hargrave, Walsh, & Bunting, 1990). Studies have shown that a voice disorder does not only affect the economic well-being of the patient, but that it also may cause a disordered self-image that negatively affects the patient socially (Ramig & Verdolini, 1998).

One school of thought identifies four general components to voice disorder: 1) voice-related muscular skill including posture, 2) behavior pertaining to lifestyle and personality, 3) gastro-esophageal reflux disease, and 4) psychological factors (M. Morrison, 1997a). Further, it is thought that pathological process may overlie the disorder; these processes being broadly divided into two groups: neurological disease or organic changes to the vocal folds (M. Morrison, 1997a). The poor muscle usage (or low level of vocal skill) is commonly referred to as muscle and/or voice misuse. This misuse is correlated in a nontrivial way to fundamental frequency, sound pressure level, and particular vocal behaviors of the patient with respect to their "normal" voice (Sander & Ripich, 1983).

CLINICAL ASSESSMENT OF VOCAL FUNCTION

Current clinical assessment of vocal function is a multidimensional process consisting of perceptual and objective measures. Current practice includes patient interview for voice and psychosocial history and status, perceptual assessment of voice quality by the clinician and patient, observation of positioning and posture of the upper body, elicitation of reflexes, direct (oral) endoscopy, flexible (nasal) endoscopy, acoustic analysis of the speech signal, electroglottography, aerodynamic assessment, evaluation of respiratory kinematics, and in some cases laryngeal electromyography (Andrews, 1999; Colton, Casper, & Leonard, 2006).

These general categories of voice-related information can provide overlapping and complementary information regarding the vocal health of the patient. The patient interview includes discussion of the history, effect, onset, and duration of his or her voice issues. Further, the patient interview can supply information about the voice use, general health, and social/psychological status of the patient (Colton, et al., 2006). The state-of-the-art in clinician perceptual assessment of voice quality is the Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V), a visual-analog scale ratings system of six perceptual attributes of voice: overall severity, roughness, breathiness, strain, pitch, and loudness (Colton, et al., 2006). Patient perception of their voice and the impact of any

Study	Material	Phonation	Pitch range	Muscle	Pitch lowering	Pitch raising
Faaborg-Andersen and Sonninen (1960)	7 males, 7 females	singing, sustained	extremes	ST MH TH	+ - -	(+) (+) +
Zenker and Zenker (1960)	human	singing	extremes	TP CP	+ +	+ +
Hirano et al. (1967)	3 males	singing	extremes	SH	+	+
Minnigerode (1967)	25 humans	phonation		CP	-	+
Ohala and Hirose (1970)	3 males	sustained	extremes	SH	+	+
Vennard et al. (1971)	1 female 2 males	singing	extremes	TH SH DG LP PP	+ + - (+) -	+ + - (+) +
Shipp (1975)	6 males	singing	extremes	ST TH	+ -	+ +
Baer et al. (1976)	human, several	singing	extremes, arpeggio	TH SH CP LP MH GG SC	+ + + o o o o	+ + + + + + +
Estill et al. (1983)	1 male	singing	two octaves	LP PP GH MC GGP TH ST	o o o o o o o	+ + + + + + +
Niimi et al. (1991)	2 males	singing	vibrato	ST	-	+
Simada and Hirose (1970)	human	speech, stress	speech	SH	o	(+)
Collier (1974)	1 male	speech, intonation	speech	SH ST TH	(+) (+) -	(+) (+) -
Erickson (1977)	1 male	speech, stress and intonation	speech	GH SH ST TH	o o o o	+ o o o
Atkinson and Erickson (1976)	1 male	speeech, intonation	speech	SH	+	-
Atkinson (1978)	1 male	speech, intonation	speech	SH ST	+ (+)	- (+)

Abbreviations: IPC, inferior pharyngeal constrictor; DG, digastrc muscle; GGP, genioglossus, posterior part; LP, levator palatini; MC, middle constriction; GH, geniohyoid muscle; MH, mylohyoid muscle; SC, superior constrictor; CP, crycopharyngeal muscle; TP, thyropharyngeal muscle; SH, sternohyoid muscle; TH, thyrohyoid muscle; GG, genioglossal muscle; PP, palatopharyngeal muscle. +, effect; -, no effect; o, not studied; (+), inconsistent.

Table 1-1. Reproduction of the summary of Vilkman et al. (1996) of 15 EMG studies of the contributions of extrinsic laryngeal muscles to phonation in humans.

disorder is addressed using perceptual "quality of life" scales such as the Voice Handicap Index (VHI) and the Voice-Related Quality of Life (V-RQOL) (Colton, et al., 2006). Observation of the head, neck, and torso can be vital in a complete evaluation of vocal health. In particular, clinicians look for anterior/posterior weight bearing, head tilt, deviated larynx (abnormally rotated), asymmetrical sternocleidomastoid muscles, an abnormally held larynx (high or low), and high muscle tone / reduced flexibility surrounding the larynx (see Lieberman, 1998, for a comprehensive tutorial). Elicitation of oral, laryngeal, and gag reflexes can be used to rule out or suggest neurological impairment (Andrews, 1999; Colton, et al., 2006).

For the most part, current clinical practice utilizes two main types of endoscopy: rigid (oral) and flexible (nasal). Rigid endoscopy is performed by passing a rigid endoscope over the top of the tongue and into the pharyngeal cavity. This procedure allows for larger, brighter images of the vocal folds, but requires the clinician to pull the patient's tongue to get an adequate view, manipulating his or her natural vocal behaviors. In flexible endoscopy, the endoscope is passed through the nose and down into the pharynx in order to observe the vocal folds and supraglottic space during more natural speech and singing tasks. Figure 1-5 shows example stills of the same larynx taken from both exam types. Stroboscopy can be utilized with both methods of endoscopy, and involves flashing light at a rate slightly different than the vibrating rate of the vocal folds. This technique allows the vocal folds to be illuminated at different phases of their vibratory cycle, creating the illusion of a slowing of the vibratory motion of the folds and enabling the clinician to view representative repeated cycles of vocal fold motion.

Acoustic signs typically indicative of vocal fold health are fundamental frequency (mean, variability, range, and perturbation), amplitude (average, variability, dynamic range, and perturbation), signal-to-noise ratio, and maximum phonation time. Electroglottography (EGG) uses two electrodes placed on the surface of the each side of the neck at the larynx to measure the current flow through the neck tissues between the two, giving an indirect measure of the closure of the vocal folds. Aerodynamic assessment can include measures of airflow, subglottal pressure, and phonation threshold pressure, all of which may provide information about phonation production. Respiratory kinematics can highlight the patient's control over respiratory movements and are usually measured using an induction plethysmograph or magnetometer, devices that can non-invasively monitor the movements of the chest and abdomen during speech tasks. Laryngeal electromyography (EMG) is an invasive procedure during which electrodes are inserted through the neck surface or brought through the mouth and inserted into the intrinsic laryngeal muscles to record their electrical activity. Due to the invasive nature of this procedure and the neurological expertise required to interpret the test findings, it is not common clinical practice. However, it can be used to support diagnosis of paralysis or to guide injections of botulinum toxin for treatment of spasmodic dysphonia. Of the measures currently available for clinical assessment of voice, objective measures include EMG, aerodynamic assessment, EGG, and acoustic assessment. Currently, these objective measures are not used as primary diagnostic procedures, but as supplemental tools to enhance diagnosis quality (Hillman, Montgomery, & Zeitels, 1997).

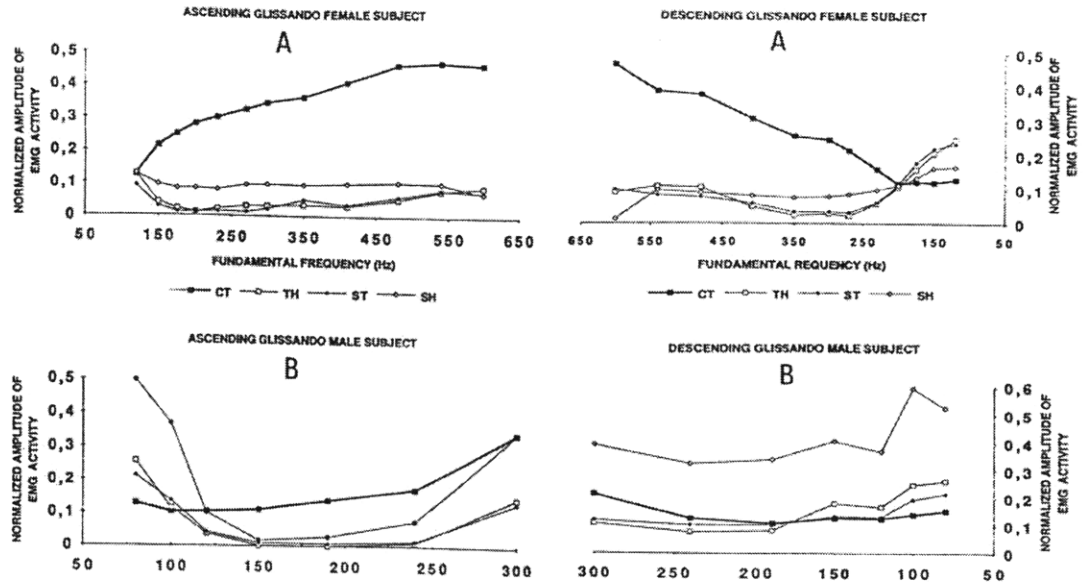


Figure 1-4. Reproduction of the results of Roubeau et al. (1997). Data for the ascending glissando are shown in the left panels, descending in the right panels. Data from the female and male participants are shown in the upper panels and lower panels, respectively.

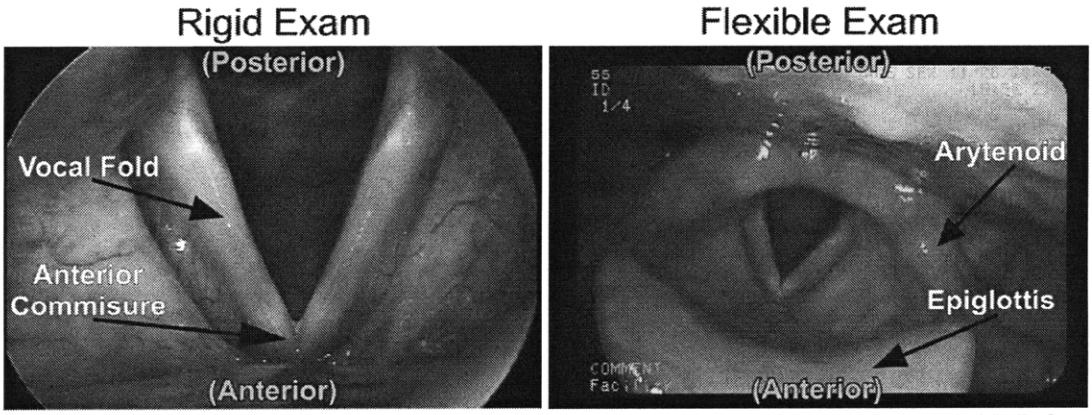


Figure 1-5. Example images of the same set of vocal folds as viewed by rigid (left panel) and flexible (right panel) endoscopy.

Vocal Hyperfunction

Functional dysphonia is an "umbrella diagnosis" for impairment of voice production in the absence of structural change or neurogenic disease of the larynx (Altman, Atkinson, & Lazarus, 2005). While this type of functional disorder can have fairly homogenous clinical symptoms, the etiology is more heterogeneous (Kinzl, Biebl, & Rauchegger, 1988). There is thought to be some psychogenic nature to this etiology, however, which is supported by the finding that some 75% of functional voice patients have other psychosomatic functional disturbances in their histories, such as anorexia nervosa, bulimia nervosa, cardiac neurosis, migraine, bronchial asthma, and diffuse abdominal complaints (Kinzl, et al., 1988). While functional voice disorders are by definition primary muscle tension dysphonias, many patients who display characteristics of functional voice disorders have underlying organic conditions. Here, the functional voice aberrations are secondary and may result from improper compensatory muscular activity (Koufman & Blalock, 1991a). Thus, most voice disorders can be viewed as completely or partly functional (Koufman & Blalock, 1991a).

Vocal hyperfunction is a common functional voice disorder referring to "conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or 'imbalanced' muscular forces" (Hillman, et al., 1989), characterized by excessive laryngeal and paralaryngeal tension (Aronson, 1980; Dworkin, Meleca, & Abkarian, 2000; Koufman & Blalock, 1991b; M. D. Morrison, et al., 1983; N. Roy, et al., 1996). Individuals with vocal hyperfunction and no other known cause of voice disorder (e.g., some type of glottal insufficiency) are often diagnosed with muscle tension dysphonia (MTD; also known as hyperfunctional dysphonia, hyperkinetic dysphonia, muscular tension dysphonia, vocal hyperfunction, muscular tension dysphonia, vocal fatigue, laryngeal tension-fatigue syndrome, and functional hypertensive dysphonia (Hsiao, Liu, Hsu, Lee, & Lin, 2001)). This is a type of functional dysphonia making up some 50% of the cases seen (Koufman & Blalock, 1982). Other estimates of the prevalence of vocal hyperfunction indicate that the condition may account for 10 – 40 % of cases referred to multidisciplinary voice clinics (N. Roy, 2003). Hyperfunction of the laryngeal and paralaryngeal muscles could be due to psychological and/or personality factors, misuse of muscles, learned adaptation based on short-term illness, or compensation for underlying disease (Aronson, 1980; Hsiung & Hsiao, 2004; M. D. Morrison, Nichol, & Rammage, 1986; M. D. Morrison & Rammage, 1993; M. D. Morrison, et al., 1983; Rammage, Nichol, & Morrison, 1987).

Symptoms / Current State of Assessment

The manifestation of muscle tension dysphonia / vocal hyperfunction has been typified as the incorporation of the following symptoms: obvious muscular tension in paralaryngeal musculature, high larynx position, vocal fry, phase asymmetry, low pitch, diplophonia,

rough voice quality, exaggerated posterior glottic chink (gap), lateral compression in the membranous vocal folds, breathiness, glottic and supraglottic compression, adduction spasms, and strained voice quality (M. Morrison, 1997b; M. Morrison, Rammage, & Emami, 1999; M. D. Morrison, et al., 1986; M. D. Morrison, et al., 1983). Also, patients are often pitch- and loudness-locked with reduced dynamic range, reporting with aches and tightness of the neck, larynx, and shoulder regions, as well as episodic neck swelling and ear "fullness"(Aronson, 1980; Nelson Roy & Bless, 1998).

Videostroboscopic evaluation of MTD patients may also show characteristic signs including an uneven mucosal layer (Hsiao, et al., 2001; Hsiao, Liu, & Lin, 2002), abnormal glottal closure, phase or amplitude asymmetry, and an irregular mucosal wave (Hsiao, et al., 2001). A further symptom visible via flexible endoscopy includes supraglottic compression. Supraglottic compression describes obstruction of part or all of the view of the vocal folds during indirect laryngoscopy and videoendoscopy (Stager, Bielamowicz, Regnell, Gupta, & Barkmeier, 2000). There are two primary components: anterior-posterior (AP) compression, in which the arytenoids cartilages are drawn toward the petiole of the epiglottis, and medial or false vocal fold (FVF) compression, which is caused by adduction of the false vocal folds (Stager, et al., 2000). Studies have cited this supraglottic activity as a symptom characteristic of MTD (M. Morrison, et al., 1999), but without comparisons to its prevalence in populations with healthy normal voice. Stager and colleagues compared the incidence of AP and FVF compression in hyperfunctioning, vocal nodule, and healthy normal voice participants, finding that the incidence in individuals with healthy normal voice was less than that in the voice patients, but was not absent (Stager, et al., 2000). However, the control group in the study consisted of patients with allergies and/or reflux, which may have been a confounding factor. Specifically, reflux patients have been described as likely to display supraglottic compression (Koufman & Blalock, 1991b). A retrospective study by Morrison and colleagues recorded the laryngoscopic examination of patients diagnosed with vocal hyperfunction (M. D. Morrison, et al., 1986). Of diagnoses of vocal hyperfunction: 11% presented with no mucosal change, 90% presented with an open posterior chink, 49% presented with visible suprahyoid tension. While 90% of MTD diagnoses present with a glottal posterior chink (M. D. Morrison, et al., 1986), this symptom is also prevalent in young women with normal voice (Linville, 1992). In a videostroboscopic study of young adult women with normal voice, a posterior-chink glottal configuration was noted 42% of the time, compared to 13% in a population of elderly adult women with healthy normal voice (Linville, 1992). Rammage and colleagues found a similar result: "virtually all" of their 50 participants identified as having bilateral vocal nodules presented with a posterior glottal chink during phonation; further, a majority of the 20 participants with healthy normal voice (women, aged between 17 and 36) also presented with a posterior glottal chink (Rammage, Peppard, & Bless, 1992).

Compared to individuals with healthy normal voice, muscle tension dysphonia patients have also been demonstrated to have a higher prevalence of abnormal glottic closure during inspiration (similar to PVFM, paradoxical vocal fold movement) (Vertigan, et al., 2006). PVFM is also referred to as episodic paroxysmal laryngospasm (EPL) and is characterized by involuntary paradoxical adduction of the vocal folds and ventricular folds during inspiration (Andrianopoulos, Gallivan, & Gallivan, 2000). It is frequently mistaken for asthma, acute respiratory distress, or other laryngeal problems, including

MTD (Andrianopoulos, et al., 2000). Morrison and colleagues have postulated that PVFM and MTD, as well as chronic cough, throat clearing, and globus pharyngeus are all forms of hyperkinetic laryngeal dysfunction, which they have termed irritable larynx syndrome (ILS) (M. Morrison, et al., 1999). Highlighting the interplay between vocal hyperfunction and other laryngeal irritation, a series of three case studies from Japan showed that there was subjective improvement in the voice and glottal closure of MTD patients following treatment via proton pump inhibitors, indicating that reflux may have been a major causative factor in the disorder (Mesuda, et al., 2007).

Commonly associated symptoms of vocal hyperfunction are not limited to the larynx. Many muscles in the neck that attach to the larynx and/or hyoid bone have voice and speech-related contractions due to their role in controlling the vertical position of the larynx in the neck and, to some degree, the position of the tongue. When individuals demonstrate an inappropriate degree of intrinsic laryngeal muscle contraction (hyperfunction), it is thought that they often simultaneously contract the extrinsic laryngeal muscles and other superficial neck muscles in a similar hyperfunctional manner (Aronson, 1980). A study by Altman and colleagues looked at 150 patients who had been diagnosed with muscle tension dysphonia; based on a speech pathology evaluation of these patients, 70% were found to have "obvious cervical neck tension visible" (Altman, et al., 2005). Clinically, excessive tension has been noted via palpation over the major horns of the hyoid bone, over the superior cornu of the thyroid cartilage, along the anterior border of the sternocleidomastoid muscle, and throughout the suprahyoid musculature (N. Roy, et al., 1996). Strap muscle tension can be noted in the MTD patient through both visual and tactile inputs. Particularly, observation of the inferior bellies of the omohyoid muscle crossing the supraclavicular fossae easily shows them to be tense and prominent during speech (M. Morrison, 1997b). Further information about the extent of muscle tension found in the patient can be gained by palpation of the larynx at rest and during voicing (M. Morrison, 1997b).

Further known symptoms of MTD include subjective observations of perceived breathiness (Aronson, 1980; M. Morrison, et al., 1999; M. D. Morrison, et al., 1986), which are supported in part by findings of increased flow rates in affected patients (Eustace, Stemple, & Lee, 1996). Another aspect of aerodynamic assessment cited by Hillman and colleagues as specific evidence of vocal hyperfunction is the ratio of subglottal pressure and sound pressure level with respect to age-and gender-specific normative data. Specifically, the use of excessive subglottal "driving pressure" to produce sound pressure levels is a feature seen in many patients with vocal hyperfunction and can be measured objectively (Hillman, et al., 1997). Morrison and colleagues found that of individuals with vocal hyperfunction: 81% presented with breathiness, 50% presented with glottal fry, 65% with hard glottal attack (M. D. Morrison, et al., 1986).

Organic Developments and Interactions

For many years, organic developments on the vocal fold surface have been assumed to be related to hyperfunctional behavior or phonotrauma (Hillman, et al., 1990). For instance, in 1962, Godrey Arnold wrote that "vocal nodules and polyps represent a local tissue reaction to the mental strain imposed by inappropriate emotional adjustment to the

demands made by society" (Arnold, 1962). However, much is still unknown about the underlying mechanisms of vocal hyperfunction and its role in developing organic disorders (Hillman, et al., 1990).

It has been argued that a patient's vocal hyperfunction begins with a structurally normal larynx, in which the imbalanced muscular activity causes an open posterior glottic chink between the arytenoids cartilages upon phonation (M. D. Morrison, et al., 1986). Prolonged voicing in this arrangement leads to excessive mechanical stress and trauma that may eventually cause mucosal changes to develop, including nodules, diffuse erythema and edema, and polyps (Hillman, et al., 1989; Johns, 2003; M. D. Morrison, et al., 1986). While the vocal ligament can protect softer tissues of the lamina propria from the high tensile stress felt at high pitches, impact stress as a result of vocal fold collision may cause damage in the form of vocal nodules, which occur at the point along the edge of the vocal fold that experiences the maximum impact stress (Titze, 1994a).

Clinically, a nodule is defined as a small protuberance located between the anterior and middle third of the vocal fold (Aronson, 1980; Marcotullio, Magliulo, Pietrunti, & Suriano, 2002). It is described as being gray/white or pearl in color and is usually bilateral (Aronson, 1980; Marcotullio, et al., 2002). Vocal fold nodules are benign lesions characterized as being bilateral, symmetrical, and involving minimal disruption of the mucosal wave on stroboscopy. The basement membrane zone is thickened, having increased fibronectin. In vocal fold polyps, there are focal depositions of gelatinous material comprised of amorphous, disorganized extracellular matrix. The lesions are exophytic (growing outward from the surface epithelium) and may be associated with an enlarged blood vessel or hemorrhage. They are not typically associated with a thickening of the basement membrane (Dikkers & Nikkels, 1995; Verdolini, Rosen, & Branski, 2006). Some of the confusion in determining differences between various laryngeal lesions may arise from differences in diagnoses. Dikkers and Schutte found a large discrepancy among a large group of clinicians with respect to the name used for various laryngeal lesions (Dikkers & Schutte, 1991). Based on their study, they offered basic clinical definitions. Figure 1-6 is a pictorial summary of the basic categories of benign fibrovascular lesions along with their defining characteristics as described by Dikkers and Schutte (1991). They termed a cyst as a unilateral lesion with a smooth surface that is usually found on the middle third of the vocal fold, and is immobile during phonation (Dikkers & Schutte, 1991). They define Reinke's edema as a condition with unilateral or bilateral white swelling of the vocal fold, in which the lesion is filled with fluid, sessile (adhering closely to the surface), and mobile during phonation (Dikkers & Schutte, 1991). A polyp, they define as a unilateral lesion on the anterior third of the vocal fold, often on the free edge, and either pedunculated (attached by a thin process of tissue rather than a large base) or sessile (Dikkers & Schutte, 1991). Further, vocal fold nodules are described as small lesions on both sides of the larynx, symmetrical on the border of the anterior and middle third of the vocal folds, which are usually immobile during phonation (Dikkers & Schutte, 1991). Also noted in the clinic is the diagnosis of "vocal fold thickening." This is widely viewed as a antecedent to the formation of vocal nodules, but this hypothesis has not been proven (Goldman, Hargrave, Hillman, Holmberg, & Gress, 1996).

The form in which mucosal changes occur seems to depend upon the patient's age, sex, particular voice use, and whether or not the patient smokes (M. D. Morrison, et al., 1986). Particularly, vocal nodules usually occur in young to mid-aged females, whereas more diffuse mucosal changes are seen mostly in men (M. D. Morrison, et al., 1986; M. D. Morrison, et al., 1983). Polyps are commonly found in adult females, of whom almost all smoke cigarettes (M. D. Morrison, et al., 1986; M. D. Morrison, et al., 1983). Vocal nodules also seem to be more common in young larynges; a study of children with muscle tension dysphonia found coexisting vocal nodules in 7 of the 8 participants (Lee & Son, 2005). This is not surprising, given the results of a recent study by Marcotullio and colleagues, who argue that there are no substantial histologic differences between nodules and polyps, but that pathologic manifestations clinically classified as nodules are merely "younger" lesions than those classified as polyps (Marcotullio, et al., 2002). This correlation between age and sex and the development of vocal nodules appears to be routed in laryngeal morphology (Pontes, Kyrillos, Behlau, De Biase, & Pontes, 2002). Specifically, vocal nodules are found in larynges with a morphology associated with young women, one that reduces the functional opening angle during abduction of the vocal folds (Pontes, et al., 2002). The idea that lesion formation is a function of morphology and not specifically behavior is supported by the work of Andrade and colleagues. They found that hard glottal attacks occur significantly more frequently in MTD patients, but that this possibly causal behavior was found in the same frequency in all subgroups of MTD patients, regardless of the existence of vocal fold lesions or the lack of such lesions (Andrade, et al., 2000). Titze postulates that, given that vocal nodules are the result of repeated collision, the risk of developing nodules is higher in high-pitched voices, such as children and young women (Titze, 1989). It should be mentioned that the work of Dikkers and Nikkels opposes the idea that there is a lack of histologic difference between nodules and polyps, with work and hypotheses supporting a lack of clinical continuum between possible laryngeal lesions (Dikkers & Nikkels, 1995, 1999). Also contrary to the work of Marcotullio and colleagues, their work classifies a polyp as a "younger" lesion than a nodule (Dikkers & Nikkels, 1999). Thibeault and colleagues have examined the transcription-level profiles of extracellular matrix proteins in vocal fold polyps and Reinke's edema, finding differing extracellular matrix regulation (Thibeault, et al., 2002). Compared to polyps, fibronectin (interstitial proteoglycan necessary for the development of fibrosis) transcription levels were found to be down-regulated in Reinke's edema whereas fibromodulin (interstitial proteoglycan whose absence leads to disorganized collagen fiber bundles) transcription levels were found to be higher. These results support the clinical findings of increased stiffness in polyps relative to Reinke's edema and a lack of continuum between the two lesions.

Some 92% of cases of vocal nodules are coincident with vocal hyperfunction (M. D. Morrison, et al., 1983). Further, of individuals with vocal hyperfunction, Morrison et al. (1986) found that 51% presented with vocal nodules, while Altman et al. (2005) found that 13% presented with vocal nodules, the development of vocal nodules appears to be highly correlated with the disorder. Nodules found in patients that do not present with vocal hyperfunction tend to be more firm and less likely to resolve with voice therapy alone, indicating a potential difference in etiology (M. D. Morrison, et al., 1983).

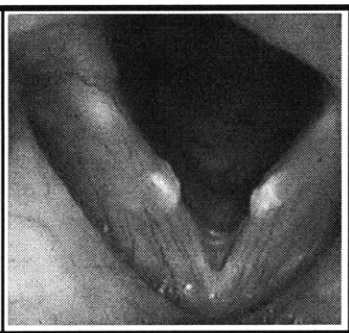
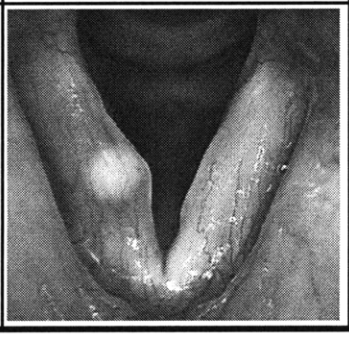
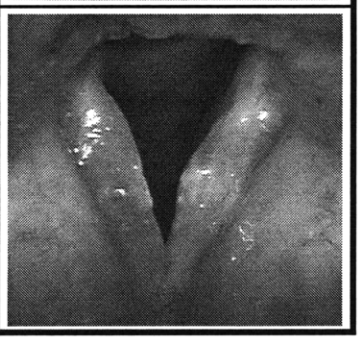
Nodules: small, bilateral, symmetrical, located on the border of the anterior and middle thirds of the vocal fold		Polyp: unilateral, often on the free edge, sessile or pedunculated, located on the anterior third of the vocal fold	
Cyst: unilateral, smooth surface, typically immobile during phonation, located on the middle third of the vocal fold		Reinke's Edema: unilateral or bilateral white swelling of the vocal fold, fluid, sessile, and mobile during phonation	

Figure 1-6. Pictorial summary of the basic categories of benign fibrovascular lesions along with their defining characteristics as described by Dikkers and Schutte (1991).

TREATMENT OPTIONS

Voice therapy has been shown to decrease nodules and improve voice quality (Carding, Horsley, & Docherty, 1999; Holmberg, Hillman, Hammarberg, Sodersten, & Doyle, 2001) and to be generally effective in treating non-organic voice disorders (Carding & Horsley, 1992). A recent study found that even polyps and cysts were effectively treated via voice therapy (Cohen & Garrett, 2007). However, it is generally agreed that time and energy must be devoted to "carryover" of behaviors learned in behaviorally based voice therapy, in order to carry over those vocal behaviors from the clinic to "real life" situations outside the therapy setting (Holmberg, et al., 2001).

Most voice therapies for MTD include the aim of reducing excess laryngeal musculoskeletal tension. Most of these therapies such as progressive relaxation, the accent method, yawn-sigh therapy, and resonant voice therapy do this indirectly (Nelson Roy & Bless, 1998). Another typical part of MTD voice therapy that addresses this abnormal tension directly is the practice of "manual laryngeal tension reduction" which includes manually lowering an abnormally elevated larynx, as well as performing circumlaryngeal massage to reduce muscle tension (N. Roy, et al., 1996; N. Roy & Leeper, 1993). This practice is meant to raise the patient's awareness of neck tension (Nelson Roy & Bless, 1998). This practice has been shown to produce meaningful perceptual and acoustic results in short-term scenarios (N. Roy, Bless, Heisey, & Ford, 1997; N. Roy & Leeper, 1993), with less impressive long-term results

(N. Roy, et al., 1997). These studies recommend that this practice may be an integral part of voice therapy for MTD, but is an "incomplete remedy" (N. Roy, et al., 1997). Specifically, patients have trouble relying on their own current feedback systems to learn long-term improved vocal habits (Nelson Roy & Bless, 1998). An extension of manual tension reduction techniques employed through voice therapy has been explored in part. Particularly, some success has been found in limited studies of multidisciplinary treatment of MTD; in this scenario, voice therapy is supplemented by physical therapy to directly invoke relaxation of head, neck, and back musculature (Jones, Murry, & Rosen, 1998). A Finnish version of circumlaryngeal massage known as "voice massage" was performed on individuals with healthy normal voice with no immediate significant post-treatment effects (Laukkanen, Leppanen, Tyrmi, & Vilkman, 2005); however, this study has not been reproduced in disordered speakers. Dworkin and colleagues offer a possible supplement to typical voice therapy techniques. Their study found that in several case studies of patients with therapy-resistant MTD, a topical lidocaine bath was able to break the hyperfunctional vocal fold behavior with good, and sustained effect (Dworkin, Meleca, Simpson, & Garfield, 2000).

It is possible that some vocal nodules cannot be treated with voice therapy alone, even if voice abuse/misuse is arrested (Gray, Hammond, & Hanson, 1995). If improper healing of the vocal fold has occurred, this can result in increased fibronectin deposition, which is usually a permanent event (Gray, et al., 1995). In these cases, a combined surgical and voice therapy treatment is likely to yield the best clinical result (Gray, et al., 1995). Surgical treatment without appropriate voice therapy is not recommended; characteristic features of MTD have been noted in non-dysphonic postoperative populations, indicating that the inappropriate vocal behaviors leading to surgery are still present and may lead to new lesion formation (Hsiung & Hsiao, 2004).

While patients with MTD appear to respond well to voice therapy, some findings indicate that there is limited success in the long term (Van Lierde, Claeys, De Bodt, & van Cauwenberge, 2006). A Belgian study looked at 27 respondents of an original 184 hyperfunctional voice patients an average of 6.1 years after a voice therapy program (Van Lierde, et al., 2006). In this subgroup, approximately 50% still showed pathological laryngological findings, and the average dysphonia severity index (DSI) was less than the average before treatment (Van Lierde, et al., 2006). However, these results are based on a small voluntary response sample, a type of sample is often biased, because people with strong (especially negative) options are more likely to respond (e.g., Moore & McGabe, 1998). A more general study of efficacy of speech therapy in 109 patients diagnosed with functional voice disorder found that only 56% were cured by speech therapy, and that therapy was necessary for three months or more in nearly half of patients who were eventually "cured" (Bridger & Epstein, 1983).

The majority of MTD patients respond well to speech therapy; however, in some cases it is inadequate (Bhalla, Wallis, Kaushik, & Carpentier, 2005). Another option for MTD patients resistant to speech therapy is the use of intravenous midazolam (Bhalla, et al., 2005). This sedative is used in conjunction with speech therapy to confirm the psychogenic nature of symptoms and to "prove" to the patient that a better voice is possible (Bhalla, et al., 2005). While this is the drug most commonly used for sedation in children and adults for procedures (Krauss & Green, 2000) and is generally felt to be safe

and effective (Parker, Mahan, Giugliano, & Parker, 1997), the use of any pharmaceutical adds extra risk and cost to patient treatment. Similarly, lidocaine bath was used on a group of MTD patients, finding a temporary reduction of symptoms (N. Roy, Smith, Allen, & Merrill, 2007). The need for more drastic methods to attempt to alleviate vocal hyperfunction supports the idea that there is a need for increased knowledge about the disorder to inform treatment decisions.

Surface electromyography

THEORY

As a nerve impulse reaches the motor end plates from an alpha motor neuron, all muscle fibers innervated by that axon comprise a single motor unit and are discharged nearly synchronously (see Figure 1-7). The electric potential field generated by the depolarization of the outer muscle-fiber membranes is essentially an amplified version of the alpha motor neuron activity; the electromyogram (EMG) is a representation of this "myoelectricity" as measured at some distance. Tissues separating the EMG signal sources (depolarized zones of the muscle fibers) act like spatial low-pass filters on the potential distribution, and constitute a volume conductor. Therefore, the EMG may be measured intramuscularly or at the surface of the skin, yielding different information based on the distance of the observation site from the muscle fibers. For surface detection particularly, the effect of the separating tissues becomes significant.
In order to remove interference sources and to compensate of the low-pass filtering effect of the tissue, surface signals are typically detected using a linear combination of different electrodes, the simplest of which is a differential electrode (Farina, Merletti, & Stegeman, 2004). Bipolar surface EMG (sEMG) is dependent upon on the inter-electrode distance (Roeleveld, Stegeman, Vingerhoets, & Van Oosterom, 1997). In measuring the sEMG, filtering is introduced by finite electrode size, inter-electrode distance, electrode configuration, electrode location, and characteristics of the front-end amplifier.

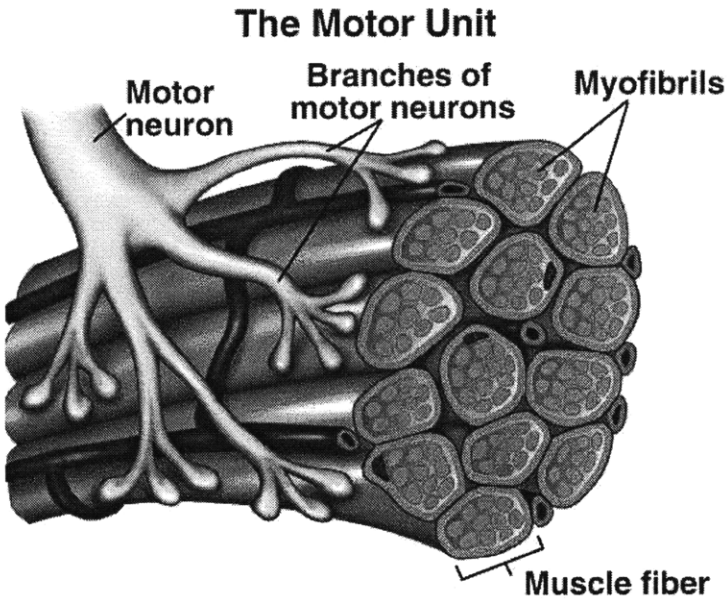


Figure 1-7. Schematic of the motor unit. Reproduced from Hall (2002).

RECORDING AND ANALYSIS RECOMMENDATIONS

The European Union sponsored a project termed SENIAM (Surface Electromyography for the Noninvasive Assessment of Muscles), one outcome of which was a set of recommendations for sEMG recording. In general, SENIAM recommends a maximum electrode size of 10 mm in the muscle fiber direction, with an interelectrode distance of approximately 20 mm or ¼ the length of the muscle fiber, whichever is smaller (Hermens, et al., 1999). Other recent recommendations include the assertion that smaller electrodes (diameter less than 5mm) are preferred for sEMG, as the larger electrodes introduce temporal low-pass filtering (Merletti & Hermens, 2004).

Skin preparation techniques can enhance electrode-skin contact, resulting in a reduction of artifacts and less noise. SENIAM recommends shaving the skin surface if it is covered with hair, and cleaning the skin in question with alcohol (Hermens, et al., 1999). Also preferred is the practice of slight skin abrasion or "peeling" with adhesive tape; this practice is known to reduce electrode-skin impedance, noise, DC voltages, and motion artifacts (Merletti & Hermens, 2004).

Recommendations for electrode placements are that the differential electrodes are applied between the innervation zone and a tendon. In the past, sensors have been placed over the belly or over the innervation zone (motor end plate zone), since this was the best location to record "large" monopolar sEMG signals. It is now well known that this location is not suitable for differential recordings; it is not stable or reproducible because relatively small displacements of the sensors with respect to the innervation zone cause large effects on the amplitude of the sEMG signal (Merletti & Hermens, 2004). Thus, in order for sEMG signals to be accurate and repeatable, there must be a clear definition of electrode position relative to the innervation zones (Hermens, et al., 1999). When the locations of innervation zones are unknown, use of double differential electrodes can diminish the effects of an ill-placed sensor (Farina, Merletti, & Disselhorst-Klug, 2004).

Ideal sEMG recording procedures would first identify the innervation zones and find the optimal electrode position on a subject by subject basis, using multi-channel electrode arrays. Falla et al. (2002), for example, examined the sternocleidomastoid (SCM) muscles in this way in 11 healthy normal individuals. Based on their findings, they have offered the following recommendations to optimize sEMG recordings from SCM muscles: the electrode should be placed 1/3 of the distance from the sternal notch to the mastoid process, in the direction of the line from the sternal notch to the mastoid process (Falla, et al., 2002). Recommendations of this type are not available for sEMG recordings of the extrinsic laryngeal muscles. With regard to ground locations, SENIAM recommends the wrist, the spinous process of C7, or the ankle as appropriate locations (Hermens, et al., 1999).

Because of the variability surrounding neck surface electrode contact and participant neck mass, sEMG signals should be normalized to a reference contraction before they are compared between conditions and/or participants (Netto & Burnett, 2006). Most especially, the layers of subcutaneous fat present can have attenuating and widening effects on the signal seen at the surface (Farina & Rainoldi, 1999). Common references include maximal voluntary contraction (MVC) and some percentage of the MVC (usually

50% or 60%). Studies have shown that for more simple, one-joint systems sub-maximal contractions are more reliable (Allison, Marshall, & Singer, 1993; J. F. Yang & Winter, 1983). However, Netto and Burnett (2006) found that for anterior neck musculature, the MVC reference was more reliable both within-day and between-days. The authors speculate that this is likely due to the complex structure and synergistic action of neck musculature.

The typical sEMG signal has 95% of its power in the frequency range less than 400 Hz. The remaining 5% is mostly electrode and equipment noise. For this reason, it is common to lowpass filter the signal with a cutoff point around 500 Hz or 1000 Hz. Movement artifacts create signals in the 0 – 20 Hz range, and can be attenuated by a high pass filter with a cut-off around 10 – 20 Hz. However, there may be some relevant information in this range of the EMG spectrum, specifically the firing rates of the active motor units. Notch filters to remove 50 or 60 Hz interference should not be used due to the high power density of the EMG signal in this range, and the phase rotation introduced to the time waveform (Hermens, et al., 1999).

Commonly used amplitude estimators are Average Rectified Value (ARV) and Root Mean Square Value (RMS; see Equation 1). In general, the "best" estimator of SEMG amplitude is RMS (smaller variance for Gaussian distributions, which sEMG approximates). The epoch used for amplitude estimation is recommended by SENIAM to be 0.25 – 0.5 sec for contraction levels above 50% MVC, or 1 – 2 sec for contraction levels below 50% MVC.

$$RMS = \sqrt{\frac{1}{N}\sum_{i=1}^{N} x_i^2}$$

Equation 1-1

Motivation

Although vocal hyperfunction is extremely common (e.g., N. Roy, 2003), it is also treatable with voice therapy (Carding & Horsley, 1992; Carding, et al., 1999; Holmberg, et al., 2001). However, the efficacious use of voice therapy for the treatment of vocal hyperfunction is limited by the ability of the therapist to reliably assess changes in the patient's vocal hyperfunction. There is no widely accepted diagnostic measure of the presence and degree of vocal hyperfunction, and currently, assessment of vocal hyperfunction during diagnosis is often primarily based on subjective impressions given the patient's history and presentation of symptoms such as auditory-perceptual and visual or tactile discrimination of muscle tension (e.g., laryngeal palpation).

Development of objective clinical assays of vocal hyperfunction could improve voice therapy treatment of patients with vocal hyperfunction by allowing for repeated, reliable assessment throughout treatment to identify progress. Past attempts to develop such measures have included the investigation of acoustic and aerodynamic parameters, both individually and in combination. However, presence of laryngeal pathology in some of the vocal hyperfunction patients (e.g., vocal fold nodules) causes glottal insufficiency that can impact aerodynamic measures, regardless of the presence of vocal hyperfunction, making it impossible to differentiate such effects from the separate influence of vocal hyperfunction. Thus, although glottal airflow magnitude and vocal efficiency are objective measures often collected in individuals with nodules to add additional information about the impairment of his or her phonatory function, they are rarely used diagnostically.

Given the symptom complex of vocal hyperfunction, work to investigate possible objective correlates of the multiple perceptual modalities involved may elucidate new objective measures. Here, we specifically examine vocal hyperfunction and many of the current perceptual measures used diagnostically (strain, supraglottic compression, and laryngeal palpation estimates of tension) using acoustics, aerodynamics, kinematics of the vocal folds, and sEMG.

Chapter 2: Use of Injection Laryngoplasty as a Platform for the study of Objective Measures of Vocal Hyperfunction

Purpose: **The validity of the use of office-based injection laryngoplasty was evaluated as a platform for the study of objective measures of vocal hyperfunction.**

Method: **Thirteen individuals undergoing office-based injection laryngoplasty for glottal insufficiency were prospectively studied using a battery of acoustic, aerodynamic, endoscopic, and surface electromyographic (sEMG) assessments before the procedure and approximately one week after.**

Results: **Perceptual ratings of strain and false vocal fold compression were both significantly reduced post-procedure ($p < 0.05$). However, there were not obvious correlations between any of the measures studied, including these two clinically-relevant perceptual tools.**

Conclusions: **Use of injection laryngoplasty as a platform for the study of some types of vocal hyperfunction is limited by the structural inability of the relevant study population to fully adduct, but may offer a convenient opportunity to study parameters associated with vocal hyperfunction that do not depend upon glottal competency, including possible correlates of strain. Based on this work, the continued use of strain and medial (FVF) ratings in clinical assessment of vocal hyperfunction is supported, whereas anterior-posterior (AP) ratings, the quantitative measures of AP and FVF compression studied here, and root-mean-squared-based sEMG measures are not currently recommended as clinical tools.**

Introduction

Vocal hyperfunction is a clinical term referring to "conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or 'imbalanced' muscular forces" (Hillman, et al., 1989). Current diagnosis of vocal hyperfunction and hyperfunctionally related voice disorders is dependent on subjective impressions of the patient's physical presentation, auditory-perceptual judgments of voice quality (in particular, vocal strain), and stroboscopy (Holmberg, et al., 2001). Clinical care is hindered by the lack of a "gold standard" objective measure for the assessment of vocal hyperfunction. Cross-sectional experimental designs comparing prospective objective measures between controls and individuals with vocal hyperfunction are limited by the tenuous assumption that all individuals in the disordered group truly display vocal hyperfunction and that none of the individuals in the control group do. Alternatively, potential measures could be tracked throughout therapy in individuals with vocal hyperfunction to determine if the measures change with successful rehabilitation. Unfortunately, extended time-courses of therapy and subsequent difficulties with participant retention diminish the usefulness of this design.

Injection laryngoplasty is an established procedure for correcting glottal insufficiency (Courey, 2004) through injection of material into the vocal fold. Hillman et al. (1989) hypothesized a relationship between glottal insufficiency and vocal hyperfunction, in which increased activity of laryngeal muscles could be used in an effort to achieve more adequate glottal closure. This agrees with the clinical observation that individuals with severe glottal insufficiency reporting for injection laryngoplasty often have concurrent vocal hyperfunction. After injection laryngoplasty, these patients can often "unload" their habituated hyperfunctional behaviors that are no longer necessary, creating a population in which vocal hyperfunction may change drastically in a short period of time. This paper evaluates the validity of the use of office-based injection laryngoplasty as a platform for the study of objective measures of vocal hyperfunction, such as neck surface electromyography (sEMG).

Method

Participants were 13 adults (6 males, 7 females; mean age = 56 years, SD = 15 years) undergoing office-based injection laryngoplasty (Restylane) due to glottal insufficiency, including vocal fold paralysis, bilateral sulci, and presbyphonia. Participants underwent acoustic, aerodynamic, endoscopic, and electromyographic assessments immediately prior to their injection laryngoplasty (PRE) and approximately 1 week after their procedure (POST), allowing time for the injected material to distribute. Actual times between PRE and POST measurements varied from 6 – 11 days, with most at 7 days.

Participants were examined using transnasal endoscopy. Short video segments of speech, repeated abduction and adduction, and an /i/ vowel captured during each PRE and POST exam were rated on a 5-point scale for anterior-posterior (AP) and medial (false vocal fold; FVF) sugraglottic compression (M. E. Smith, Ramig, Dromey, Perez, & Samandari, 1995) by a certified speech-language pathologist (SLP) and a laryngologist, both of whom subsequently re-rated approximately 25% of the samples. Images of the adducted folds during /i/ production were analyzed using MATLAB® (Mathworks Inc., Natick, MA) to obtain proposed quantitative parameters for AP and FVF compression (Behrman, Dahl, Abramson, & Schutte, 2003).

Using an Phonatory Aerodynamic System (KayPENTAX, Lincoln Park, N.J.), participants produced a series of /pæ/ vocalizations at both comfortable and loud sound pressure levels while the airflow and intra-oral air pressure was measured with a translabially placed catheter connected to a pressure transducer. Signals were digitized and analyzed with the Phonatory Aerodynamic System acquire airflow and indirect estimates of subglottal air pressure. Vocal effectiveness was calculated, operationally defined as the ratio of the sound pressure level in dB SPL to the estimated subglottal air pressure in cm H_2O.

Simultaneous neck sEMG and acoustic signals from a lavalier microphone (Sennheiser MKE2-P-K, Wedemark, Germany) were filtered and recorded digitally with Delsys ™ (Boston, Massachusetts) hardware (Bagnoli Desktop System) and software (EMGworks 3.3) with a sampling frequency of 20 kHz during production of vowels /a/ and /i/ and read speech. The sEMG was recorded with two double-differential electrodes (Delsys ™ 3.1) placed parallel to the underlying muscle fibers of the 1) thyrohyoid, omohyoid, and sternohyoid muscles and 2) cricothyroid and sternohyoid muscles, and a ground electrode on the superior aspect of the participant's left shoulder. For detailed descriptions of electrode placement and a figure showing electrode placements, please see Figure 4-3 of Chapter 4 of this thesis. sEMG signals were amplified (1000 gain) and filtered (20 – 450 Hz band-pass) using Delsys™ Bagnoli systems. All participants performed maximal voluntary contraction (MVC) maneuvers, consisting of neck contraction against manual resistance for the purpose of normalizing sEMG data. For a subset of the participants (N = 8), the maximal force was measured with a dynamometer (Chatillon DPP-50, Ametek,

Inc., Paoli, PA) during neck muscle contraction. These MVC forces ranged from 16 – 47 lb_f by participant, and did not significantly differ between PRE and POST recordings. Potential variability associated with surface electrode contact and placement between PRE and POST recordings was minimized by normalizing sEMG to the reference contraction at MVC (e.g., Netto & Burnett, 2006). All sEMG was analyzed as root-mean-squared (RMS) in windows of 1 s and normalized via MVC using custom MATLAB® software, resulting in RMS sEMG in terms of % MVC.

A certified SLP perceptually rated the strain of each participant's voice as recorded PRE and POST with the CAPE-V (Kempster, Gerratt, Verdolini Abbott, Barkmeier-Kraemer, & Hillman, 2009). Approximately 25% of samples were re-rated by the SLP and by a second certified SLP. The acoustic rise times of the vowels /a/ and /i/ were used as acoustic correlates of abruptness of attack, as suggested by Peters et al. (1986). Here, due to the disordered nature of the voice signals, the rise time of the acoustic signal was modified as the time required for an envelope of the acoustic signal to go from 20% to 80% of the maximum amplitude, which was implemented in MATLAB®, with the RMS of the acoustic signal in 80 ms rectangular windows calculated in intervals of 2.5 ms (97% overlap).

Minitab® Statistical Software (Minitab Inc., State College, PA) was used to calculate Pearson's correlations between changes in the various measures and Student's *t*-tests to assess possible changes between the PRE and POST collected parameters. Statistical testing was not adjusted for alpha inflation due to the exploratory nature and small sample size of this study. Calculations of Pearson's correlations had 90% power to identify correlations with effect sizes greater than $R^2 \geq 0.5$.

Results

Student's *t*-tests were performed on all parameters between POST and PRE conditions, and are tabulated in Table 2-1. The following measures showed statistically significant (paired, two-sided, $p < 0.05$) differences: loud vocal effectiveness, strain, FVF ratings, comfortable airflow, and loud airflow. Pearson's correlations among POST – PRE changes in parameters thought to be associated with vocal hyperfunction are shown in Table 2-2. Estimates of intra- and inter-rater reliability for strain, AP compression, and FVF compression are shown in Table 2-3.

Parameter	Difference	*p*-value
Strain	-28	**0.001**
/a/ Rise Time (ms)	0.5	0.984
/i/ Rise Time (ms)	1.0	0.934
AP rating	-0.31	0.120
FVF rating	-0.69	**0.041**
AP quantitative measure	-0.05	0.424
FVF quantitative measure	-0.27	0.152
V Eff C (dB SPL / cm H_2O)	-1.01	0.054
V Eff L (dB SPL / cm H_2O)	-1.01	**0.013**
Airflow C (L / s)	-0.13	**0.037**
Airflow L (L / s)	-0.24	**0.016**
Difference = mean POST – PRE, V Eff = vocal effectiveness, C = comfortable, L = loud, AP = anterior-posterior, FVF = false vocal fold. *p*-values shown in bold indicate parameters found to be statistically significantly different POST relative to PRE at a $p < 0.05$ level.		

Table 2-1. Student's *t*-tests for POST vs. PRE conditions (two-tailed, paired, N=13)

	a)	b)	c)	d)	e)	f)	g)	h)	i)	j)	k)	l)	m)	n)	o)
a) Strain															
b) /a/ Rise Time	-0.25														
c) /i/ Rise Time	**-0.56**	**0.88**													
d) AP rating	0.10	0.49	0.39												
e) FVF rating	0.32	0.42	0.25	**0.60**											
f) AP quant	-0.10	0.08	0.07	0.49	0.20										
g) FVF quant	**-0.59**	-0.08	0.09	-0.34	-0.50	-0.27									
h) Vocal Effectiveness C	-0.43	0.53	**0.64**	0.11	0.29	-0.04	0.26								
i) Vocal Effectiveness L	-0.07	0.41	0.48	0.39	0.43	-0.17	0.24	0.68							
j) EMG1 /a/	0.05	-0.31	-0.23	-0.04	-0.23	0.33	-0.01	0.05	-0.04						
k) EMG2 /a/	0.21	**-0.74**	**-0.73**	-0.50	-0.32	0.03	0.04	-0.14	-0.37	**0.64**					
l) EMG1 /i/	0.24	-0.07	-0.19	0.10	0.06	0.04	0.20	-0.01	0.28	**0.66**	0.26				
m) EMG2 /i/	0.02	-0.45	-0.32	0.20	0.05	0.51	-0.19	0.05	0.03	0.36	0.49	-0.11			
n) EMG1 speech	0.18	-0.08	-0.14	0.21	0.14	0.05	0.12	0.09	0.27	**0.74**	0.36	**0.92**	0.01		
o) EMG2 speech	-0.13	-0.29	-0.18	0.19	0.06	0.39	-0.11	0.25	0.08	0.15	0.37	-0.32	**0.91**	-0.16	

C = comfortable, L = loud, AP = anterior-posterior, FVF = false vocal fold, quant = quantitative measure. Numbers in bold indicate correlations statistically significant at a $p < 0.05$ level.

Table 2-2. Pearson's correlations for POST - PRE changes between parameters

	Strain			AP	FVF
Intra-rater	0.69		Rater 1 Intra-rater	1.00	0.78
Inter-rater	0.65		Rater 2 Intra-rater	0.94	0.64
			Inter-rater	0.69	0.73

Table 2-3. Inter- and Intra-rater reliabilities (Pearson's correlations) of perceptual measures

Discussion

Testing between PRE and POST conditions showed that, as expected, individuals displayed lower airflow rates, lower strain ratings, and lower ratings of FVF supraglottic compression post-injection. However, few collected measures were significantly correlated. This might suggest that injection laryngoplasty is an inadequate platform for the study of vocal hyperfunction, that the measures tested were inadequate correlates of vocal hyperfunction, some combination of these two, or that vocal hyperfunction might not exist as a measurable phenomenon.

Interestingly, although both strain and FVF compression ratings were significantly reduced in the POST condition relative to the PRE condition, they were not significantly correlated, even though the intra- and inter-rater reliabilities of the judges in this study were equal or greater than those published in previous studies (e.g., Kelchner, et al., 2009; M. E. Smith, et al., 1995). These data suggest that the widely used perceptual measures of strain and FVF ratings may not be measuring the same type of vocal hyperfunction. Hillman and colleagues (1989) also proposed multiple types of vocal hyperfunction: the adducted variety associated with the formation of vocal fold lesions such as nodules, and the non-adducted variety associated with combinations of muscle activity associated with functional dysphonia. Results here suggest there may be more than one modality of vocal hyperfunction, and that the auditory perception of strain is likely not associated with supraglottic compression. It is possible that perception of strain is more related to the longitudinal tension of the vocal folds caused by activity of the cricothyroid and/or thyroarytenoid musculature than it is to supraglottic postures.

The measures utilizing estimates of subglottal pressure were not correlated with either perceptual measure and even indicated that individuals had lowered vocal effectiveness after their procedure, using higher levels of subglottal pressure, which was not expected. An explanation might be that estimates of subglottal pressure are not accurate approximations in individuals with severe glottal insufficiency. In fact, the structural inability to fully adduct the vocal folds likely hindered measurements of both vocal effectiveness and also vowel rise times.

We assert that there are at least two types of vocal hyperfunction, one associated with medial (FVF) compression and the other associated with the perception of strain. The latter type could be studied very easily using injection laryngoplasty, and given the short time-frame that this procedure allows, this is a recommended experimental design. However, due to the structural deficiencies in the relevant population, injection laryngoplasty does not present an appropriate platform for which to study vocal hyperfunction related to medial compression or other adductory functions.

Although we believe that some of the parameters collected here (vocal effectiveness and vowel rise times) were compromised by the structural inability to adduct the vocal folds in the PRE condition, the remainder of the parameters investigated should not be

adversely affected, and so can be evaluated for their utility in measuring vocal hyperfunction. The perceptual measures of strain and supraglottic compression (AP and FVF) are currently primary clinical indicators of vocal hyperfunction. The continued use of strain and FVF ratings is supported by the fact that both measures were significantly reduced in the POST condition. Although the perception of strain has poor reliability (e.g., Kelchner, et al., 2009), we believe its use is warranted until a more objective measure can be found. While ratings of FVF compression were only significantly correlated with AP compression ratings, this perceptual measure was reasonably reliable and seems like an appropriate perceptual measure with which to judge possible objective measures. Ratings of AP compression are also used in the clinic as a primary indicator, but they did not significantly change between the PRE and POST conditions, and were only correlated with FVF ratings. This agrees with the previous suggestion that AP compression is more associated with typical laryngeal articulation than with vocal hyperfunction (Stager, et al., 2000).

Use of objective measures of supraglottic compression is tempting, but the results indicate that the quantitative measures of AP and FVF suggested by Behrman et al. (2003) are not accurate correlates of the perceptual measures. This work does not support their use as indicators of vocal hyperfunction, or even more specifically of supraglottic compression.

The relationship between vocal hyperfunction and extrinsic laryngeal muscle tension is still unclear, despite the fact that extrinsic neck musculature is a primary therapy target. Two past studies attempted to use sEMG to objectively quantify neck muscle tension in individuals thought to have vocal hyperfunction (Hocevar-Boltezar, Janko, & Zargi, 1998; Redenbaugh & Reich, 1989), both finding higher RMS sEMG in individuals with voice disorder. However, both studies were compromised by methodological failings, and more current work has not shown consistent differences in RMS sEMG between individuals with vocal hyperfunction and controls [see Chapter 5 of this thesis work]. Further, previous work to classify sEMG in individuals with vocal hyperfunction have been cross-sectional designs, which are hindered by possible contamination of groups. The current study does not show consistent correlations between RMS sEMG measures from the anterior neck and any of the other possible measures of vocal hyperfunction. Given the care taken to control for sources of measurement variability in this experimental study, the current work does not support the addition of sEMG measures to the armamentarium of voice assessment.

SUMMARY

Use of injection laryngoplasty as a platform for the study of some types of vocal hyperfunction is limited by the relevant study population's impaired vocal fold adduction. However, it may offer a convenient opportunity to study parameters associated with vocal hyperfunction that do not depend upon glottal competency, including possible correlates of strain. Based on this work, the continued use of strain and medial (FVF) ratings in clinical assessment of vocal hyperfunction is supported, whereas AP ratings, the quantitative measures of AP and FVF compression studied here, and RMS-based sEMG measures are not currently recommended as clinical tools.

Chapter 3: A Virtual Trajectory Model Predicts Differences in Vocal Fold Kinematics in Individuals with Vocal Hyperfunction

A simple, one degree of freedom virtual trajectory model of vocal fold kinematics was developed to investigate whether kinematic features of vocal fold movement confirm increased muscle tension. Model simulations verified that increases in stiffness were associated with changes in kinematic parameters, suggesting that increases in gesture rate would affect kinematic features to a lesser degree in vocal hyperfunction patients given the increased levels of muscle tension they typically employ to phonate. This hypothesis was tested experimentally in individuals with muscle tension dysphonia (MTD; N = 10) and vocal nodules (N = 10) relative to controls with healthy normal voice (N = 10) who were examined with trans-nasal endoscopy during a simple vocal fold adductory-abductory task. Kinematic measures in MTD patients were less affected by increased gesture rate, consistent with the hypothesis that these individuals have elevated typical laryngeal muscle tension. Group comparisons of the difference between medium and fast gesture rates (Mann-Whitney, one-tailed) showed statistically significant differences between the control and MTD individuals on the two kinematic features examined (p <0.05). Results in nodules participants were mixed and are discussed independently. The findings support the potential use of vocal fold kinematics as an objective clinical assay of vocal hyperfunction.

Introduction

VOCAL HYPERFUNCTION

Vocal hyperfunction refers to "conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or 'imbalanced' muscular forces" (Hillman, et al., 1989), characterized by excessive laryngeal and paralaryngeal tension (Aronson, 1980; Dworkin, Meleca, & Abkarian, 2000; Koufman & Blalock, 1991b; M. D. Morrison, et al., 1983; N. Roy, et al., 1996) that commonly accompanies disordered voice production. Estimates of the prevalence of vocal hyperfunction indicate that the condition may account for 10 – 40 % of cases referred to multidisciplinary voice clinics (N. Roy, 2003). Vocal hyperfunction could be due to psychological and/or personality factors, misuse of muscles, learned adaptation following a short-term respiratory illness such as a cold, or compensation for underlying vocal disease (Aronson, 1980; Hsiung & Hsiao, 2004; M. D. Morrison, et al., 1986; M. D. Morrison & Rammage, 1993; M. D. Morrison, et al., 1983; Rammage, et al., 1987). However, claims of increased muscle tension or stiffness are largely based on subjective accounts of the symptom complex. Although Redenbaugh and Reich (1989) showed increased surface electromyographic signals on the anterior neck in individuals with a "hyperfunctional voice disorder" relative to normal controls, no study has shown objective evidence for increased tension of intrinsic laryngeal muscles and structures.

Individuals who display disordered voice production that is associated solely with vocal hyperfunction in the absence of any other laryngeal pathology are often diagnosed as having muscle tension dysphonia (MTD; Hsiao, et al., 2001), according to a common definition of MTD. Benign lesions such as nodules and polyps on the vocal fold surface have been assumed to be related to hyperfunctional behavior for many years (Hillman, et al., 1990). For instance, in 1962, Godrey Arnold wrote that "vocal nodules and polyps represent a local tissue reaction to the mental strain imposed by inappropriate emotional adjustment to the demands made by society" (Arnold, 1962). Vocal fold nodules are defined as benign lesions that occur bilaterally between the anterior and middle third of the vocal fold (Aronson, 1980; Marcotullio, et al., 2002). One clinic found that 92% of cases of vocal nodules were coincident with symptoms of vocal hyperfunction (M. D. Morrison, et al., 1983) indicating a strong relationship between the incidence of vocal hyperfunction and vocal nodules. However, due to the possible effects of compensation once there is organic pathology on the vocal folds (i.e., reactive hyperfunction), no clear cause-and-effect relationship between vocal hyperfunction and nodules has been established. Therefore, while individuals diagnosed with MTD, by definition, have vocal hyperfunction, this may not be true in all cases of vocal nodules.

An obstacle to efficacious use of voice therapy for the treatment of vocal hyperfunction is the limited ability of the therapist to reliably ascertain if and when there are changes in the degree to which a patient is hyperfunctioning during voice production. Currently, the

best measures for rating the success of therapy approaches are voice quality, aerodynamic changes, and visual examination of the vocal folds using videoendoscopy (Holmberg, et al., 2001). However, none of these measures provide unambiguous assessment of the underlying hyperfunctional status of the larynx during phonation, particularly in cases where the interpretation of measures is confounded by the presence of organic pathology such as vocal nodules (e.g., nodules can impact aerodynamic measures by interfering with glottal closure). If differences in vocal fold kinematics between vocally-normal individuals and individuals with vocal hyperfunction (with or without organic changes) exist, these could be used as an objective clinical assay of vocal hyperfunction to aid in the assessment and treatment of patients with hyperfunctionally-related voice disorders.

VOCAL FOLD KINEMATICS

Using rigid (trans-oral) videostroboscopy and visual detection of vocal fold landmarks, Cooke et al. (1997) examined the linear kinematics of vocal fold adductory gestures during different types of glottal attack ("hard", "normal", and "breathy") in individuals with healthy normal voice. Overall articulator stiffness ("stiffness parameter") was approximated as in the work of Munhall and Ostry (1983) as the ratio of the maximum (linear) adduction velocity to the extent of the motion, and was found to be increased for "hard" voice onset relative to normal and breathy vocal onset. Cooke et al. (1997) suggested that this finding offers quantitative support for the idea that hard vocal onset involves greater laryngeal muscle tension than normal vocal onset.

More recently, Dailey and colleagues (2005) monitored the angle of the true vocal folds (glottic angle) as a function of time during a "sniff – eee" task in women with healthy normal voice using flexible (trans-nasal) videoendoscopy. The glottic angle is defined as the angle between the two vocal folds at the anterior commissure. They found that the mean angular velocities of the glottic angle during abduction and adduction were higher when participants were asked to perform the repetitions of the sniff-eee task more quickly; however, there was a greater influence seen on adduction than on abduction. Specifically, increasing the gesture rate caused a 46% average increase in the mean angular velocity during adduction, but only a 6.1% average increase during abduction (see Table 3-1 for reproduction of the results of Dailey et al. (2005)). The authors speculate that this increased velocity during "quick adduction" is likely the result of the "greater control over recruitment of motor units in adduction."

	Experimental						Modeling		
Gesture Rate	Dailey *et al.*		CTRL		MTD		Stiffness		
	AB	AD	AB	AD	AB	AD		AB	AD
Medium	396	318	366	390	312	361	Medium	434	357
Fast	420	464	398	481	390	420	High	443	438
% Increase	6.1 %	46 %	8.8 %	23 %	25 %	16 %	% Increase	2.2 %	23 %

*velocities are presented in deg/s. CTRL = control participants (N = 10) from the current work. MTD = MTD participants (N = 10) from the current work. AB = abduction; AD = adduction.

Table 3-1. Abductory and adductory mean angular velocities

However, it is possible that increased repetition rate results in a global increase in stiffness. If so, a simple one-joint virtual trajectory model of laryngeal adduction and abduction may capture these changes in velocity based on increases in stiffness. If a "smooth" input of the virtual trajectory results in a glottic angle similar to data gathered by Dailey et al. (2005), this would indicate applicability of such an approach (and specifically the minimum jerk variant used here) to modeling the abduction and adduction of the human vocal folds. As the overall stiffness of the muscles modeled is increased (modeling an increase in gesture rate of the task), there may be changes due purely to the mechanics causing the differences in adductory and abductory speed changes noted by Dailey et al. (2005). Further, correspondence of the "stiffness parameter" measured by Cooke et al. (1997) with increases in the stiffnesses of modeled muscles will validate the relationship between muscle stiffness and that kinematic parameter. This is an important objective given that this parameter does not have dimensions physically appropriate for stiffness or tension, but rather [1 / time].

This work employs a simple model of vocal fold abduction and adduction in order to confirm the relationship of intrinsic laryngeal muscle stiffness with kinematic estimators of stiffness. Based on the results of the model, the work further experimentally tested the hypothesis that increases in gesture rate of a repetitive abduction-adduction task would affect kinematic estimators of stiffness less in individuals with vocal hyperfunction due to their typically increased laryngeal muscle stiffness.

Modeling Vocal Fold Abduction and Adduction

Model Formulation

Although virtual trajectory models (also referred to as equilibrium point models) have been applied to speech systems by many researchers (e.g., Perrier, Loevenbruck, & Payan, 1996; Sanguineti, Laboissiere, & Ostry, 1998) with some success, previously described biomechanical models of the larynx have largely focused on the dynamics of vocal fold tissues to produce and manipulate the pitch of voicing (e.g., Alipour, Berry, & Titze, 2000; J. J. Jiang, Diaz, & Hanson, 1998; e.g., Story & Titze, 1995). Models of abduction and adduction (vocal fold posturing) have been created by several investigators (Farley, 1996; Hunter, Titze, & Alipour, 2004; Titze & Hunter, 2007). These rigorous models of the larynx explore the role of various muscle activations on the control of abduction and adduction, but their simulations require the estimation of complex muscle activation functions, adding complexity and uncertainty. Here, a simple one-joint (unilateral) virtual trajectory model based around the arytenoid joint was designed to examine key aspects of the biomechanics of laryngeal abduction and adduction. The choice of a virtual trajectory model was made based on the simplicity of the approach: virtual trajectory models assume that higher levels of the central nervous system encode movement by specifying a series of postures (the virtual trajectory), and that the dynamic details of movement are a result of the peripheral neuro-muscular system. Thus, for a simple model of abductory and adductory gesturing, the only input required to the model is the single virtual trajectory. The model employed here greatly simplifies the physiological system and ignores the translation of the arytenoids as well as their rotation in the sagittal plane, and also assumes symmetry of the control of the bilateral arytenoid joints, formally modeling only one side. Figure 3-1 panel A shows a schematic of the key anatomy of this transverse section at the level of the vocal folds while panels B and C show the geometric model employed in the abductory and adductory states, respectively.

The geometry of the model was based on anatomical measurements of women, so that the simulation results could be compared with data from Dailey et al. (2005) who examined vocal fold motion in 21 women aged 19 – 27 years, making the modeling results semi-specific to these smaller larynges. It uses a transverse section at the level of the glottis and includes three muscles: the thyroarytenoid (TA), lateral cricoarytenoid (LCA), and posterior cricoarytenoid (PCA). The muscles are modeled as simple springs, each represented by a parallel stiffness and damping, with the single "input" from the neural system defined as the joint angle, θ, the angle between the muscular process of the arytenoid and the line perpendicular to the midline of the glottis. Damping values of 10% of the stiffness were used to mimic the work of Flash and colleagues (1987) who found that these values often produced results closest to those seen physiologically. The initial

Parameter	Physical Meaning	Value	Reference
ℓ_1	length of arytenoid in the x-direction	0.014 m	Measurement 'Ab' from Kim *et al.* (2004) (human women only)
ℓ_2	length of arytenoid in the y-direction	0.0126 m	Measurement 'ab' from Kim *et al.* (2004) (human women only)
ℓ_3	distance from the origin to the vocal fold apex	0.0229 m	Measurement 'jF' from Kim *et al.* (2004) (human women only)
M	total mass of the arytenoid cartilage	0.5 g	Measured by James T. Heaton and James B. Kobler on cadaver larynges
d	diameter of the "cylinders" of the arytenoid	0.00365 m	Estimated as 2/3 of the length 'VW' in Kim *et al.* (2004) (human women only)
y_1	distance from the origin to the insertion of the PCA	0.00348 m	Measurement 'ON' from Kim *et al.* (2004) (human women only)
x_2	distance from the insertion of the LCA to the y-axis	0.0144 m	Estimated as ½ of the length of 'hh' in Kim *et al.* (2004) (human women only)
y_2	distance from the insertion of the LCA to the x-axis	0.0114 m	Estimated as ½ of the length of 'jF' in Kim *et al.* (2004) (human women only)
k_{PCA}	stiffness of the PCA	119 N/m	Estimated from young's modulus data and average length data from Hunter *et al.* (2004) and Alipour *et al.* (2005)
k_{LCA}	stiffness of the LCA	76.5 N/m	Estimated from young's modulus data and average length data from Hunter *et al.* (2004) and Alipour *et al.* (2005)
k_{TA}	stiffness of the TA	640 N/m	Estimated from young's modulus data and average length data from Hunter *et al.* (2004) and Alipour *et al.* (2005)
b_{PCA}	viscosity of the PCA	11.9 N/m/s	10% of kPCA estimate as in (Flash, 1987)
b_{LCA}	viscosity of the LCA	7.65 N/m/s	10% of kLCA estimate as in (Flash, 1987)
b_{TA}	viscosity of the TA	64 N/m/s	10% of kTA estimate as in (Flash, 1987)

Table 3-2. Initial values for constant parameters.

values used in the model and the basis for those choices are shown in Table 3-2. A sensitivity analysis was performed to explore alternative values. Although varying the model values did quantitatively alter the modeling results, the overall effects were qualitatively similar. The muscle models used in this work are a simplified version of the Hill model, which is composed of a spring in series with the parallel combination of a spring, damper, and force generator (see McMahon, 1984). This highly simplified muscle model was chosen because of its computational tractability and the uncertainty associated with choosing multiple muscle parameters. It should, however, be acknowledged that actual muscle physiology differs significantly from that of a simple spring.

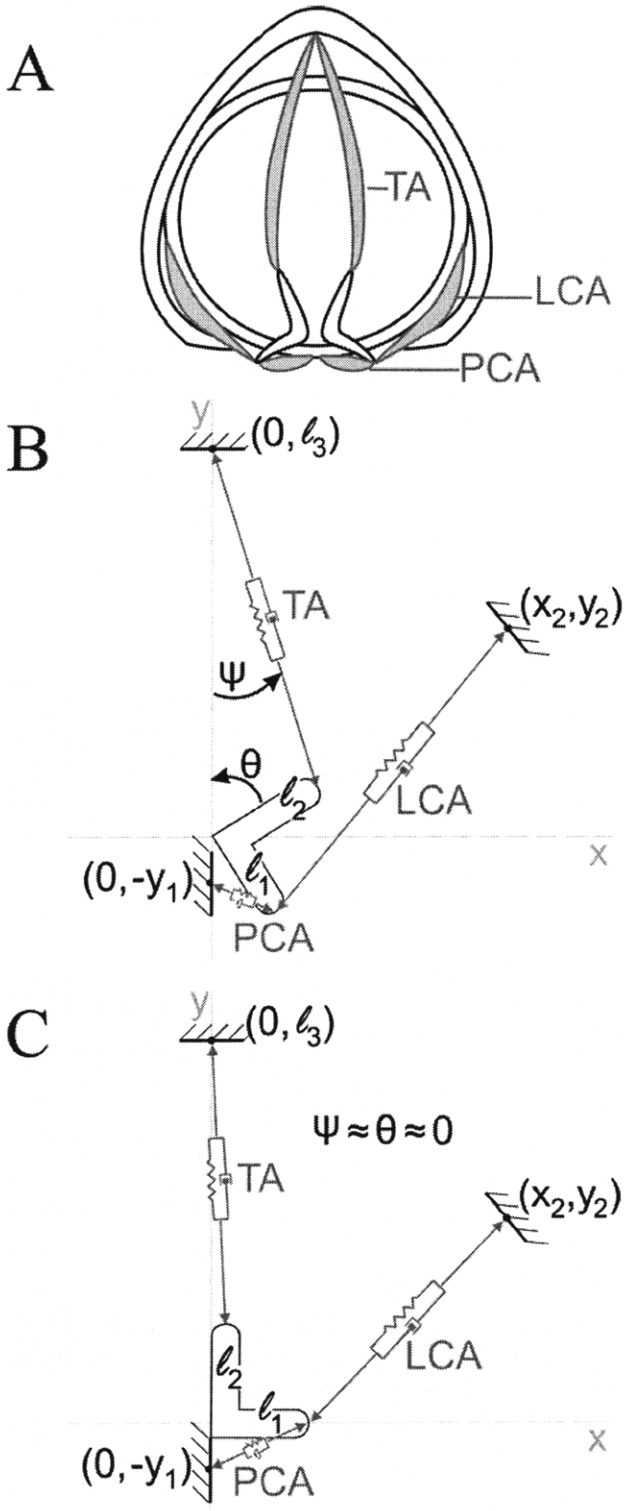


Figure 3-1. Panel A. A schematic of a transverse section through the larynx at the level of the vocal folds. Panel B. Model of vocal fold abduction. Double-sided arrows indicate muscles modeled as parallel springs and dampers. Panel C. Model of vocal fold adduction.

In this formulation, the specific form of the system dynamics is as shown in Equations 3-1 and 3-2, where the net torque is equal to the product of the inertia, I, and the angular acceleration, as well as to the sum of the torques, τ, imposed by the three muscles, designated with the subscripts TA, LCA, and PCA.

$$\tau_{net} = I\ddot{\theta} \qquad \text{Equation 3-1}$$

$$I\ddot{\theta} = \tau_{TA}(\theta,\dot{\theta},\theta_v) + \tau_{LCA}(\theta,\dot{\theta},\theta_v) + \tau_{PCA}(\theta,\dot{\theta},\theta_v) \qquad \text{Equation 3-2}$$

The torque imposed by each muscle is a function of its force and moment arm, MA, as is indicated in the example in Equation 3-3, both of which are dependent upon the current angle of rotation θ. The force produced by each muscle is a function of current muscle length, ℓ, with respect to the virtual length, ℓ_v, which is made explicit in equation 3-4.

$$\tau_{musc} = MA_{musc}(\theta)F_{musc}(\theta,\dot{\theta},\theta_v) \qquad \text{Equation 3-3}$$

$$F_{musc} = k_{musc}(\ell_v(\theta_v) - \ell(\theta)) - b_{musc}\dot{\ell}(\dot{\theta}) \qquad \text{Equation 3-4}$$

The inertia of the arytenoid cartilage was calculated based on its dimensions and mass (see Table 3-2). Its geometry was approximated as three cylinders, meeting at the origin of rotation.

The choice of the form of the virtual trajectory is a key factor in the performance of a virtual trajectory model. The most straightforward approach might be to supply the system with a step function in which the virtual trajectory moves directly from the initial position to the final position ("final position control"). Although this approach may be appropriate for ballistic movements, it has shown to be inadequate for slow and medium speed arm reaching movements (Bizzi, Accornero, Chapple, & Hogan, 1984). In the model, use of this "final position control" lead to movement simulations that were not physiologic. Thus, here, the virtual trajectory used was a gradual shift from initial to final position. Specifically, the virtual trajectory was chosen based on an accepted criterion function minimizing the mean-square jerk and optimizing the "smoothness" of the resulting trajectory (e.g., Hogan, 1984). The criterion function is minimized as is shown in Equation 3-5, where θ is the angle and D is the desired duration of the movement.

$$\min C = \min\left(\int_0^D (\dddot{\theta})^2\, dt\right) \qquad \text{Equation 3-5}$$

Dynamic optimization was used to find the minimum mean-square jerk trajectory, which was found to be in the form of a fifth-order polynomial in time. The coefficients for the polynomial were solved for with the following boundary conditions (Equations 3-6 - 3-11), where θ_i and θ_f are the initial and final values of the desired θ, respectively, and D indicates the duration of the movement.

$$\theta(0) = \theta_{start} \qquad \theta(D) = \theta_{finish}$$
$$\dot{\theta}(0) = 0 \qquad \dot{\theta}(D) = 0$$
$$\ddot{\theta}(0) = 0 \qquad \ddot{\theta}(D) = 0$$

Equations 3-6 – 3-11

These boundary conditions lead to an equation for a minimum mean-square jerk virtual trajectory, shown as Equation 3-12.

$$\theta_v(t)=\theta_i+(\theta_f-\theta_i)\left[10\left(\frac{t}{D}\right)^3-15\left(\frac{t}{D}\right)^4+6\left(\frac{t}{D}\right)^6\right]$$

Equation 3-12

Here, the virtual trajectory used as inputs to the system were θ_v values from 0 degrees to 35 degrees (abduction) and 35 degrees to 0 degrees (adduction) and D = 250 ms. These motions result in changes in the glottic angle ranging from 0 degrees (adduction) to 60 degrees (abduction). These values were chosen to correspond with the results reported by Dailey et al. (2005).

The initial values of stiffness chosen (see Table 3-2) were unable to recreate movements within the ~250 ms as reported in the literature (Dailey, et al., 2005). In order to create realistic motions, a scaling factor was applied to all muscle stiffness parameters. The minimum scaling factor necessary to produce movements fast enough was a value of 3, a reasonable increase given the uncertainty in the estimates used, as well as the fact that stiffness-based estimates were made from static measurements. Because muscle stiffness increases with force (Hoffer & Andreassen, 1981), the dynamic stiffnesses are likely to be higher than measured static values. Modeling work by Flash and colleagues found physiologically-relevant simulation results when values of dynamic stiffness were at least two times higher than the static values (Flash, 1987).

This virtual trajectory model was used to simulate abduction and adduction at "normal" stiffness (scaling factor = 3) as well as at "high" stiffness, in which the scaling factor was increased by a factor of 4 (scaling factor = 12). The variation in stiffness was used to approximate the changes in muscle stiffness thought to be a result of increasing the gesture rate during the "sniff – eee" task. Thus, normal stiffness was used to correspond to the medium gesture rate and high stiffness to the fast gesture rate. Various increases in muscle stiffness were explored to approximate "high" stiffness. Small variations about the chosen scaling factor (10 – 14) did not show major differences in performance; however, extremely high scaling factors (> 15) resulted in minimal increases in the effects on the modeled movement, due to a ceiling effect. Specifically, at these very high stiffnesses, movements did not significantly differ from the given virtual trajectory, and increases in stiffness caused imperceptible changes.

The resulting abductory and adductory movements created by the model at "normal" and "high" stiffness were used to calculate several kinematic parameters of interest. The mean angular velocity was calculated as in Dailey et al. (2005): the slope between the points of intersection of the movement with the values 20% and 80% of the maximum angle was used as the mean velocity for each gesture. The "stiffness parameter" was calculated as the ratio between the maximum angular velocity and the maximum glottic angle.

MODELING RESULTS AND PREDICTIONS

This simple model was able to simulate abductory and adductory vocal fold movements consistent with the data of Dailey et al. (2005). For both abduction and adduction, the

20%-80% mean angular velocity was found at both high and normal stiffness (see Table 3-2). These mean values were consistent with those found during "regular" and "fast" gesture rates by Dailey and colleagues (2005); further, the percent increase between medium and high stiffness showed the same trend as that between regular and fast gesture rates seen by Dailey et al. (2005).

Although Dailey et al. (2005) speculated that the increased velocity during "quick adduction" was likely the result of the "greater control over recruitment of motor units in adduction," the purely mechanical model employed here was able to reproduce the qualitative difference between abduction and adduction changes in velocity. However, the model results do not quantitatively agree with the published data (23% vs. 46%, found in Table 3-1). This could be due to a number of factors: over-simplification of the mechanical system, poor parameter choices, or real neural recruitment differences as postulated in Dailey et al. (2005). It is more likely the differences are related to the overly simplified model and uncertainty in model parameters.

At the normal model stiffness values (scaling factor = 3), the stiffness ratio was found to be 6.4, increasing to 7.8 at high model stiffness values (scaling factor = 12). The increase found here between normal and high model stiffness is roughly comparable to the average difference seen between normal and hard onset stiffness values measured using linear velocities previously (Cooke, et al., 1997).

These modeling results support the idea that the differential changes in the abductory and adductory velocities are a mechanical consequence of higher stiffness, and that this higher stiffness is consistent with increasing the gesture rate of abduction and adduction in normal controls (i.e., Dailey, et al., 2005). Because the underlying characteristic of vocal hyperfunction is thought to be an increase in the tone (stiffness) of laryngeal musculature, these modeling results suggest that increases in gesture rate will affect the speed of adduction and the "stiffness parameter" far less in patients with vocal hyperfunction since their typical stiffness may already be elevated, an hypothesis which this work subsequently tested experimentally.

Vocal Fold Kinematics in Individuals with Vocal Hyperfunction

EXPERIMENTAL METHODS

Participants were 10 adult female volunteers diagnosed with MTD prior to any therapeutic intervention (mean age = 23.9 years, SD = 5.4 years), 10 adult female volunteers diagnosed with bilateral vocal nodules prior to any therapeutic intervention (mean age = 25.8 years, SD = 9.4 years), and 10 females with healthy normal voice (mean age = 24.8 years, SD = 4.0 years). All participants were female due to the prevalence of vocal nodules in the female population compared to the male population (M. D. Morrison, et al., 1986; M. D. Morrison, et al., 1983; Pontes, et al., 2002; Titze, 1989). No participants with MTD or nodules had a history of any other speech or language disorder. The individuals with healthy normal voice reported with no complaints related to their voice, and had no abnormal pathology of the larynx as observed during flexible endoscopy with stroboscopy.

Participants were examined using trans-nasal (flexible) endoscopy in order to visualize the vocal folds. Trans-nasal endoscopy was chosen rather than rigid endoscopy to provide the best view of normal laryngeal function, and to avoid the biomechanical effects of tongue protrusion created by rigid endoscopy. The endoscopy procedure involved the insertion of a flexible endoscope through the nose and past the soft palate to the back of the throat, and in many cases also involved local anesthetization of the nasal passageways on one side with a light topical anesthetic (1% lidocaine) and a decongestant (0.5% neosynephrine). The procedure was digitally recorded via distal chip flexible endoscopy at 30 frames per second. During the exam, the participants produced 3 – 4 trials of the "sniff – eee" (between 5 – 6 separate gestures) at both "medium' and "fast" rates. Gesture rate was cued for participants using a metronome with visual (LED) cueing for the participant. Specifically, the medium rate was performed at 72 gestures / minute, and the fast rate at 104 gestures / minute. These speeds were chosen based on the typical rate used in the MGH Voice Clinic currently (medium) and a reasonable (easily reproducible) excursion from this value for the fast rate. All participants were able to produce the gestures at both rates. Production of the sniff-eee was modeled by the first author for each participant prior to endoscopy and for some participants during endoscopy as well. Participants were encouraged to practice the sniff-eee several times prior to endoscopy.

Video data were analyzed using the custom MATLAB software employed in Dailey et al. (2005). The glottic angle was marked on each frame of the exam video recording and an asymmetric sigmoid was fit to each single abduction or adduction movement set of angles. Due to the possible vertical translation of the endoscope during recording, angular rather than linear kinematics were used since they are more robust to changes in

vantage point. Marking of glottic angles was performed by two individuals. In order to assess inter- and intra-rater reliability, approximately 5% of samples were independently analyzed by both researchers yielding inter-rater reliability as measured with Pearson's R of 0.990, and intra-rater reliabilities of 0.995 and 0.986 for each researcher. The sigmoidal rather than the cubic polynomial fit used by Dailey et al. (2005) was used because it produced better fits to the kinematic data, which show two asymptotes in time. Figure 3-2 shows an illustration of a set of adduction angles with their fit, along with the specific kinematic features extracted for use in parameter calculation. Using the sigmoidal fit, the 20%-80% angular velocity of each abduction and adduction was calculated as well as the stiffness parameter (the ratio for the maximum angular adduction velocity to the extent of the motion). Tests on the null hypothesis that the difference between increases in adduction and abduction velocities and the changes in the "stiffness parameter" with increased gesture rate would be of the same level in the MTD and control groups were performed with non-parametric Mann-Whitney tests using Minitab® Statistical Software (Minitab Inc., State College, PA).

EXPERIMENTAL RESULTS

Boxplots of the changes in the "stiffness parameter" with increased gesture rate (fast versus medium) are shown in panel A of Figure 3-3 for individuals with MTD and controls. Also shown are the change values for individuals with vocal nodules. The changes in the stiffness parameter in these individuals presented with a marked bimodal distribution. For this reason, these individuals were not analyzed as a single group, but were examined case-by-case. Due to the relatively small sample sizes in the groups and the non-normal distributions, differences in the distributions of stiffness parameter changes between the MTD and control groups were examined with non-parametric testing. Median changes in the stiffness parameter in the MTD and control groups were 1.0 and 2.6, respectively. The distributions in the two groups differed significantly, as evaluated by the Mann-Whitney test (one-tailed, N1 = N2 = 10, $p = 0.03$).

Differences in the increase in 20%-80% velocity in adductory versus abductory motion between the medium and fast gesture rate were examined similarly. Boxplots of the differences between the adductory and abductory percent increase with increased gesture rate are shown in panel B of Figure 3-3 for the MTD and control groups, with values shown for individuals with vocal nodules. Although less marked than with the stiffness parameter, the differences in velocity increase in individuals with nodules presented with a somewhat bimodal distribution, or at least a significant outlier. For this reason, individuals with vocal nodules were not analyzed as a single group, but were examined on a case-basis. Median differences between the adductory and abductory 20%-80% velocity percent increase with increased gesture rate in the MTD and control groups were -8.9% and 11.3%, respectively. The distributions in the two groups differed significantly, as evaluated by the Mann-Whitney test (one-tailed, N1 = N2 = 10, $p = 0.002$). The average values of abductory and adductory 20%-80% velocity are tabulated in Table 3-1.

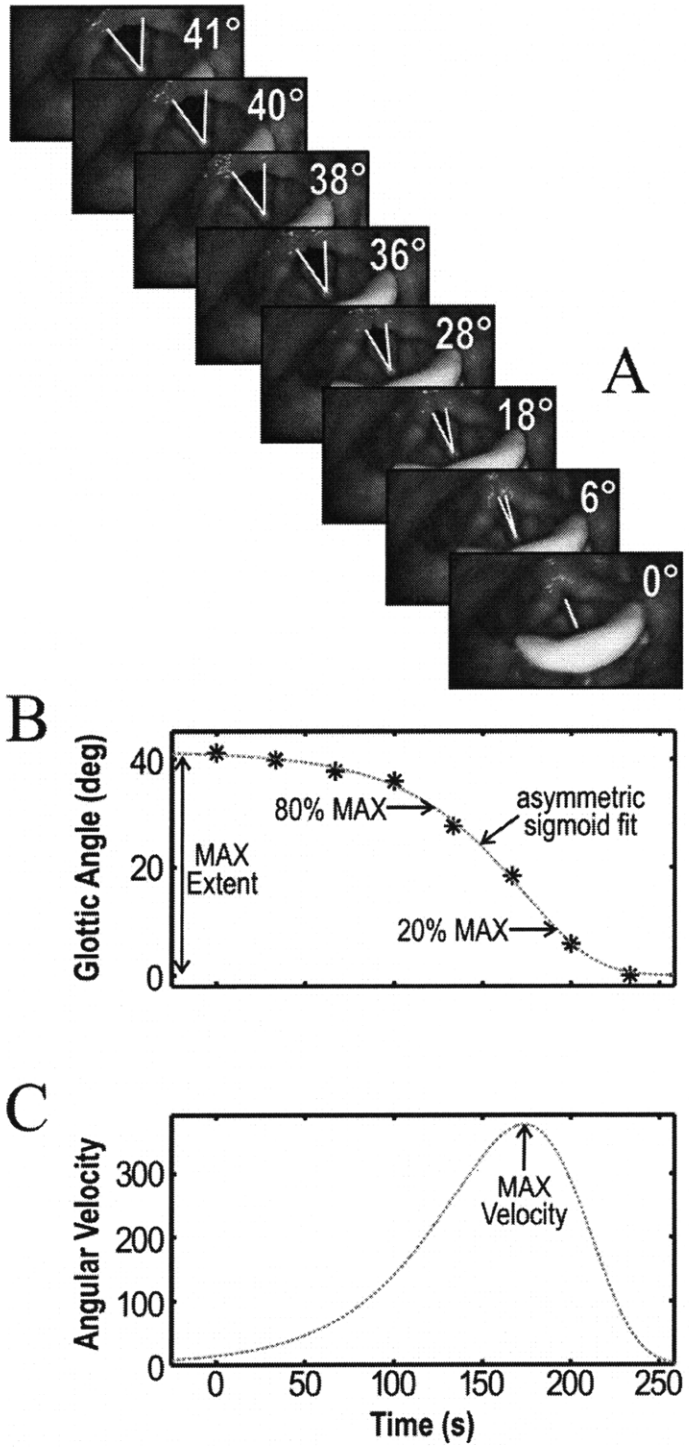


Figure 3-2. Overview of the experimental methodology. Panel A. An example of the images associated with one adductory movement with marked angles. Panel B. An example of the asymmetric sigmoid fit to the angles marked in one adductory movement. The 20%, 80%, and maximum angle extents of the sigmoidal fit are marked. Panel C. An example of the angular velocity of one asymmetric sigmoidal fit. The maximum velocity used for calculation of the stiffness parameter is noted.

Figure 3-4 shows a scatter plot of the differences in adductory and abductory 20%-80% velocity percent increase with faster gesture rate and the changes in the "stiffness parameter" with faster gesture rate. The two kinematic measures were found to be statistically significantly correlated, as assessed by Pearson's R ($R = 0.745$, $p < 0.001$).

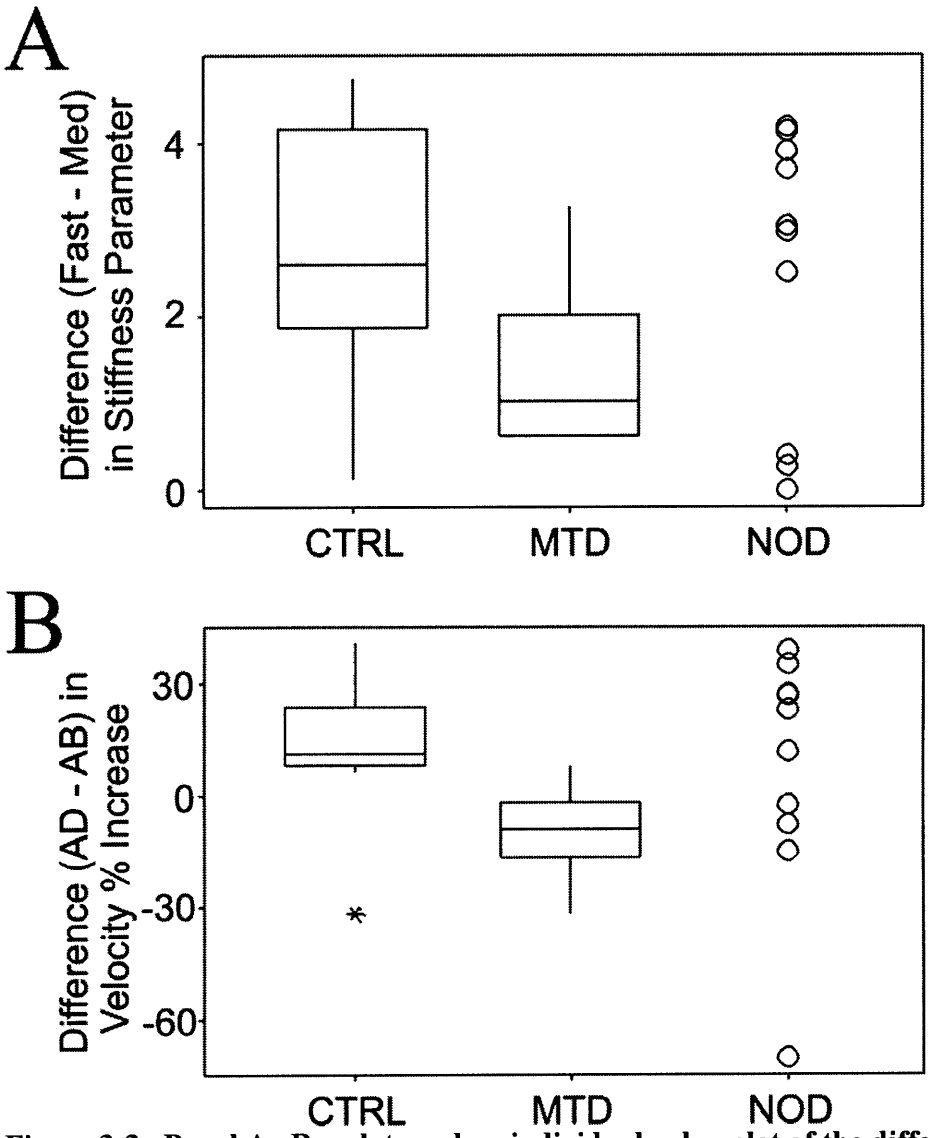


Figure 3-3. Panel A. Boxplots and an individual value plot of the differences in the stiffness parameter between the medium and fast gesture rate conditions. CTRL = individuals with healthy normal voice, MTD = individuals with MTD, NOD = individuals with vocal fold nodules. Panel B. Boxplots and an individual value plot of the differences between medium and fast gesture rate 20%-80% abduction and adduction velocities.

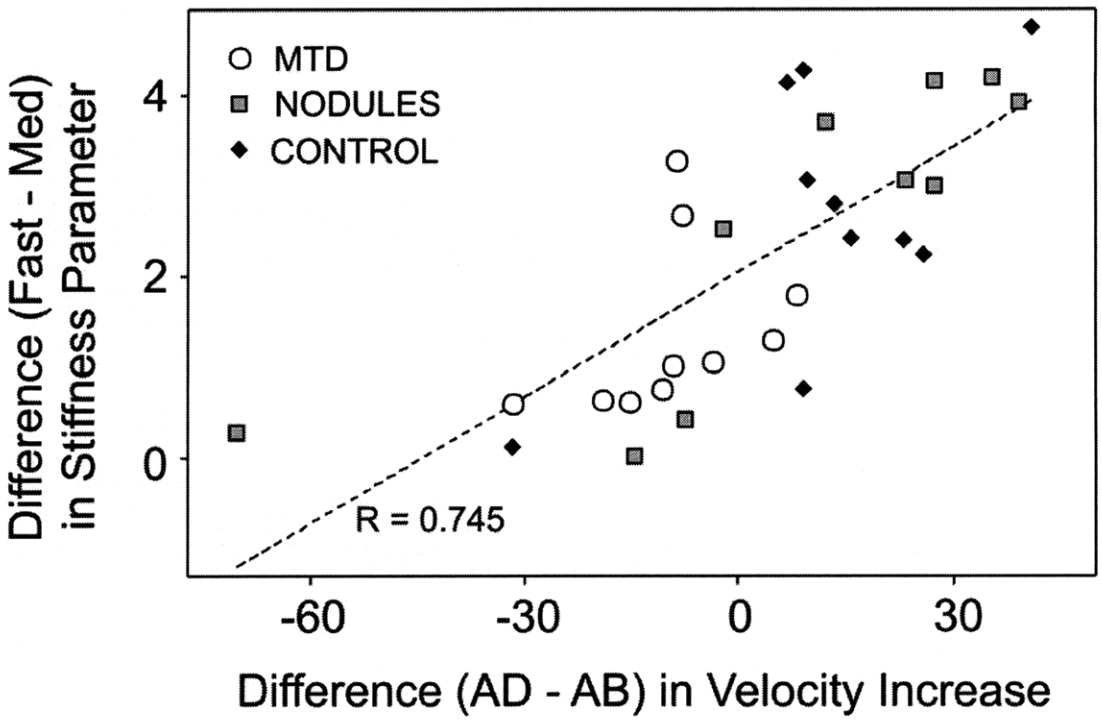


Figure 3-4. Differences in the stiffness parameter between the medium and fast gesture rate conditions are plotted with respect to the differences between medium and fast gesture rate 20%-80% abduction and adduction velocities.

Discussion

Average Abduction and Adduction Velocities in Individuals with Healthy Normal Voice

The mean 20%-80% abduction and adduction velocities and resulting percent increase between the medium and fast conditions showed similar qualitative results to those of Dailey et al. (2005; see Table 3-1). The percent increase of the adduction velocity was greater than the percent increase in abduction velocity. However, when compared to the results of Dailey et al. (2005), the differences between abduction and adduction were less striking. Some differences in methodology could explain the differences between the current results and those of Dailey et al. (2005). The medium and fast rates used here (72 rpm and 104 rpm) were precisely controlled for using a metronome, whereas the rates used by Dailey et al. (2005) were likely more varied and may have been, on average, produced at significantly different rates. Another methodological difference that could affect these results is the choice of function used for data fitting. Data fitting is necessary to estimate the kinematic parameters due to the sparse sampling rate of the videoendoscopy, but basing kinematic measures on a function fit to data rather than the raw data is a possible source of estimation error. While Dailey et al. (2005) used a cubic polynomial to fit their data, here a sigmoidal function was selected. Cubic polynomials have a maximum of two extremas and a single inflection point, which is consistent with vocal fold abduction and adduction; they also have a maximum of three roots, and do not contain horizontal asymptotes, causing curvature that is inconsistent with the raw kinematic data. Here, an asymmetric sigmoidal function was chosen to fit the data due to its characteristic two horizontal asymptotes and single inflection point. This difference in data fitting methodology could have contributed to the quantitative difference between the current work and that of Dailey et al. (2005).

Experimental Confirmation of the Modeling Hypothesis in Individuals with MTD

Individuals with MTD showed smaller changes with increases in gesture rate in both kinematic parameters than did controls. These results are consistent with the modeling hypothesis, and suggest that these individuals have elevated laryngeal stiffness, such that increases in gesture rate therefore have smaller effects on the laryngeal muscle stiffness. This work objectively substantiates claims that individuals with MTD have increased muscle tension, albeit indirectly.

The median changes seen experimentally in the stiffness parameter in the MTD and control groups were 1.0 and 2.6, which were roughly similar to the change seen in the stiffness parameter (1.4) between the medium and high stiffness conditions using the

model. However, stiffness parameter changes in controls were somewhat larger than those predicted by the model.

Vocal Fold Kinematics in Individuals with Nodules

In individuals with vocal fold nodules, the difference between the percent increase in the 20%-80% abduction and adduction velocities, and the change in the stiffness parameter from medium to fast gesture rates showed mixed results. Some individuals presented with kinematic measures consistent with those hypothesized for individuals with vocal hyperfunction, while others displayed values consistent with those hypothesized for controls. Although it is possible that this effect is due to estimation error in the kinematic measures caused by the presence of the lesion, a more likely possibility is that some individuals with vocal fold nodules present with vocal hyperfunction, and that others do not. Although organic developments on the vocal fold surface have been assumed to be related to hyperfunctional behavior or phonotrauma (Hillman, et al., 1990), there is still much unknown about the underlying mechanisms of vocal hyperfunction and its role in developing organic disorders (Hillman, et al., 1990). In some individuals, nodule formation may be the result of other factors such as excessive vocal use, laryngopharyngeal reflux, etc., rather than inappropriate muscle tension. Further, once nodules are present it is not possible to determine whether any signs of hyperfunction are related to the etiology of the nodules, or if these are primarily a phonatory reaction (straining) to the presence of the nodules. Progress in this area suffers from two shortcomings. First, there is the lack of a gold standard in vocal hyperfunction assessment with which to compare potential objective measures, such as kinematic parameters. Second, previous investigations have not been able to delineate the actual role of hyperfunction in the etiology of voice disorders like vocal nodules. Future work should collect the kinematic measures used in the present study with multiple available modes of assessment to attempt to ascertain correspondence of kinematic measures with other possible indicators of vocal hyperfunction in the population of vocal fold nodules. In parallel with the identification of better measures, there is also a need for studies that can correlate levels of vocal hyperfunction across time (longitudinal cross-sectional) with changes in vocal status (deterioration or improvement) to gain better insights into the role of hyperfunction in voice disorders.

Use of Vocal Fold Kinematics as a Measure of Vocal Hyperfunction

Both the difference between the percent increase in the 20%-80% abduction and adduction velocities and the change in the stiffness parameter from medium to fast gesture rates showed statistically significant differences between individuals with MTD and controls, but the velocity measure provided somewhat better discrimination between the two groups. The velocity measure and stiffness parameter changes were statistically significantly correlated, indicating that they may be measuring the same phenomenon. However, the modest Pearson's correlation ($R = 0.745$) suggests that there is likely noise in the measurement and estimation process.

Although the individuals with healthy normal voice showed the expected results as a group, some individuals presented with vocal fold kinematics that were inconsistent with the rest of the control group. This is not a completely unexpected result, given that past studies examining some indicators of vocal hyperfunction have indicated that individuals without voice disorder may display symptoms of vocal hyperfunction (Behrman, et al., 2003; Colton & Casper, 1996; Stager, Neubert, Miller, Regnell, & Bielamowicz, 2003). These studies have shown that some individuals without an expressed voice disorder showed visible compression during endoscopy (Behrman, et al., 2003; Stager, et al., 2003), as well as frequent hard glottal attack (i.e., Colton & Casper, 1996, pg 80), both of which are thought of as symptoms of vocal hyperfunction. It is possible that some of the individuals examined here who reported with healthy normal voice also engaged in vocal hyperfunction, without it leading to a voice disorder. Although this leads to less discrimination between groups and may make these kinematic measures less useful for diagnosis purposes, it does not necessarily rule out their use for follow-up and assessment of individuals with vocal hyperfunction throughout rehabilitation. A further possibility is that these measures could be used preventatively, to identify individuals engaging in vocal hyperfunction so that they may be able to avoid developing vocal pathology.

Another factor to consider with respect to the use of vocal fold kinematic measures in the assessment of vocal hyperfunction is the measurement technique of nasal endoscopy. This technique is usually well-tolerated, but can be a source of stress for many individuals. The stress of undergoing this procedure could temporarily induce vocal hyperfunction in individuals who otherwise have healthy normal voices, making it more difficult to differentiate between typically hyperfunctional individuals and those specifically reacting to the procedure. Alternatively, the stress of endoscopy could differentially elicit hyperfunctional behaviors in predisposed individuals, effectively exacerbating their symptoms. Several studies have suggested a role for hyper-reactivity to emotional stressors and anxiety in the etiology of MTD (Demmink-Geertman & Dejonckere, 2002, 2008; Goldman, et al., 1996; N. Roy, Bless, & Heisey, 2000), especially in women.

Eliciting symptoms by increasing task complexity is a technique that is often used in motor disorders, such as Parkinson's Disease (PD). Although PD is a common neurological disorder known to degrade movement ability, it can be difficult to objectively measure the kinematic differences between PD and controls during movement tasks, especially in early stages of the disease. However, performing two movements simultaneously can strikingly reduce PD patient performance, while having little effect on controls (Benecke, Rothwell, Dick, Day, & Marsden, 1986). Similarly, simultaneous performance of a cognitive task can increase PD tremor, also with little effect on controls (Sturman, et al., 2007), increasing the ability to ascertain treatment efficacy. It is possible that inducing stress in individuals with MTD may intensify symptoms of vocal hyperfunction, without causing similar effects in healthy controls. Well-controlled studies of individuals with MTD and healthy normal voice are needed to validate this hypothesis.

As described in the methods, local anesthetization of the nasal passageways on one side with 1% lidocaine was performed on most participants, and some portion of the anesthetic dripped back into the laryngopharyngeal area. It is unlikely that this had a

significant effect on vocal fold kinematics, given that recent work has found that application of topical anesthetic does not perceptibly affect vocal fold motion (Rubin, Shah, Moyer, & Johns, 2009). Furthermore, the lidocaine was administered to all participant groups. However, Dworkin et al. (2000) used a 3 cc injection of 4% lidocaine through the cricothyroid membrane to treat three therapy-resistant cases of MTD, finding that the application caused immediate and long-standing resolution of symptoms. Although the dosage used there was quite different than in the present work, it is possible that the use of the lidocaine temporarily reduced symptoms, leading to smaller differences between the MTD and control groups. This possible effect is difficult to study due to the large percentage of individuals unwilling to tolerate nasal endoscopy without use of anesthesia.

Summary and Future Work

A simple, one degree of freedom, virtual trajectory model of vocal fold kinematics was used to confirm that vocal fold movement during a repetitive abduction/adduction gesture suggested excessive muscle tension in individuals with MTD relative to healthy normal controls. The model simulations verified that increases in the modeled muscle stiffnesses were correlated with changes in a kinematic parameter known to increase with gesture rate and changes in a kinematic parameter known to increase with hard glottal attack. These modeling results suggested that increases in gesture rate would affect kinematic features to a smaller degree in patients with vocal hyperfunction because of the presence in these individuals of increased tension in intrinsic laryngeal muscles and structures during typical phonation. This hypothesis was tested experimentally, finding that kinematic measures in individuals with MTD showed less change with increased gesture rate, consistent with the hypothesis that these individuals have elevated laryngeal muscle tension during phonation. However, kinematic results in individuals with vocal fold nodules were mixed. The findings support the potential use of vocal fold kinematic data as an objective clinical assay of vocal hyperfunction. Suggested future work includes correlation of vocal fold kinematic data in individuals with vocal hyperfunction with other modes of vocal hyperfunction assessment, as well as exploring the role of stress inducement in exacerbating the known symptoms of vocal hyperfunction.

Chapter 4: Comparison of Neck Tension Palpation Rating Systems with Surface Electromyographic and Acoustic Measures in Vocal Hyperfunction

Objectives/Hypothesis: **The purpose of this study was to evaluate current neck tension palpation rating systems to determine inter-rater reliability and possible correlation with neck surface electromyography (sEMG, collected from three electrode recording locations) and measures of the third formant for /a/ during various vocal behaviors.**

Study Design: **This prospective study examined the neck muscle tension of 16 participants before and after a single session of voice therapy.**

Methods: **Inter-rater reliability and relationships between palpation ratings and objective measures of sEMG (anterior neck) and the third formant for /a/ were assessed using Pearson's correlations (R).**

Results: **Inter-rater reliability was relatively low as measured by Pearson's correlations, although Wilcoxon Signed Rank Test results were similar to those in a previous study. Correlations between palpation ratings and sEMG, and between ratings of laryngeal height and the third formant for /a/ were generally low. Correlations increased between anterior neck sEMG and ratings of suprahyoid muscle tension when examined in a reduced set of individuals with higher inter-rater reliability.**

Conclusions: **Palpation rating scales do not reliably capture changes that may occur in neck muscle tension of typical voice therapy patients over one session. Consequently, little can be concluded from correlations between sEMG and palpation ratings.**

Introduction

When individuals demonstrate increased intrinsic laryngeal muscle contraction (vocal hyperfunction), it is thought that they often simultaneously contract the extrinsic laryngeal muscles and other superficial neck muscles in a similar hyperfunctional manner (Aronson, 1980). Strap muscle tension can be assessed through both visual and tactile inputs. A study by Altman and colleagues (2005) reported on 150 patients who had been diagnosed with muscle tension dysphonia (MTD; a voice disorder with symptoms of vocal hyperfunction and no known structural change to the vocal fold or neurogenic disease of the larynx). Based on a speech pathology evaluation of these patients, 70% were found to have "obvious cervical neck tension visible" (Altman, et al., 2005). Practitioners have previously reported that observation of the inferior bellies of the omohyoid muscle crossing the supraclavicular fossae may show them to be tense and prominent during speech (M. Morrison, 1997b), while further information about the extent of muscle tension can be gained by palpation of the larynx at rest and during voicing (M. Morrison, 1997b). Excessive tension in disordered individuals has been noted via palpation over the major horns of the hyoid bone, over the superior cornu of the thyroid cartilage, along the anterior border of the sternocleidomastoid muscle, and throughout the suprahyoid musculature (N. Roy, et al., 1996).

While palpation of neck musculature is a routine clinical procedure in the assessment and management of vocal hyperfunction (Aronson, 1980; Lieberman, 1998; Nelson Roy & Bless, 1998; N. Roy, et al., 1997; N. Roy & Leeper, 1993), only a few standardized rating scales have been developed. As part of a surface electromyography (sEMG) study, one speech-language-pathologist rated "laryngeal-area tonicity" on a 1 - 5 linear scale, finding a high correlation between a single clinician's scores and mean sEMG during vowel production (1989). Angsuwarangsee and Morrison (2002) developed a linear 0 – 3 grading system of neck muscle tension based on the experiences and work of Lieberman (1998) for research use in which each muscle group is graded based on specific text descriptors (see Table 4-1). Kooijam *et al.* (2005) modified the system proposed by Angsuwarangsee and Morrison to include more muscle categories, as well as documentation about body posture. Mathieson *et al.* (2009) recently proposed a new rating system in which the muscle resistance of four categories is rated on a linear scale of 1 – 5 and laryngeal position is noted as being one of the following: high held, neural, lowered, or forced lowered (see Figure 4-1).

Angsuwarangsee and Morrison (2002) assessed their rating system on 57 successive voice patients, with two independent investigators (otolaryngologists) examining each patient. Inter-rater reliability numbers based on Wilcoxon Signed-Ranks Tests were presented, with the reliabilities presented in the form of *p*-values. Only one category, pharyngolaryngeal, exhibited statistically significant scores (less than 0.05), which was interpreted by the authors as having low inter-rater reliability. Mathieson *et al.* (2009) used palpatory evaluations in 10 individuals with MTD pre- and post- laryngeal manual

therapy. Inter-rater reliability was not noted, as more than one clinician was not used for evaluation. Further, it appears that the evaluator (a speech-language pathologist) was the same individual providing therapy.

Rating	Description
Suprahyoid muscles	
0	soft at rest, may slightly contract on phonation
1	soft at rest, mild low-pitch and moderate high-pitch contraction
2	some tension at rest, tense with jaw protrusion on phonation
3	tense all the time, maximally tight on phonation
Thyrohyoid muscles	
0	no muscular contraction at rest, mild on phonation
1	soft thyrohyoid space at rest, some contraction on phonation
2	tense, narrow thyrohyoid space at rest, moderate contraction on phonation
3	very tense with closed thyrohyoid space all the time
Cricothyroid muscles	
0	normal cricothyroid space and phonatory movement
1	narrowing of cricothyroid space at rest, some movement on phonation
2	anterior displacement of cricoid cartilage with narrowing of cricothyroid space at rest, closing of the space on phonation
3	closed cricothyroid space all the time
Pharyngolaryngeal muscles	
0	soft, easy to rotate the larynx for 90° and palpate posterior cricoarytenoid (PCA) muscle and arytenoids movement on sniffing
1	slightly tense, cannot palpate PCA muscle movement on sniffing
2	moderately tense, difficult to rotate the larynx but still can palpate the posterior edge of thyroid cartilage
3	very tense, cannot rotate the larynx at all

Table 4-1. Neck tension palpation system. Reprinted from Angsuwarangsee T, Morrison M. Extrinsic laryngeal muscular tension in patients with voice disorders. *Journal of Voice*. 2002; 16:333-343.

The work of Redenbaugh and Reich (1989) is the only study to explore the relationship between sEMG and neck palpation ratings. In their study, laryngeal-area tonicity was evaluated by a single speech-language pathologist during tidal breathing, production of the vowel /a/ for 15 seconds, and reading aloud. Laryngeal-area tonicity was rated using a 5-point, equal-appearing-interval scale. The Pearson's correlations between the palpation score and the sEMG during the vowel and speech tasks were found to be 0.86 and 0.9, respectively. No inter-rater reliability measures were attempted given that there was only one rater. The study examined seven individuals with MTD and seven individuals with healthy normal voice. Due to the bimodal nature of this sample, with the participants likely representing alternate ends of the spectrum of neck muscle tension

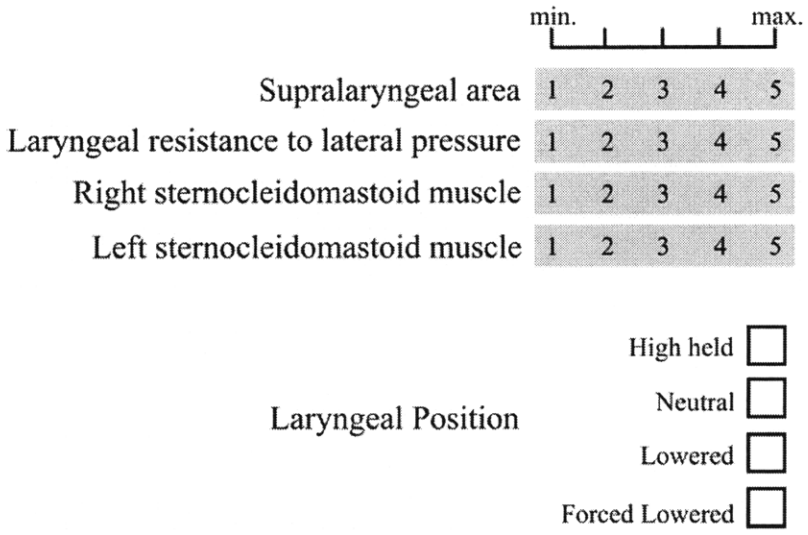


Figure 4-1. Palpation Rating Scale. Adapted from Mathieson L, Hirani SP, Epstein R, Baken RJ, Wood G, Rubin JS. Laryngeal Manual Therapy: A Preliminary Study to Examine its Treatment Effects in the Management of Muscle Tension Dysphonia. *Journal of Voice*. 2009; 23:353-366.

related to voice disorder, correlation values may possibly be inflated. Despite the fact that the palpation procedure and scale utilized by Redenbaugh and Reich (1989) was published in 1989, it has not been the subject of further published research, and to our knowledge is not widely used in the clinic. Further, this previous study utilized only one electrode position overlying the thyrohyoid membrane. In order to understand the relationships between sEMG and clinical ratings of palpation, it is necessary to determine among multiple electrode recording locations and vocal behaviors, those that may correlate most accurately with more widely used clinical ratings.

Roy and Ferguson (2001) examined changes in formant frequencies pre-therapy versus post-therapy in 75 participants with functional dysphonia, finding significant decreases in the first, second, and third formants after therapy. The authors interpreted this finding as evidence of laryngeal lowering as a result of therapeutic intervention. The neck palpation rating system proposed by Mathieson *et al.* (2009) requires the evaluator to note laryngeal position of the participant as being high held, neural, lowered, or forced lowered using a nominal scale. Their study applied this system to 10 participants and found insignificant changes post-therapy in average laryngeal height. Acoustic analysis at both time points found a trend of increased second formant during vowel production post-therapy, which would be inconsistent with laryngeal lowering. Their study did not attempt to correlate changes in perceived larynx height with formant changes.

The third formant shows less variation across different vowel productions than the second formant, which is more likely to be affected by changes in vowel articulation. Moreover, recent work has shown that treatment for MTD can also affect articulation, leading to increased vowel space (N. Roy, Nissen, Dromey, & Sapir, 2009). The third formant should be more correlated with vocal tract length, such that changes to the third formant may offer objective confirmation of changes in laryngeal position, especially due to short-term therapy effects. Assessing the relationship between judgments of laryngeal height and corresponding changes in the third formant may offer more useful information about the utility of this clinical scale for assessing laryngeal position.

Clinically, the presence of excessive neck tension is noted as a sign of vocal hyperfunction, informing both diagnosis and treatment (Angsuwarangsee & Morrison, 2002; Kooijman, et al., 2005; Lieberman, 1998; Mathieson, et al., 2009; Nelson Roy & Bless, 1998; N. Roy, et al., 1997; N. Roy, et al., 1996; N. Roy & Leeper, 1993). However, current methods of assessment of neck muscle tension (e.g., Angsuwarangsee & Morrison, 2002; Mathieson, et al., 2009) depend on tactile measures, which are subjective and lack a large dynamic range of measurement. The use of sEMG and objective acoustic methods to monitor changes in neck tension and/or laryngeal position in patients with voice disorders could lead to more standardized care, as well as improved information about patient progress. It is currently still unknown whether neck sEMG recordings or formant changes correlate well with clinical palpation-based ratings. Also, in order to use sEMG optimally, it necessary to determine the electrode recording locations and vocal behaviors that correlate most accurately with clinical ratings. The purpose of this study was to evaluate the neck tension palpation tension rating systems of Angsuwarangsee and Morrison (2002) and Mathieson *et al.* (2009) to determine whether reproducible results could be obtained, as measured by inter-rater reliability measures (Pearson's correlations), when administered by speech-language pathologists previously unfamiliar with these systems. A further goal of this study was to ascertain whether the systems were correlated with objective measures of neck tension (sEMG) and laryngeal height (third formant for /a/) of individuals receiving therapy for voice disorders related to vocal hyperfunction. These two scales were used as a comparison with acoustic changes in the third formant and neck sEMG collected from multiple electrode recording locations during various vocal behaviors to understand how differences in scale structure may affect correlations with objective measures.

Methods

PARTICIPANTS

Participants were 16 adult volunteers (13 females, 3 males) with mean age of 24.9 years (R=18-41 years) receiving voice therapy due to a voice disorder related to vocal hyperfunction (e.g., muscle tension dysphonia, vocal nodules). Table 4-2 lists the diagnoses of the participants, as well as age and sex. Participants were varied in their progress in voice therapy, with their research participation taking place during one of multiple visits in the course of their therapy.

Participant	Age	Sex	Diagnosis
P1	31	M	muscle tension dysphonia
P2	32	F	vocal fold nodules
P3	22	F	muscle tension dysphonia
P4	22	F	vocal fold nodules
P5	18	F	muscle tension dysphonia
P6	26	M	muscle tension dysphonia
P7	27	F	muscle tension dysphonia
P8	19	F	muscle tension dysphonia
P9	22	F	muscle tension dysphonia
P10	24	F	muscle tension dysphonia
P11	21	F	vocal fold nodules
P12	41	M	muscle tension dysphonia
P13	20	F	muscle tension dysphonia
P14	22	M	vocal fold nodules
P15	26	F	vocal fold nodules
P16	25	F	muscle tension dysphonia

Table 4-2. Participant diagnosis and demographic information.

CLINICAL PALPATION METHODOLOGY

Two of three total participating certified speech-language pathologists assessed each participant before and after therapy using the two clinical palpation ratings of Angsuwarangsee and Morrison (2002) and Mathieson *et al.* (2009). The 'primary' rater was the same clinician providing therapy to the participant. A second rater was another speech-language pathologist who was unfamiliar with the patient.

A total of three certified speech-language pathologists who specialize in voice participated in the clinical assessment portion of this study. All three of the speech-language pathologists who participated in this study completed their clinical fellowship training in a specialized voice clinic, and all had at least one year of experience working full-time in a specialized voice clinic, with case loads consisting exclusively of patients with voice disorders. All of them had extensive experience with laryngeal palpation and manipulation as a part of clinical practice prior to the initiation of this study. Participation among the three speech-language pathologists was approximately equal, with each completing pre-therapy and post-therapy assessments for between 8 and 12 participants. The speech-language pathologists were trained internally by reading the primary literature behind the rating systems (Angsuwarangsee & Morrison, 2002; Mathieson, et al., 2009) as well as the chapter on techniques of manual therapy (Lieberman, 1998), and then each applying the two neck tension ratings systems to the same individual, comparing rating decisions, and discussing scoring issues at length. This internal training lasted approximately 1.5 hours. After official recruitment and recording of participants had been initiated, no feedback was given to participating clinicians regarding their agreement with one another.

sEMG and Acoustic Recording Methodology

The sEMG and acoustic recordings consisted of a brief vocal assessment of the participant, which included three trials of the vowel /a/, read speech (The Rainbow Passage; Fairbanks, 1960), and spontaneous running speech. Spontaneous speech was elicited in response to the investigator asking the participant a probing question (e.g., "Can you tell me what you do in a typical therapy session?"). After completion of these speech tasks, maximal voluntary contraction (MVC) maneuvers were performed. These consisted of asking the participants to perform neck contraction against manual resistance for the purpose of normalizing sEMG data (see following Data Analysis section). In order to ensure that systematic differences did not exist in the MVC force production produced in the pre-therapy and post-therapy recordings, a dynamometer (Chatillon DPP-50, Ametek, Inc., Paoli, PA) was used during neck muscle contraction against manual resistance for all but three participants, and the maximal force was recorded. The MVC forces ranged from 14 – 42 lb_f by participant, but there was not a statistically significant difference between pre-therapy and post-therapy MVC forces (Paired Student's *t*-test, df = 12, $p = 0.85$).

Simultaneous neck sEMG and acoustic signals from a lavalier microphone (Sennheiser MKE2-P-K, Wedemark, Germany) were recorded digitally with Delsys™ (Boston, Massachusetts) hardware (Bagnoli Desktop System) and software (EMGworks 3.3) at 20 kHz. The sEMG signals in this study were recorded and analyzed in view of current European standards (Hermens, et al., 1999). Participants' necks were prepared for electrode placement by cleaning the neck surface with an alcohol pad and "peeling" with tape to reduce electrode-skin impedance, noise, DC voltages, and motion artifacts. The neck sEMG was recorded using two 2-channel Bagnoli systems (Delsys ™ Inc, Boston, Massachusetts) with three Delsys ™ 3.1 double differential surface electrodes placed parallel to the underlying muscle fibers of the 1) thyrohyoid, omohyoid, and sternohyoid

muscles, 2) cricothyroid and sternohyoid muscles, and 3) sternocleidomastoid muscle (see Figure 4-2). The Delsys ™ 3.1 double differential surface electrodes consisted of three 10-mm silver bars with inter-electrode distances of 10-mm. Double differential electrodes were chosen to increase spatial specificity of the sEMG recordings and to eliminate the possibility of electrical crosstalk, a risk given the electrode proximity.

Electrode 1 was centered about 1 cm lateral to the neck midline, as far superior as was possible without impeding jaw opening of the participant. Electrode 2 was centered on the gap between the cricoid and thyroid cartilages of the larynx, and centered at 1 cm lateral to the midline, contralateral to Electrode 1. Electrode 3 was centered one-third of the distance from the sternal notch of each participant to his or her mastoid process following the recommendations of Falla *et al.* (2002) A ground electrode was placed on the superior aspect of the participant's left shoulder. The sEMG signals were pre-amplified and filtered using Delsys™ Bagnoli systems set to a gain of 1000 with a band-pass filter (roll-off frequencies of 20 Hz and 450 Hz).

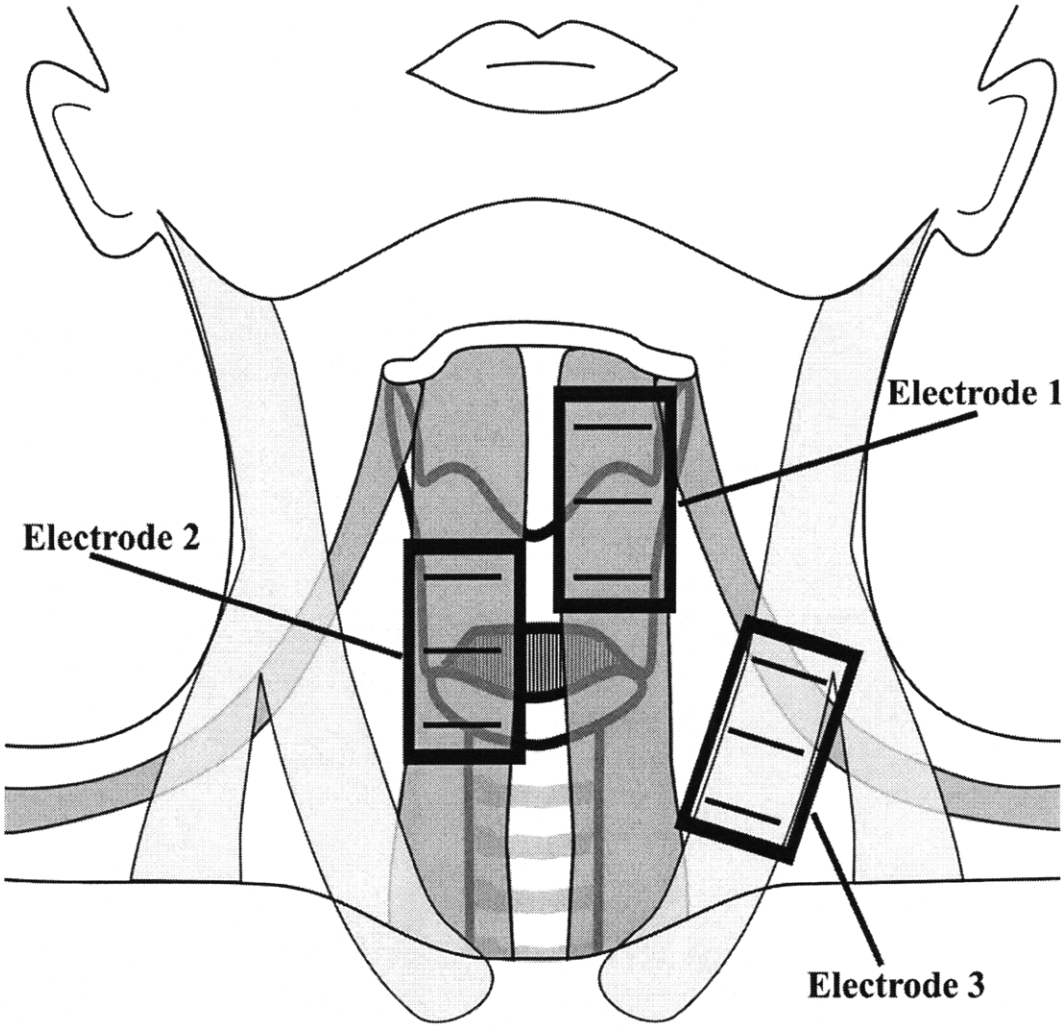


Figure 4-2. Schematic of sEMG electrode recording locations.

DATA ANALYSIS

So that sEMG data gathered could be compared between pre-therapy and post-therapy recordings, the variability associated with differences in neck surface electrode contact and placement was minimized by normalizing the sEMG to a reference contraction at MVC. All sEMG data were computed as the root-mean-squared (RMS) and then normalized via MVC (in RMS) in windows of 1 s using custom software written in MATLAB® (Mathworks Inc., Natick, MA). While studies have shown that for simple, one-joint systems, sub-maximal contractions are more reliable for normalization (Allison, et al., 1993; J. F. Yang & Winter, 1983), it has been shown that the MVC references is more reliable for anterior neck musculature (Netto & Burnett, 2006). Consequently, all sEMG data were analyzed in terms of % MVC. The third formant during three trials of the vowel /a/ was estimated using the linear predictive coding analysis in Praat acoustic analysis software (Boersma & Weenink, 2008). All of the formants found were consistent with the expected ranges specified in the literature (e.g., Stevens, 2000).

Correlations were calculated between the normalized RMS sEMG and clinical ratings of various muscle groups to ascertain the level of association between the assorted measures. Inter-rater reliability measures were calculated with Pearson's correlation for most elements of the two clinical rating systems using the assessment of the two speech-language pathologists. To compare these data with previous reports of inter-rater reliability, Wilcoxon Signed-Ranks Tests were also performed between raters. Inter-rater reliability of the larynx position measure of the Mathieson *et al.* (2009) palpation system was assessed using Cohen's Kappa due to the nominal nature of the scale. A two-factor ANOVA was used to examine the effect of rater and perceived larynx height change (the larynx position measure of the Mathieson *et al.* (2009) palpation system) on the measured changes in the third formant for the /a/ vowel. Statistical analysis was performed using Minitab® Statistical Software (Minitab Inc., State College, PA).

Results

INTER-RATER RELIABILITY

Inter-rater reliability between the two raters of neck tension using all pre-therapy and post-therapy judgments was assessed using Pearson's correlations for all categories of the Angsuwarangsee & Morrison system, and for the first four categories of the Mathieson *et al.* system. Pearson's correlations were generally poor, but differed slightly as a function of muscle group. For comparison with the work of Angsuwarangsee & Morrison (2002), Wilcoxon Signed-Ranks tests were also performed on rater judgments. The Pearson's correlations and *p*-values from the Wilcoxon Signed-Ranks Tests are shown in Figure 4-3.

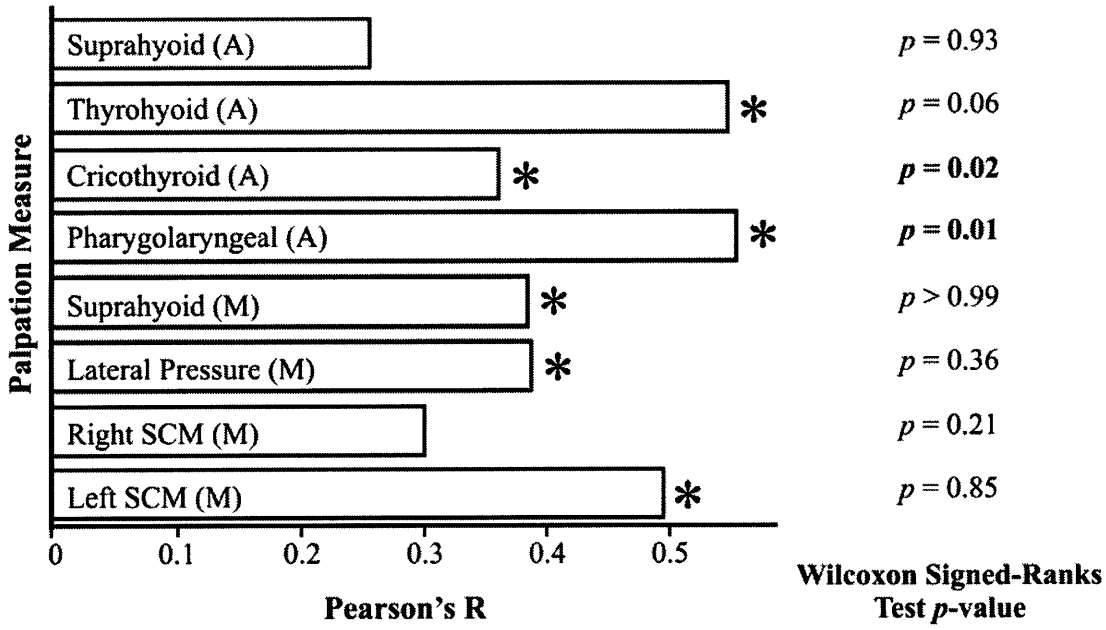


Figure 4-3. Inter-rater reliability for pre-therapy and post-therapy palpation. Palpation measures marked with an (A) are part of the Angsuwarangsee & Morrison system (2002); those marked with an (M) are part of the Mathieson *et al.* system (2009). Asterisks note those measures for which the Pearson's correlation was significantly ($p < 0.05$) greater than 0.

None of the Pearson's correlations were greater than 0.6, with the lowest at 0.23. The Wilcoxon Signed-Ranks showed no significant difference between raters for most

categories, excepting the cricothyroid and pharyngolaryngeal measures of the Angsuwarangsee & Morrison system.

When judging laryngeal position, no raters used the designations for "Lowered" or "Forced Lowered", essentially creating a binary rating system of "High held" or "Neutral." Of the 32 assessments of laryngeal position, a total of 22 matched perfectly (69%). Cohen's Kappa was calculated for each response ("High held" and "Neutral"), equaling 0.38 for both.

Inter-rater reliability between the two raters of neck tension using the change between pre- and post-therapy judgments was also assessed. The Pearson's correlations and *p*-values from the Wilcoxon Signed-Ranks Tests are shown in Figure 4-4. Several of the Pearson's correlations were near zero or even negative, although the left SCM of the Mathieson *et al.* system had a Pearson's correlation greater than 0.6. The Wilcoxon Signed-Ranks showed no significant difference between raters for any category. Of the 16 assessments of laryngeal position change, a total of 10 matched perfectly (63%). Cohen's Kappa was calculated for each response, equaling 0.02 for both.

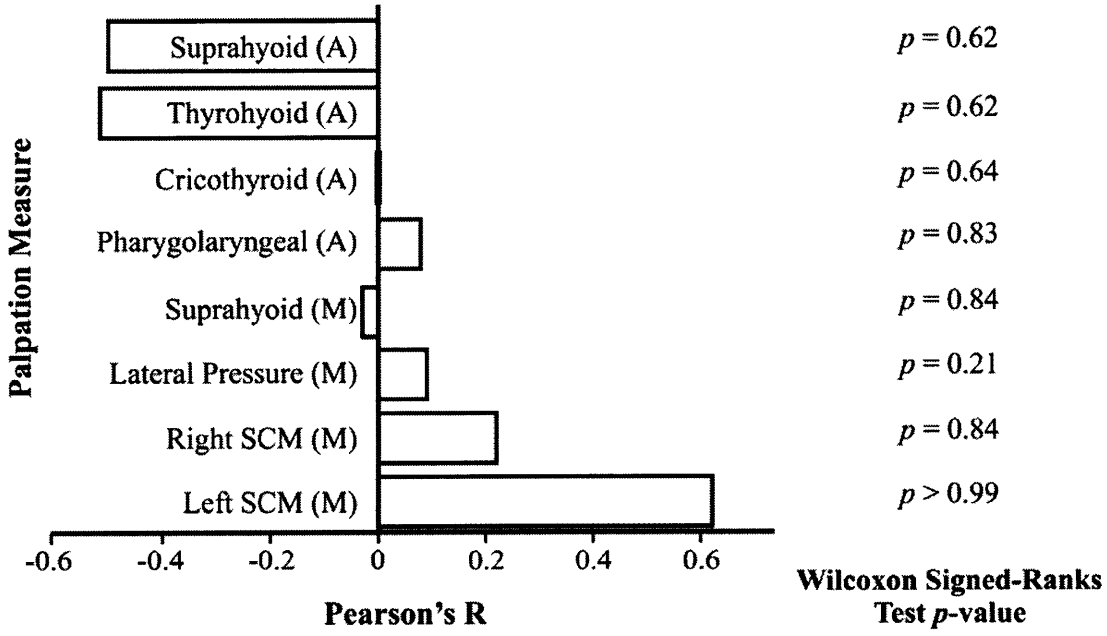


Figure 4-4. Inter-rater reliability for the change between pre-therapy and post-therapy judgments. Palpation measures marked with an (A) are part of the Angsuwarangsee & Morrison system (2002); those marked with an (M) are part of the Mathieson *et al.* system (2009).

CORRELATION BETWEEN PALPATION RATINGS AND SEMG

The left panel of Figure 4-5 shows the Pearson's correlations between each palpation measure and sEMG from all possibly relevant electrode locations during rest, read

speech, and spontaneous speech. The pharyngolaryngeal measure was not included in the correlation analysis since there were no appropriate electrode locations. The sEMG from electrode positions 1 and 2 were compared with suprahyoid, thyrohyoid, and cricothyroid ratings from the Angsuwarangsee & Morrison system, and the supralaryngeal and lateral pressure ratings from the Mathieson *et al.* system. The sEMG from electrode position 3 was compared with both left and right SCM ratings, despite the fact that sEMG was collected only from the patient's left SCM.

To reduce the effects of poor inter-rater reliability on correlations between sEMG and palpation ratings, participants whose pre- and post change differed between rater by 2 or more scale points on any dimension were excluded, resulting in a reduced set of N = 8 "high reliability" participants. The right panel of Figure 4-5 shows the Pearson's correlations for the reduced set.

Relationship between Perceived Laryngeal Height and the Third Formant

Laryngeal height (the larynx position measure of the Mathieson *et al.* (2009) palpation system) was most frequently rated as the same in both pre-therapy and post therapy recordings. In some cases, one or both raters felt that a participant moved from "high held" to "neutral" during the course of therapy. Changes in the third formant averaged at 1 Hz, ranging from -164 Hz (indicating a lower larynx post-therapy) to 281 Hz (indicating a higher larynx post-therapy). These changes did not appear to be associated with perceived laryngeal height. A two-factor ANOVA assessing the effect of rater and perceived larynx height change on the measured changes in the third formant showed no effect of either variable ($p > 0.05$).

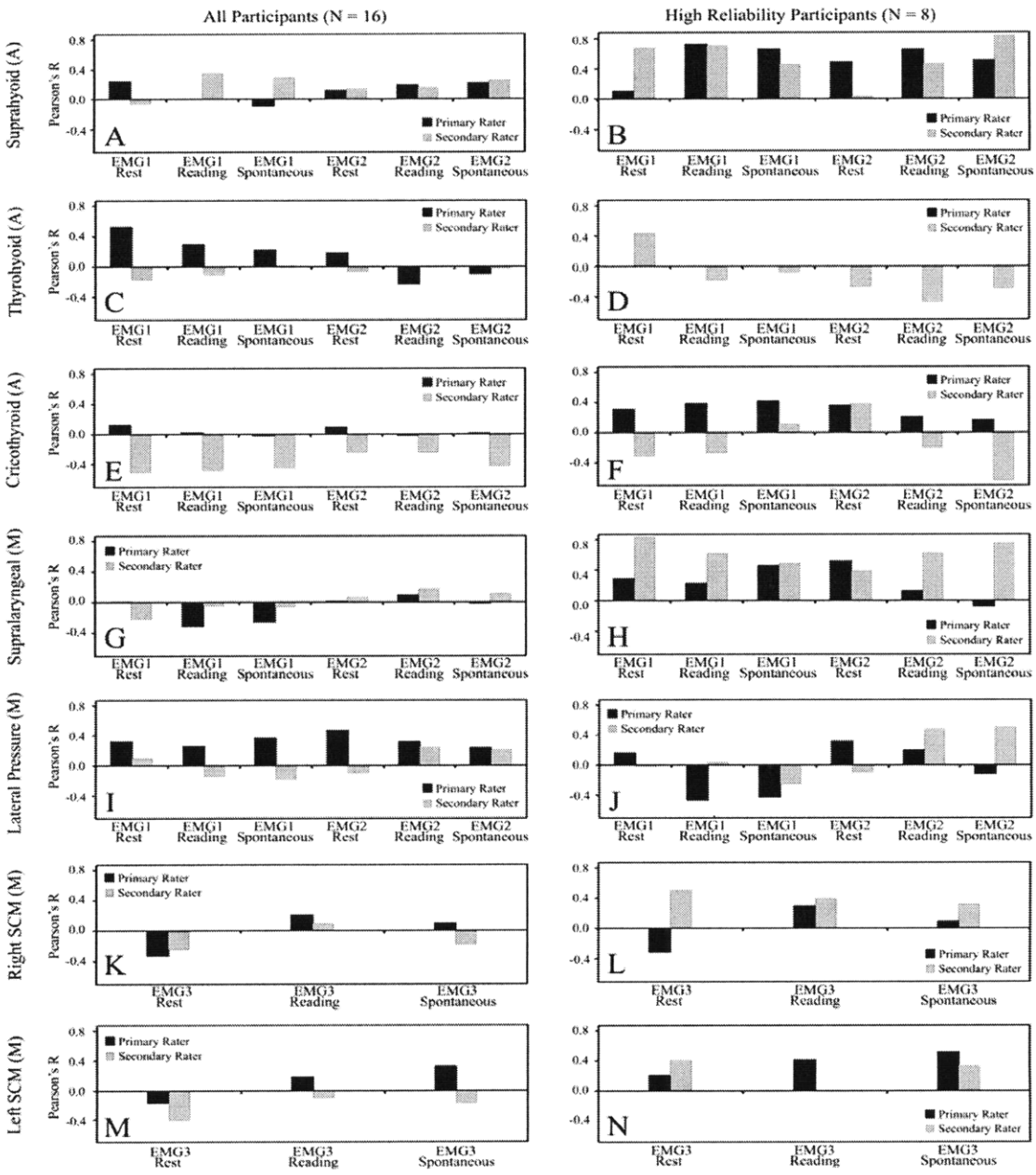


Figure 4-5. Pearson's correlations between palpation measures and sEMG. Palpation measures marked with an (A) are part of the Angsuwarangsee & Morrison system (2002); those marked with an (M) are part of the Mathieson *et al.* system (2009). The left panel (A, C, E, G, I, K, M) is for the entire set of participants (N = 16); the right panel (B, D, F, H, J, L, N) is for the reduced set of "high reliability participants" (N = 8).

Discussion

INTER-RATER RELIABILITY

Inter-rater reliability based on single time-point assessments as measured with Pearson's correlation was generally low across all dimensions of both scales, and did not improve with the use of pre- and post-therapy differenced data. The highest reliabilities were seen for the thyrohyoid and pharyngolaryngeal assessments of the Angsuwarangsee and Morrison system and the left SCM assessment of the Mathieson *et al.* system. No systematic differences in the inter-rater reliability emerged between the two systems. The difference in reliability between the right (R = 0.30) and left (R = 0.49) SCM assessments is puzzling given that clinicians tended to use both hands during both SCM assessments. One possibility is that patient asymmetries could perhaps have affected the variability of palpable muscle tension, leading to reduced inter-rater reliability, but there is no evidence here to support overall right-left asymmetry.

Angsuwarangsee and Morrison (2002) used Wilcoxon Signed-Ranks Tests as a measure of inter-rater reliability, finding the only significant differences ($p < 0.05$) in judgments for the pharyngolaryngeal assessment (interpreted by them as poor reliability). Similarly, we also found p-values greater than 0.05 for all assessments excepting the cricothyroid and pharyngolaryngeal assessments of the Angsuwarangsee and Morrison system, indicating that the rater performance was not dissimilar than those from that study. However, the relationship between Pearson's correlation values and the p-values resulting from the Wilcoxon Signed-Ranks testing calls into question the appropriateness of using the Wilcoxon Signed-Ranks test as a measure of inter-rater reliability. The Wilcoxon Signed-Ranks test assesses the likelihood of mean differences between measures being non-zero, not reliability. Greater overall variance of two judges (lack of agreement) would therefore increase p-values, whereas they could lead to lowered Pearson's correlations, and vice-versa. Raters who are highly unreliable but do not consistently agree in the direction of their disagreement would have a high p-value due to the large variance in their differences, but a low Pearson's correlation. As an example, in the inter-rater reliability data shown in Figure 4-3, the pharyngolaryngeal measure has both the highest Pearson's correlations (indicative of high reliability) and the smallest p-values (indicative of a non-zero difference between raters). This throws into doubt the high inter-rater reliability reported by Angsuwarangsee and Morrison (2002), since the interpretation was based on the use of Wilcoxon Signed-Ranks Tests, and no Pearson's correlations were reported.

The nominal scale used to assess larynx position in the Mathieson *et al.* system showed moderately low values of Cohen's kappa, with non-significant p-values to assess the likelihood of kappa > 0. Kappa values range from -1 to 1 where a kappa of 1 indicates perfect agreement between raters, and a kappa of 0 indicates agreement the same as that expected by chance. The results of kappa analysis indicate that the inter-rater agreement

of laryngeal height is not significantly higher than that which would be due to chance. One possible factor in this lack of agreement is the prevalence of different internal definitions of laryngeal height: some clinicians may associate a high larynx position with merely a high hyoid, whereas others might require the entire larynx to be raised. Regardless, the low values of kappa indicate that these scales to not provide reliable indications of laryngeal height.

Correlations between Palpation Ratings and Objective Measures

Using the full dataset, correlations between sEMG and palpation ratings were generally low, with many near zero or even negative. This is not surprising given the low inter-rater reliability of the palpation ratings. There does not appear to be an effect of task on correlations, with resting sEMG resulting in correlations similar to those for sEMG collected during running speech. Repeating correlation analyses on the high reliability participants resulted in much higher correlations over all. In particular, correlations between sEMG from electrode positions 1 and 2 and suprahyoid/supralaryngeal ratings of both systems increased. Also, correlations between sEMG from electrode position 3 and both left and right SCM ratings increased. One interpretation is that there is an underlying correlation between these sets of ratings and corresponding sEMG that was made clearer with the elimination of some of the variance in the palpation scoring. However, we cannot rule out the possibility that these increases are mere artifact produced by our manipulation of the dataset.

No association was seen between mean changes in the third formant of the /a/ vowels and the larynx position palpation rating changes pre- and post-therapy. Mathieson and colleagues also found a lack of changes in formant frequencies (first and second) pre- and post- manual therapy in 10 patients with MTD (Mathieson, et al., 2009).

Participants in this study were current therapy patients reporting for one of a number of recommended therapy sessions. Unlike so-called functional dysphonia patients for whom voice quality frequently changes drastically over the course of a single therapy session, it is more likely that these individuals displayed patterns of voice production and muscle tension that were more resistant to change. Further, therapy sessions were not necessarily directly targeting muscle tension (e.g., laryngeal massage), but varied as a function of individual patient needs. The lack of association between palpation ratings and objective measures could, therefore, also be a result of a lack of effectively-large tension changes in the pre-therapy and post-therapy conditions, given that the study was only conducted over a single session. However, these types of patients who report for multiple therapy sessions over time are those for whom a reliable palpation scale and/or objective assessment protocol would be most useful as a way of marking therapeutic progress.

ISSUES WITH RESPECT TO CLINICAL ADOPTION OF PALPATION RATING SCALES

Although neck muscle palpation for assessment and management of vocal hyperfunction is commonplace in specialized voice clinics (e.g., Aronson, 1980; Lieberman, 1998; Nelson Roy & Bless, 1998; N. Roy, et al., 1997; N. Roy & Leeper, 1993), formal documentation of neck tension is not widely practiced. The reliable recording of neck tension through palpation ratings or objective measures could lead to more standardized and well-informed patient care. Obstacles to the advised use of the two scales evaluated here stem from poor inter-rater reliability. The clinicians who participated as raters in this study described several major flaws that they perceived with these scales that included overly-broad distinctions, general lack of bilateral (left versus right) discriminations, neglect of essential categories, and inappropriate guiding text. None of the raters in this study felt that either system was a valuable addition to their current (qualitative) protocol for monitoring neck muscle tension across the course of therapy.

A major criticism of both systems was the lack of discrimination possible. The 4- and 5-point scales were often insensitive to within-therapy changes, even when the clinician believed that they could palpate a change in muscle tension. It is possible that a scale with more divisions, or a visual analog scale such as the one employed by the Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V; Kempster, et al., 2009) could result in more reliable within-therapy results. However, more studies should be performed on this matter given that increasing sensitivity from a 4-point scale to a visual-analog scale can, in some cases, result in decreased inter-rater reliability (Wuyts, De Bodt, & Van de Heyning, 1999).

Vocal hyperfunction often causes patients to present with imbalanced muscular patterns (Lieberman, 1998). These patterns cause asymmetry that may be evident during laryngoscopy as well as through palpation. However, with the exception of the right and left SCM categories of the Mathieson *et al.* scale, no other categories distinguish between right and left muscle behaviors. The raters of the present study often felt significant differences bilaterally, leading to rater confusion given the limited options for ratings. Likewise, lack of discrimination between anterior and posterior stiffness for the thyrohyoid category of Angsuwarangsee and Morrison's system also lead to rater confusion, since the accompanying text referred to both muscular contraction and differences in the thyrohyoid space. Further, in palpation of the SCM (right and left), often differences were felt between the superior and inferior ends of the SCM. Ratings based on the 'average' muscle tension in cases like these might mask clinically-relevant changes in muscle tension that speech-language-pathologists have the ability to palpate.

The accompanying text of the Angsuwarangsee and Morrison system often caused frustration for the raters of this study. The text descriptions of this system have multiple parts, and raters frequently identified parts of multiple text descriptors that spanned different numerical ratings within the same patient. One example of this was seen more than once for the thyrohyoid measure: agreement with "some contraction on phonation" for a rating of "1", as well as agreement with "tense, narrow thyrohyoid space at rest" for a rating of "2" (see Table 4-1 for reference to this system). In some cases, raters even identified with text descriptors of non-adjacent ratings (e.g., agreeing with text for a

rating of 0, as well as a rating of 2). In the particular case of the CT text descriptors, raters in this study felt that the emphasis on the size of the cricothyroid space rather than the tension felt in the cricothyroid muscle was misplaced. Likewise, raters felt that the description for pharyngolaryngeal category that asks the rater to attempt to rotate the larynx a full 90˚ was in most cases inappropriate. The general consensus of the raters of this study, all of whom had several years of experience working exclusively in voice, was that the text descriptions were a distraction. It is possible, however, that the text descriptors in this system may be of more use for clinicians having less experience with voice therapy, in which specific text descriptors may serve as a much-needed guide.

Conclusions

This study examined two recently published clinical neck tension palpation tension rating systems in individuals receiving a single session of voice therapy for hyperfunction-related disorders to determine whether the systems could produce reliable results when administered by speech-language pathologists previously unfamiliar with them. The study further attempted to determine whether either of the systems was correlated with objective measures of neck tension (sEMG and change in the third formant for the vowel /a/). For the 16 individuals studied, Pearson's correlations between raters were generally low, and little correspondence was found between ratings and objective measures. However, a smaller set of subjects with greater inter-rater agreement showed a stronger relationship between palpation ratings of the supralaryngeal area and sEMG measured on the anterior neck. These scales may be helpful in providing guidance for beginning voice practitioners and may be useful to mark long-term progress from a disordered to fully rehabilitated state, but the current results indicate that they may not be sensitive enough for use as monitoring tools across individual sessions in the course of therapy and their clinical use is not recommended for this purpose.

Chapter 5: Acoustic, Aerodynamic, and Electromyographic Characteristics of Phonatory Behaviors in Individuals with Vocal Fold Nodules

Purpose: **The goal of this study was to characterize established and proposed objective measures of phonatory function in professionally trained singers and non-singers with vocal fold nodules relative to individuals with healthy normal voice (controls).**

Method: **Surface electromyography (sEMG) from three anterior neck locations and acoustic rise times for vowels /a/ and /i/ were measured in 10 singers with nodules, 8 non-singers with nodules, and 10 controls. In individuals with nodules, vocal efficiency, glottal airflow, and nodule location and size were also measured.**

Results: **Vowel rise times and sEMG during vocal tasks did not differentiate groups. Sternocleidomastoid sEMG during initiation of the vowel /a/ was statistically significantly higher in non-singers with nodules relative to singers with nodules and controls. There were no significant differences between singers and non-singers with nodules with respect to airflow, vocal efficiency, nodule size, or nodule location. In individuals with nodules, significant correlations were seen between various sEMG measures, and also between airflow and nodule size. When non-singers and singers were examined separately, correlations among measures differed.**

Conclusions: **Neither anterior neck sEMG nor the vowel rise times differentiated individuals with nodules from controls. Future work should monitor objective measures of vocal hyperfunction over the course of therapy.**

Introduction

One of the most frequently occurring voice disorders is vocal fold nodules (Herrington-Hall, Lee, Stemple, Niemi, & McHone, 1988). Vocal fold nodules are benign lesions, clinically defined as small protuberances located between the anterior and middle third of the vocal fold (Aronson, 1980; Dikkers & Schutte, 1991; Marcotullio, et al., 2002). They are described as being gray, white, or pearl in color and bilateral (Aronson, 1980; Marcotullio, et al., 2002), and can impede complete closure of the glottis leading to breathy voice production (e.g., Colton, et al., 2006).

While nodules occur in a variety of occupations, they are commonly found in singers (operationally defined here as individuals with professional training in singing; Herrington-Hall, et al., 1988). There has been some question as to whether nodules in singers and non-singers have a common etiology and/or presentation (e.g., Peppard, Bless, & Milenkovic, 1988). Previous studies have compared singer and non-singers with nodules with respect to nodule size and location, as well as acoustic and aerodynamic measures. Peppard et al. (1988) rated vocal fold mass on an equal-appearing interval scale from 1 to 6, finding that 10 non-singers had significantly larger nodules than 10 singers. In a retrospective qualitative review of 312 professional voice users reporting with voice defects (singers, teachers, and actors), Sedláčková (1961) reported a trend toward differences in size, shape, and location of the nodules seen in singers and non-singers. Specifically, Sedláčková reported that singers' nodules tend to be small, pale/white, and located at the anterior third of the of the vocal fold, whereas nodules in non-singers tend to be larger, sitting on a larger base, having a gel-like appearance, and located further posterior. Peppard et al. (1988) found that singers with nodules performed similarly to non-singers without nodules on a selection of acoustic and aerodynamic measures, and even outperformed the non-singers on maximum performance tasks such as maximum phonation time and maximum frequency range. However, in comparison to non-singers, singers are much more attuned to their voices, and thus they are more likely to seek treatment in the early stages of formation, when the nodules are relatively small. Further, singers are trained to adapt to day-to-day changes in their vocal mechanism to produce their best voice, making them likely to use differing compensatory mechanisms than non-singers. These confounding factors make it difficult elucidate possible differences between singers and non-singers.

One possible difference between singers and non-singers is the role of vocal hyperfunction in both nodule formation and the resulting compensation. Vocal hyperfunction refers to "conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or 'imbalanced' muscular forces" (Hillman, et al., 1989), characterized by excessive laryngeal and paralaryngeal tension (Aronson, 1980; Dworkin, Meleca, & Abkarian, 2000; Koufman & Blalock, 1991b; M. D. Morrison, et al., 1983; N. Roy, et al., 1996), and commonly accompanies voice disorders. For many years, organic developments on the vocal fold surface have been assumed to be related to

hyperfunctional behavior or phonotrauma (Hillman, et al., 1990). For instance, in 1962, Godrey Arnold wrote that "vocal nodules and polyps represent a local tissue reaction to the mental strain imposed by inappropriate emotional adjustment to the demands made by society" (Arnold, 1962). However, much is still unknown about the underlying mechanisms of vocal hyperfunction and its role in developing organic disorders (Hillman, et al., 1990). Given the extensive vocal demands of singers, it is possible that vocal hyperfunction plays a smaller role, and that nodule formation is more typically a result of their massive amounts of vocal use.

It is generally believed that voice therapy has the potential to decrease nodules and improve voice quality (Carding, et al., 1999; Holmberg, et al., 2001) and to be generally effective in treating non-organic voice disorders (Carding & Horsley, 1992). However, an obstacle to efficacious use of voice therapy for the treatment of disorders associated with vocal hyperfunction is the limited ability of the therapist to reliably ascertain changes in the patient's vocal hyperfunction. There is no widely accepted diagnostic measure of the presence and degree of vocal hyperfunction, and currently, assessment of vocal hyperfunction during diagnosis is often primarily based on subjective impressions given the patient's history and presentation of symptoms such as auditory-perceptual and visual or tactile discrimination of muscle tension (e.g., laryngeal palpation).

The fundamental qualification for voice disorders is, in fact, the subjective (auditory-perceptual) judgment of dysphonia. However, changes in auditory-perceptual assessment of vocal behaviors are subjective, and may also be temporally separated from changes in the patient's vocal hyperfunction, especially in individuals with glottal insufficiency such as nodules. In the case of vocal hyperfunction, the perception of vocal strain is the most commonly associated auditory-perceptual correlate (Colton, et al., 2006), and the frequency of hard glottal attack is also thought to be related to vocal hyperfunction (e.g., Andrade, et al., 2000).

It is thought that signs of vocal hyperfunction can be seen during flexible (trans-nasal) endoscopy as supraglottic compression. Supraglottic compression describes obstruction of part or all of the view of the vocal folds during indirect laryngoscopy and videoendoscopy (Stager, et al., 2000). There are two primary components: anterior-posterior (AP) compression, in which the arytenoid cartilages are drawn toward the petiole of the epiglottis, and medial or false vocal fold (FVF) compression, which is caused by adduction of the false vocal folds (Stager, et al., 2000). Studies have cited this supraglottic activity as a symptom characteristic of vocal hyperfunction (M. Morrison, et al., 1999), but without comparisons to its prevalence in populations with healthy normal voice. Stager and colleagues compared the incidence of AP and FVF compression in individuals with nodules and healthy normal voice participants, finding that the incidence in individuals with healthy normal voice was less than that in the disordered voice patients, but was not absent (Stager, et al., 2000). Further, this mode of assessment requires the individual to undergo flexible endoscopy for assessment; while this procedure is routinely performed in multidisciplinary voice clinics, it is not always well-tolerated by some patients.

Vocal hyperfunction symptoms may not be limited to the larynx. Many muscles in the neck that attach to the larynx and/or hyoid bone have voice and speech-related contractions due to their role in controlling the vertical position of the larynx in the neck

and, to some degree, the position of the tongue. When individuals demonstrate an inappropriate degree of intrinsic laryngeal muscle contraction (vocal hyperfunction), it is thought that they often simultaneously contract the extrinsic laryngeal muscles and other superficial neck muscles in a similar hyperfunctional manner (Aronson, 1980). This hypothesis is most recently supported by the work of Roy and colleagues, who report that individuals with vocal hyperfunction have vowel space expansion after therapy, suggesting modification of speech articulation (N. Roy, et al., 2009). Thus, another common method for ascertaining vocal hyperfunction is through physical palpation of the laryngeal and paralaryngeal muscles. A study by Altman and colleagues looked at 150 patients who had been diagnosed with muscle tension dysphonia; based on a speech pathology evaluation of these patients, 70% were found to have "obvious cervical neck tension visible" (Altman, et al., 2005). Clinically, excessive tension has been noted via palpation over the major horns of the hyoid bone, over the superior cornu of the thyroid cartilage, along the anterior border of the sternocleidomastoid muscle, and throughout the suprahyoid musculature (e.g., N. Roy, et al., 1996). Strap muscle tension can be noted in patients with vocal hyperfunction through both visual and tactile inputs. In particular, it is thought that observation of the inferior bellies of the omohyoid muscle crossing the supraclavicular fossae shows them to be tense and prominent during speech (M. Morrison, 1997b). Further information about the extent of muscle tension found in the patient can be gained by palpation of the larynx at rest and during voicing (M. Morrison, 1997b). However, there are only a few formal rating scales for consistent rating of laryngeal muscle tension (e.g., Angsuwarangsee & Morrison, 2002; Mathieson, et al., 2009), and recent work suggests that these scales may not reliably capture voice-related neck muscle tension (see this Thesis Chapter 4).

There is a need to develop relatively non-invasive objective clinical assays of vocal hyperfunction to aid in voice therapy treatment of patients with vocal hyperfunction to allow for repeated assessment throughout treatment to identify progress. Although they are rarely used diagnostically, glottal airflow magnitude and vocal efficiency are objective measures often collected in individuals with nodules to add additional information about the impairment of his or her phonatory function. Two other possible modes of vocal hyperfunction assessment are the objective correlates of the subjective measures of voice quality and muscle tension.

The glottal airflow magnitude (referred to simply as airflow and typically reported in units of L/s) represents the magnitude of airflow through the glottis during vocal fold oscillation. Airflow values have been reported for both singers and non-singers with nodules and healthy normal voice (Peppard, et al., 1988). Results indicated that mean airflow values did not differ between singers with nodules and those without, but that non-singers with nodules had significantly greater airflow values than non-singers with healthy normal voice. Airflow values for all participants were found to be significantly correlated ($R^2 = 0.20$) with subjective ratings of nodule mass viewed during endoscopy; however, correlations were not calculated within singer and non-singer categories, leaving the question as to whether airflow values are more or less indicative of nodule size in the two populations.

Subglottal pressure is the driving force behind vocalization, and abnormally high values of subglottal pressure can indicate inefficient valving of the glottal airstream. Vocal

efficiency is an aerodynamic-acoustic measure representing the ratio of acoustic power (sound pressure level) to aerodynamic power (subglottal pressure), often represented in units of dB SPL / cm H_2O, and is associated with the level of effort necessary to achieve phonation. Jiang et al. (2004) reported significant differences in the vocal efficiency measures of individuals with nodules and individuals with healthy normal voice at various vocal intensities. However, no comments were made regarding the singing training of these individuals, nor were measures of nodule size indicated. In an unpublished Master's thesis, Fulton (2007) explored the vocal characteristics of 10 females who were trained singers with healthy normal voice relative to 10 females with healthy normal voice who were non-singers, finding no significant differences between the vocal efficiency measures of the two groups when producing vowels at typical pitch and loudness levels. It is unclear, however, if this lack of difference in healthy normal speakers is relevant in a population of individuals with nodules.

Although strain is likely the most common auditory-perceptual quality attributed to vocal hyperfunction, there is no known good acoustic correlate (e.g., Colton, et al., 2006). Although electroglottography may be used to indirectly assess closure of the vocal folds (how "pressed" the voice is), this measure is difficult to interpret. Another perceptual correlate of vocal hyperfunction is hard glottal attack, which is a method of vowel initiation perceptually "characterized by rapid and complete adduction of the vocal folds before the initiation of phonation" (Colton, et al., 2006, pg 78). Alternative terminology for hard glottal attack includes "abrupt glottal attack", "abrupt voice onset", and "hard voice onset". A retrospective study by Morrison and colleagues found that 65% of patients with hyperfunction presented with hard glottal attack (M. D. Morrison, et al., 1986) and comparison of the frequency of hard glottal attack in vocal disorders thought to be related to vocal hyperfunction found higher frequencies of hard glottal attack in all groups with voice disorder when compared with a control group of individuals with healthy normal voice (Andrade, et al., 2000). While the clinic continues to rely on broad auditory perception to characterize glottal attack, more objective methods do exist (see Orlikoff, Watson, Baken, & Deliyski, 2006 for review). Peters et al. (1986) calculated the rise time of the acoustic signals of vowel production and found a correlation of -0.69 with auditory perceptual ratings of voice onset abruptness, indicating the usefulness of vowel rise time as an objective measure of glottal attack.

Some previous studies have attempted to characterize neck muscle contraction in individuals with vocal hyperfunction. Specifically, a study by Angsuwarangsee and Morrison (2002) compared the muscle tension of four extrinsic laryngeal muscle groups via palpation in participants diagnosed with and without vocal hyperfunction. This study found that individuals with vocal hyperfunction had significantly more tension in their thyrohyoid muscle than individuals without vocal hyperfunction, but relied on the subjective palpation methodology.

Surface electromyography (sEMG) could possibly be used to objectively quantify neck muscle tension. Redenbaugh and Reich (1989) measured mean neck sEMG of 7 individuals with healthy normal voice and 7 "hyperfunctional" individuals, finding that the individuals with disordered voice had significantly greater mean normalized neck sEMG during phonation than individuals with healthy normal voice. The sEMG signal corresponded with a single electrode position, centered over the thyrohyoid membrane, a

recording position likely to sample electrical activity from the sternohyoid and possibly cricothyroid muscles during production of vowels and a reading passage. The disordered population in this study was varied, consisting of seven individuals with very different clinical presentation, history, and even sex (5 women, 2 men), and the data collection method was relatively rudimentary. The sEMG signals were amplified, filtered, and integrated (2-sec interval) in real-time, with the integrated values displayed on-screen only and recorded by hand. Hocevar-Boltezar et al. (1998) also attempted to characterize the sEMG in individuals with vocal hyperfunction. Eleven women with disorders associated with vocal hyperfunction (nodules, muscle tension dysphonia) were examined with respect to 5 women with healthy normal voice. The sEMG recorded corresponded to 18 pairs of surface electrodes (differential recording setup) applied bilaterally to the face and anterior neck while the participants produced sustained vowels. Although this study found significant differences between the mean sEMG of many electrode positions in the two groups, sEMG signals were not normalized. In order to reduce the variability due to neck surface electrode contact and participant neck mass, sEMG signals should be normalized to a reference contraction before they are compared among conditions and/or participants (Netto & Burnett, 2006).

Neither of these two sEMG studies comments on the past singing training of participants, which could have significant effects on the use of the extrinsic musculature for speech. For instance, in a study of four professional, classically trained singers (baritones) and four non-singers producing the vowel /a/ at seven frequency points between 90 and 350 Hz, qualitative differences between frequency and laryngeal height were found (Shipp & Izdebski, 1975). Non-singers tended to increase laryngeal height with increases in fundamental frequency, whereas singers tended to keep their larynges below their resting heights for all fundamental frequencies. It is possible that singers use their extrinsic laryngeal musculature in very different ways than non-singers to attempt to compensate for their vocal pathology.

Descriptive aerodynamic, acoustic, and electromyographic data in singers and non-singers with nodules relative to individuals with healthy normal voice will provide baseline data for future studies in these populations. In addition to the more established objective measures of airflow, vocal efficiency, and nodule size and location (measured quantitatively here), the acoustic vowel rise time, and anterior neck sEMG measures were also investigated as possible non-invasive and objective indicators of vocal hyperfunction in singers and non-singers with nodules.

Methods

PARTICIPANTS

Participants were 10 adult females with a history of professional singing training diagnosed with vocal fold nodules prior to any therapeutic intervention (mean age = 19.7 years, SD = 0.8 years), 8 adult females diagnosed with vocal fold nodules prior to any therapeutic intervention with no history of singing training (mean age = 34.1 years, SD = 12.1 years), and 10 adult females with healthy normal voice (mean age = 23.8 years, SD = 2.0 years) to act as experimental controls. The group of singers with nodules was comprised of working professional singers and full-time college or graduate students of voice. Based on self-report, their primary styles of singing were: musical theater (N = 6), pop (N = 2), gospel (N = 1), and opera (N = 1). Individuals recruited with healthy normal voice were volunteers with no voice-related complaints and were screened for abnormal pathology of the larynx using trans-oral or trans-nasal endoscopy with stroboscopy. While 2 of these 10 individuals sang for pleasure, none were professionally trained. Disordered participants were diagnosed based on comprehensive voice evaluation procedures that included endoscopic, acoustic, aerodynamic, and perceptual assessment by a team comprised of a laryngologist and one or more certified speech-language pathologists, and images of their vocal folds were collected via standard trans-oral endoscopy with stroboscopy.

RECORDING PROCEDURE

Recordings consisted of a brief vocal assessment of the participant including three trials of the vowels /a/ and /i/, read speech (The Rainbow Passage; Fairbanks, 1960), six read CAPE-V sentences (i.e., Kempster, et al., 2009), and spontaneous running speech. Spontaneous speech was elicited by questions from the investigator or a speech-language pathologist, asking the participant to describe their voice issues (if relevant), or describe what they did the previous weekend (or similar). After completion of these speech tasks, maximal voluntary contraction (MVC) maneuvers were performed. These consisted of asking the participants to perform neck contraction against manual resistance for the purpose of normalizing sEMG data (see Data Analysis section).

Simultaneous anterior neck sEMG and acoustic signals from a lavalier microphone (Sennheiser MKE2-P-K, Wedemark, Germany) were recorded digitally with Delsys ™ (Boston, Massachusetts) hardware (Bagnoli Desktop System) and software (EMGworks 3.3) at 20 kHz. The sEMG was recorded and analyzed in view of current European standards (Hermens, et al., 1999). Participants' necks were prepared for electrode placement by cleaning the neck surface with an alcohol pad and "peeling" with tape to reduce electrode-skin impedance, noise, DC voltages, and motion artifacts. The anterior neck sEMG was recorded with three Delsys ™ 3.1 double-differential surface electrodes

placed parallel to the underlying muscle fibers of the 1) thyrohyoid, omohyoid, and sternohyoid muscles, 2) cricothyroid and sternohyoid muscles, and 3) sternocleidomastoid muscle. The Delsys ™ 3.1 double-differential surface electrodes consisted of three 10-mm silver bars with inter-electrode distances of 10-mm.

Electrode 1 was centered about 1 cm lateral to the anterior neck midline, as far superior as was possible without impeding jaw opening of the participant. Electrode 2 was centered on the gap between the cricoid and thyroid cartilages of the larynx, and centered at 1 cm lateral to the midline, contralateral to Electrode 1. Electrode 3 was centered one-third of the distance from the sternal notch of each participant to his or her mastoid process (following Falla et al.2002). For some further discussion of electrode-placement methodology, see Stepp et al. (submitted) A ground electrode was placed on the superior aspect of the participant's left shoulder. The sEMG signals were pre-amplified and filtered using Delsys™ Bagnoli systems set to a gain of 1000 and a band-pass filter with roll-off frequencies of 20 Hz and 450 Hz.

Individuals with nodules underwent aerodynamic voice assessment with a Phonatory Aerodynamic System (KayPENTAX, Lincoln Park, NJ). The participants produced a series of /pæ/ vocalizations at a comfortable pitch and loudness while the airflow and intra-oral air pressure were measured with a translabially placed catheter connected to a pressure transducer.

Data Analysis

The mean of the root-mean-squared (RMS) values of anterior neck sEMG data computed in 1 s windows (no overlap) was calculated for the entire length of all completed vocal tasks using custom MATLAB® (Mathworks Inc., Natick, MA) software. Since intrinsic laryngeal musculature is most active during initiation and cessation of vowel production relative to stable vowel production (e.g., Gallena, Smith, Zeffiro, & Ludlow, 2001), for the production of the vowels /a/ and /i/, the RMS was also calculated for 100 ms windows (no overlap) during the 500 ms before and 300 ms after vowel initiation, as well as during the 300 ms before and 500 ms after vowel termination. In order to compare anterior neck sEMG gathered among participants, the variability associated with neck surface electrode contact and placement was minimized by normalizing the sEMG to the MVC reference contraction (calculated as the RMS in a 1 s window). It has been shown that for anterior neck musculature, the MVC reference is more reliable (Netto & Burnett, 2006). For this reason, all of the sEMG data presented here are in terms of % MVC.

The pressure signals measured during the aerodynamic voice assessment were digitized and analyzed with the Phonatory Aerodynamic System to produce indirect estimates of subglottal air pressure. This subglottal air pressure and the sound pressure level produced by the participant were then used to calculate vocal efficiency, here defined as the ratio of the sound pressure level in dB SPL to the subglottal air pressure in cm H_2O. Glottal airflow estimates were collected from the steady-state values of airflow during the /æ/ portions of testing during repeated productions of /pæ/.

In order to find an acoustic correlate for abruptness of attack, the acoustic rise times of the vowels /a/ and /i/ were measured by analyzing the acoustic signals similarly to the

method utilized in Peters et al. (1986). Peters et al. (1986) defined the rise time of the acoustic signal as the time needed for an envelope of the acoustic signal to go from 10% to 90% of the maximum amplitude. To implement this method, the RMS of the acoustic signal in 80 ms rectangular windows was calculated in intervals of 2.5 ms (97% overlap). Due to the disordered nature of the voice signals being analyzed, the window size employed here was modified from the method employed by Peters et al. (1986) to be 80 ms rather than 40 ms, the slope initiation was defined as 20% of the maximum amplitude rather than 10%, and the slope termination was defined as 80% of the maximum amplitude rather than 90%. An example of the rise time for a vowel production is shown in Figure 5-1.

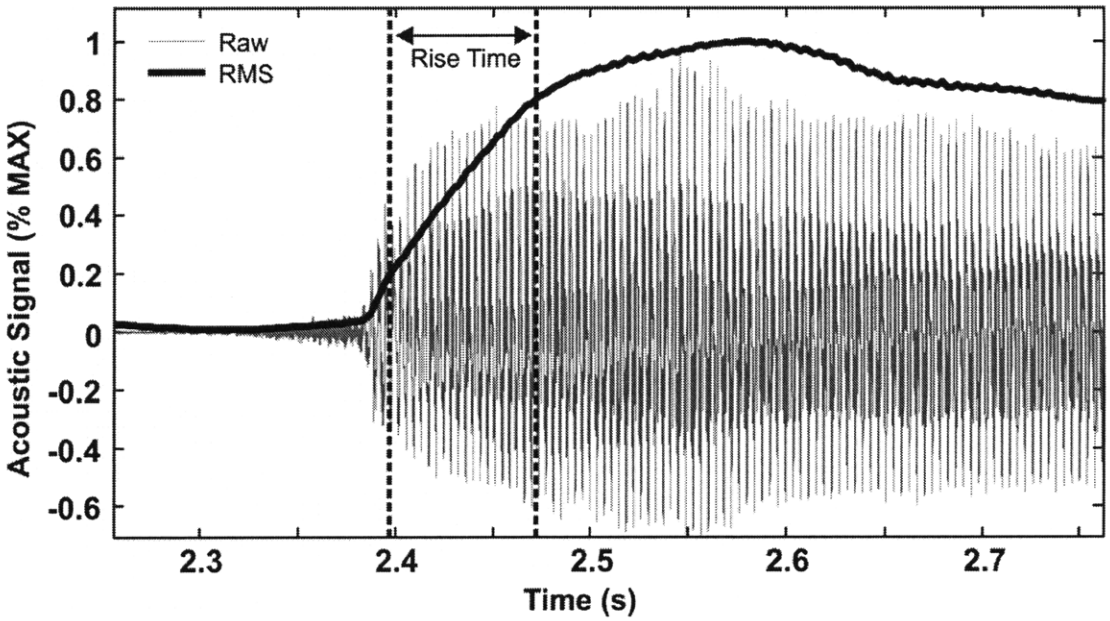


Figure 5-1. An example of the calculation of the acoustic rise time for a production of the vowel /a/. The thin line shows the raw acoustic signal, and the thick line shows the RMS of the acoustic signal. The acoustic signals are plotted as a function of the maximum values for clear display.

For the individuals with nodules, a representative digital still was taken from their trans-oral stroboscopy exam. Stills were chosen to maximize image quality, while showing the full extent of the vocal folds during abduction. The vocal fold stills were analyzed using custom MATLAB® software that allowed for the following landmarks to be marked (in pixels): the length of each vocal fold (right and left) from the anterior commissure to and including the vocal process of the arytenoid cartilage, the distance from the center of each nodule (right and left) to the anterior commissure, and the visible extent (area) of each

nodule (right and left). The marked extent of each nodule was based on the visible changes to the vocal fold surface. In some more mild cases, the area defined as the nodule consisted of only the protrusion past where the smooth edge of the vocal fold would have been. In more extreme cases, distinct fibrovascular changes to the vocal fold could be seen lateral to where the smooth edge would be, and these changes were included as part of the extent of the nodule. The nodule location for each participant was defined as the mean of the ratio of the distance from the nodule to the aperture to the total length of the vocal fold. The nodule size was calculated as the mean of the two nodule extents normalized by the mean of the length of the two vocal fold.

Minitab® Statistical Software (Minitab Inc., State College, PA) was used to calculate Pearson's correlations, and to execute statistical testing, which was performed by analysis of variance (ANOVA), Tukey simultaneous *t*-tests, and Student's *t*-tests. Statistical testing was not adjusted for alpha inflation due to the exploratory nature of this study.

Results

Mean Anterior neck sEMG and Acoustic Rise Time in All Participants

The mean anterior neck sEMG at recording positions 1, 2, and 3 during the production of all vocal tasks was analyzed with a two-factor ANOVA by group and vocal task, which failed to find a statistically significant ($p < 0.05$) effect of group (control, singer nodules, non-singer nodules) for any anterior neck sEMG location, but a statistically significant effect of vocal task (rest, /a/, /i/, high /a/, low /a/, read sentences, read paragraph, and spontaneous speech) for all three recording locations (EMG1, $p < 0.001$; EMG2, $p < 0.001$; EMG3, $p < 0.001$) was found. The mean anterior neck sEMG at all three recording positions tended to be lower during rest and production of the vowels /a/ and /i/, and higher during both read and spontaneous speech production.

The anterior neck sEMG recorded from positions 1, 2, and 3 was averaged in 100 ms segments during the 500 ms before and 300 ms after vowel initiation, as well as the 300 ms before and 500 ms after vowel termination. The results of this analysis are shown as a function of group and vowel (/a/ and /i/) in Figure 5-2. Based on the trends seen in the vowel initiation, a one-factor ANOVA of the mean anterior neck sEMG at recording positions 1, 2, and 3 during the 500 ms prior to vowel production was performed; however, it failed to find a statistically significant effect of group (control, singer nodules, non-singer nodules) for both vowels (/a/ and /i/) and all recording positions (1, 2, 3), with the exception of recording position 3 prior to the vowel /a/, which showed a statistically significant effect of group ($p = 0.014$). Tukey simultaneous *t*-tests showed a statistically significant difference between the anterior neck sEMG at recording position 3 prior to /a/ between the controls (MEAN = 0.015 % MVC) and non-singers with nodules (MEAN = 0.037 % MVC; $p_{adj} = 0.020$) and between singers with nodules (MEAN = 0.015 % MVC) and non-singers with nodules ($p_{adj} = 0.028$). No statistically significant difference ($p_{adj} < 0.05$) was found between controls and singers with nodules. Example traces of the raw voice and anterior neck sEMG data for one control participant and one non-singer with nodules are shown in Figure 5-3.

A one-factor ANOVA of the acoustic rise time during vowel production (/a/ and /i/) failed to find a statistically significant effect of group (control, singers with nodules, non-singers with nodules). However, a general trend was seen in the acoustic rise time measure for both vowels, suggesting smaller rise times for non-singers with nodules relative to the singers with nodules and controls. Boxplots of the acoustic rise time measures in the three groups are shown in Figure 5-4.

Correlations between acoustic rise time and the anterior neck sEMG at recording positions 1, 2, and 3 500 ms prior to vowel production are shown for all groups in Table 5-1. No statistically significant ($p < 0.05$) correlations were found between any of the

rise time measures and anterior neck sEMG measures, and correlations were generally weak with R^2 values ranging from 0.01 – 0.11.

AIRFLOW, VOCAL EFFICIENCY, AND MEASURE OF NODULE SIZE AND LOCATION IN SINGERS AND NON-SINGERS WITH VOCAL NODULES

Student's *t*-tests on airflow, vocal efficiency, and nodule size and location did not show statistically significant (two-sided, $p < 0.05$) differences between singers and non-singers with nodules. There was a trend, however, for larger nodule size and greater airflow in non-singers relative to singers. Boxplots of the airflow, vocal efficiency, and nodule size and location for the two groups are shown in Figure 5-5.

CORRELATIONS BETWEEN MEASURES IN SINGERS AND NON-SINGERS WITH VOCAL NODULES

Correlations among vowel rise time measures, anterior neck sEMG prior to vowel production, airflow, vocal efficiency, nodule location, and nodule size in all individuals with nodules (singers and non-singers) are shown in Table 5-2. Statistically significant ($p < 0.05$) correlations were found between a number of the anterior neck sEMG measures, as well as between airflow and nodule size.

Correlations among vowel rise time measures, anterior neck sEMG prior to vowel production, airflow, vocal efficiency, nodule location, and nodule size were calculated separately in non-singers and singers with nodules and are shown in Tables 5-3 and 5-4, respectively. In non-singers, statistically significant ($p < 0.05$) correlations were found between a number of the anterior neck sEMG measures, as well as between: vocal efficiency and EMG 3 prior to /a/, airflow and EMG 3 prior to /a/, airflow and nodule size, and airflow and vocal efficiency. In singers, statistically significant correlations were found between a number of the anterior neck sEMG measures, as well as between: airflow and linear rise time for /i/, nodule size and EMG3 prior to /i/, and vocal efficiency and EMG3 prior to /a/.

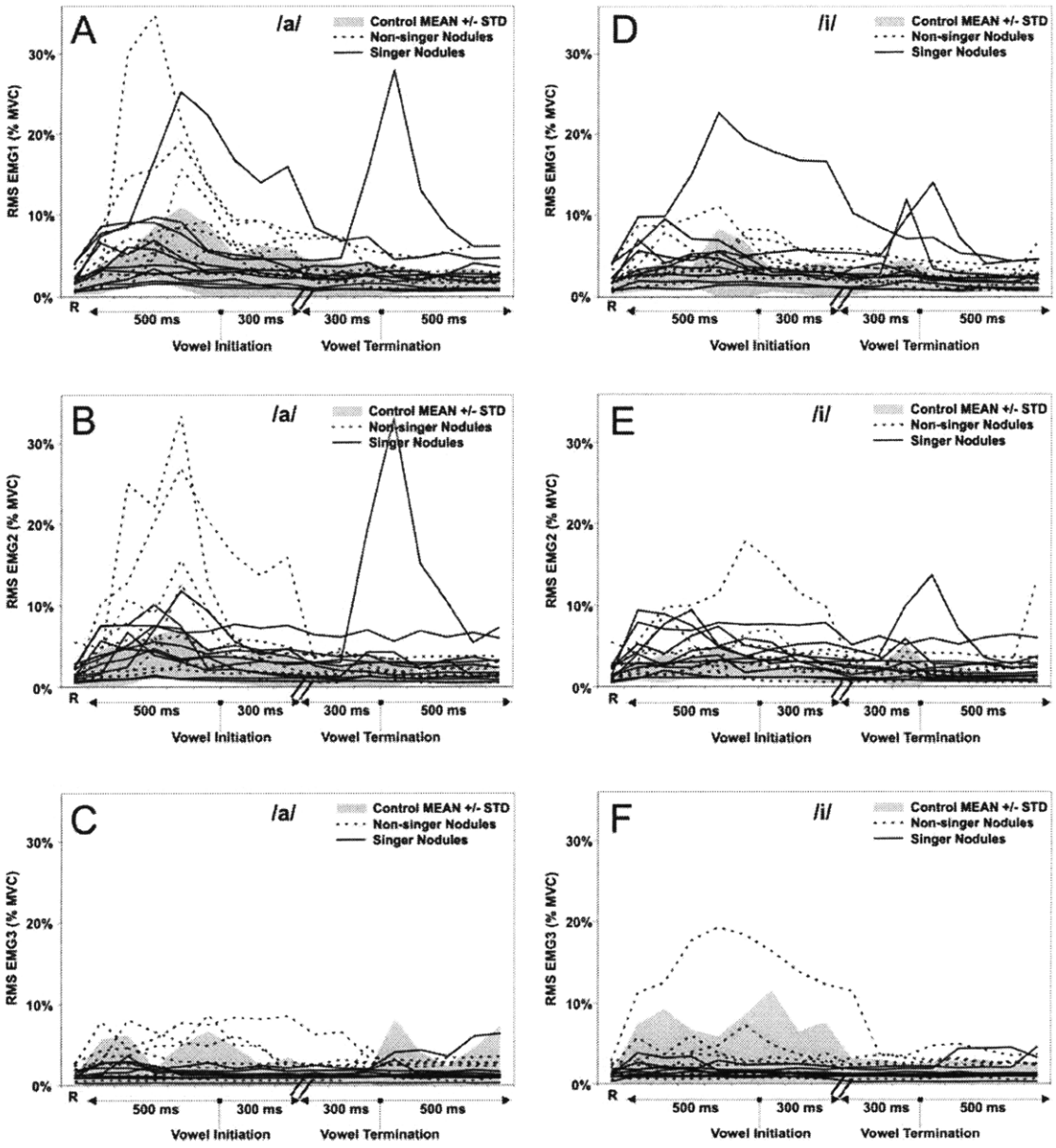


Figure 5-2. The anterior neck sEMG recorded from positions 1, 2, and 3, averaged in 100 ms segments during the 500 ms before and 300 ms after vowel initiation, as well as the 300 ms before and 500 ms after vowel termination for the vowels /a/ and /i/. The shaded area indicates +/- one standard deviation from the mean of the participants with healthy normal voice. Individual data for individuals with nodules are shown. Singers are shown in the solid line, whereas non-singers are shown in the broken line. The initial data point labeled 'R' indicates at rest sEMG values for reference.

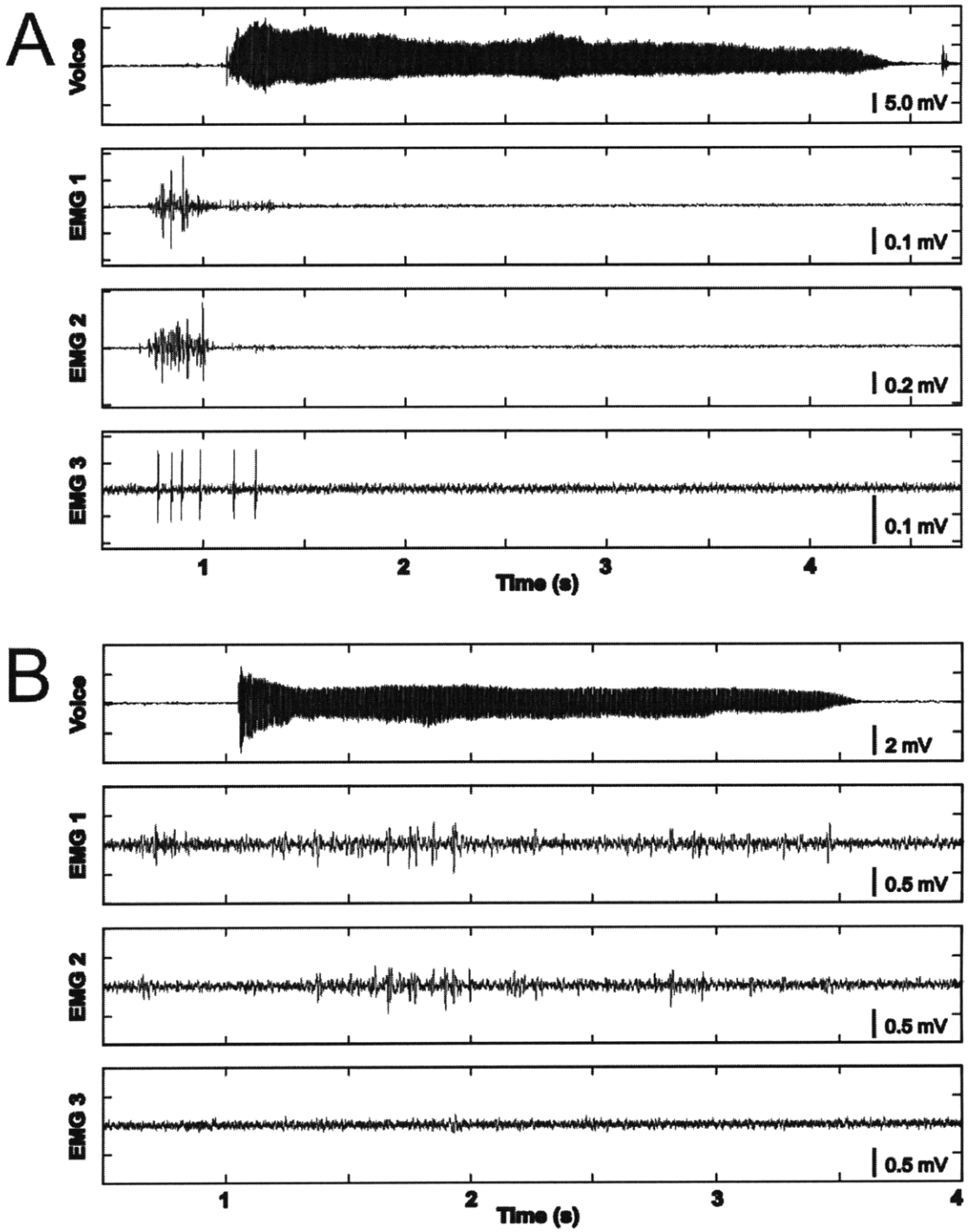


Figure 5-3. Two examples of the raw acoustic signals and anterior neck sEMG leading up to the production of the vowel /a/. Panel A shows the signals of a non-singer with nodules. Panel B shows the signals of a participant with healthy normal voice.

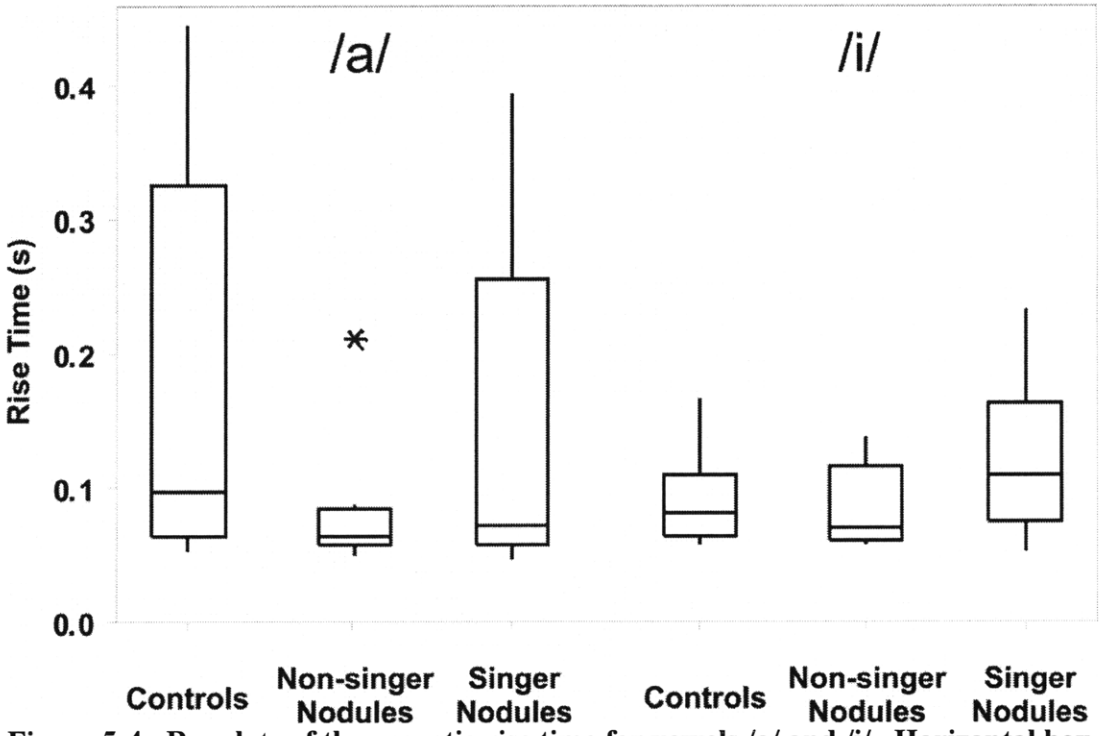


Figure 5-4. Boxplots of the acoustic rise time for vowels /a/ and /i/. Horizontal box lines indicate the lower and upper quartiles of the data, with the center line marking the data median. Vertical whiskers extend from the boxes to the minimum and maximum values of each dataset. The asterisk marks a data observation falling more than 1.5 times the interquartile range higher than the third quartile.

		T_r /a/			T_r /i/
sEMG 500 msec prior to /a/ initiation	EMG1	-0.084 (0.582)	sEMG 500 msec prior to /i/ initiation	EMG1	0.331 (0.086)
	EMG2	-0.240 (0.218)		EMG2	-0.085 (0.668)
	EMG3	-0.152 (0.459)		EMG3	-0.112 (0.587)

Table 5-1. Correlations between acoustic rise time and electromyographic measures in all participants (controls with healthy normal voice, singers with nodules, and non-singers with nodules). Associated *p*-values for the Pearson's correlations are shown in parentheses. The linear rise time (s) is abbreviated as T_r during vowels /a/ and /i/.

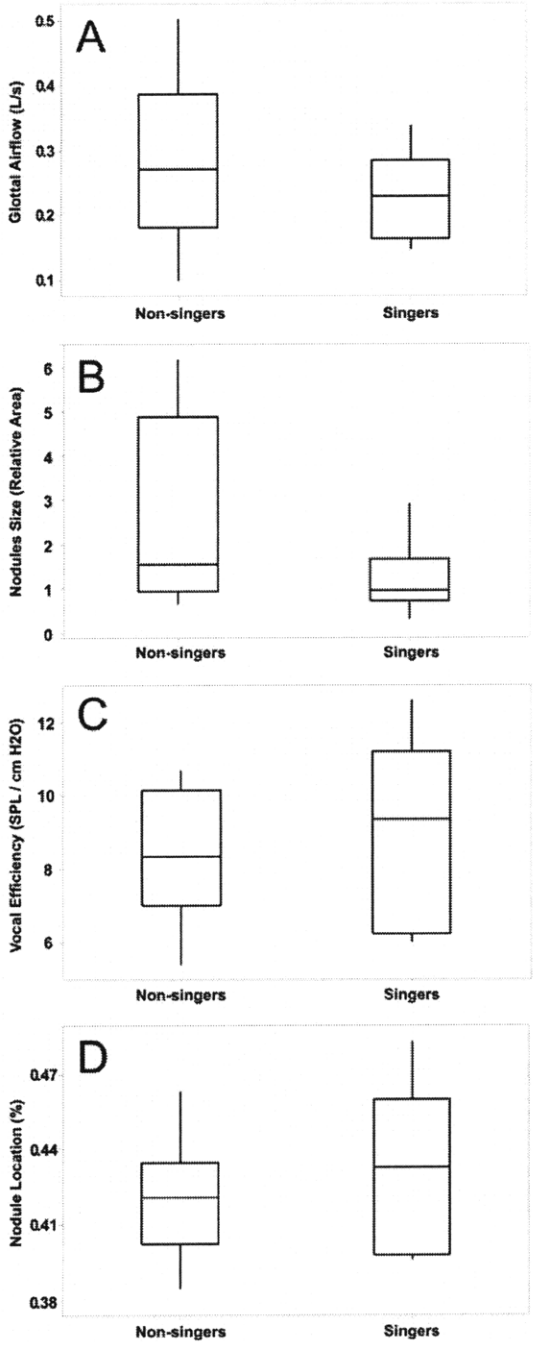


Figure 5-5. Boxplots of the airflow (Panel A), nodule size (Panel B), vocal efficiency (Panel C), and nodule location (Panel D) in individuals with vocal fold nodules. Horizontal box lines indicate the lower and upper quartiles of the data, with the center line marking the data median. Vertical whiskers extend from the boxes to the minimum and maximum values of each dataset. "Relative area" refers to the approximation of the nodule size, which was calculated as the mean of the two hand-marked nodule extents normalized by the mean of the length of the two vocal folds (pixels^2 / pixels).

	T_r /a/	T_r /i/	E_1 /a/	E_1 /i/	E_2 /a/	E_2 /i/	E_3 /a/	E_3 /i/	N_{loc}	N_{size}	Eff	Flow
T_r /a/												
T_r /i/	0.214 (0.394)											
E_1 /a/	-0.139 (0.582)	0.288 (0.246)										
E_1 /i/	-0.175 (0.487)	0.400 (0.100)	0.622 (**0.006**)									
E_2 /a/	-0.271 (0.276)	-0.034 (0.895)	0.683 (**0.002**)	0.183 (0.467)								
E_2 /i/	-0.211 (0.402)	-0.204 (0.417)	-0.028 (0.911)	0.131 (0.603)	0.506 (**0.032**)							
E_3 /a/	-0.094 (0.728)	-0.033 (0.904)	0.522 (**0.038**)	0.062 (0.821)	0.768 (**0.001**)	0.363 (0.167)						
E_3 /i/	-0.150 (0.578)	-0.203 (0.451)	0.328 (0.215)	0.265 (0.320)	0.288 (0.280)	0.167 (0.537)	0.697 (**0.003**)					
N_{loc}	-0.392 (0.108)	0.213 (0.395)	0.045 (0.858)	0.076 (0.766)	0.107 (0.672)	0.263 (0.291)	-0.217 (0.419)	-0.218 0.417				
N_{size}	-0.289 (0.245)	-0.105 (0.680)	-0.221 (0.379)	-0.216 (0.388)	-0.277 (0.265)	-0.247 (0.323)	-0.145 (0.592)	-0.026 (0.925)	0.108 (0.669)			
Eff	-0.128 (0.613)	-0.086 (0.733)	0.036 (0.887)	0.216 (0.390)	0.131 (0.606)	0.088 (0.728)	-0.013 (0.961)	0.047 (0.864)	-0.264 (0.290)	-0.439 (0.068)		
Flow	-0.043 (0.867)	0.244 (0.328)	-0.123 (0.625)	0.035 (0.891)	-0.335 (0.174)	-0.292 (0.240)	-0.373 (0.154)	-0.416 (0.109)	0.173 (0.493)	0.624 (**0.006**)	-0.465 (0.052)	

Table 5-2. Correlations between acoustic, aerodynamic, and electromyographic measures in individuals with nodules (both singers and non-singers). Associated *p*-values for the Pearson's correlations are shown in parentheses. Abbreviations are as follows: T_r, rise time (s) during vowels /a/ and /i/; $E_{1\text{-}3}$, sEMG at recording locations 1 - 3 in the 500 ms prior to vowel initiation (% MVC); N_{loc}, nodule location (% vocal fold length); N_{size}, nodule size (square pixels / pixels of vocal fold length); Eff, vocal efficiency (dB SLP / cm H_2O); Flow, glottal airflow magnitude (L / s). Bold text highlights the parameters for which a correlation significant at the $p \leq 0.05$ level was found.

	T_r /a/	T_r /i/	E_1 /a/	E_1 /i/	E_2 /a/	E_2 /i/	E_3 /a/	E_3 /i/	N_{loc}	N_{size}	Eff	Flow
T_r /a/												
T_r /i/	0.441 (0.274)											
E_1 /a/	-0.181 (0.668)	0.372 (0.363)										
E_1 /i/	-0.231 (0.582)	-0.423 (0.296)	0.336 (0.416)									
E_2 /a/	-0.240 (0.567)	0.243 (0.562)	0.716 (**0.046**)	0.105 (0.805)								
E_2 /i/	-0.099 (0.815)	-0.299 (0.472)	-0.137 (0.746)	0.127 (0.764)	0.532 (0.174)							
E_3 /a/	0.078 (0.868)	0.321 (0.483)	0.637 (0.124)	0.424 (0.343)	0.758 (**0.048**)	0.463 (0.295)						
E_3 /i/	-0.099 (0.832)	-0.188 (0.687)	0.326 (0.475)	.893 (**0.007**)	0.127 (0.785)	0.159 (0.733)	0.617 (0.140)					
N_{loc}	-0.614 (0.105)	-0.147 (0.728)	0.267 (0.523)	-0.181 (0.668)	0.201 (0.633)	-0.174 (0.680)	-0.385 (0.393)	-0.359 (0.429)				
N_{size}	-0.185 (0.660)	0.080 (0.851)	-0.369 (0.369)	-0.160 (0.705)	-0.579 (0.132)	-0.517 (0.190)	-0.671 0.099	-0.285 (0.535)	0.096 (0.822)			
Eff	-0.140 (0.741)	-0.164 (0.699)	0.272 (0.515)	0.465 (0.246)	0.505 (0.202)	0.538 (0.169)	0.824 (**0.023**)	0.531 (0.220)	-0.526 (0.181)	-0.396 (0.331)		
Flow	0.189 (0.654)	0.103 (0.807)	-0.501 (0.206)	-0.565 (0.145)	-0.607 (0.110)	-0.480 (0.229)	-0.935 (**0.002**)	-0.749 (0.053)	0.281 (0.499)	0.714 (**0.047**)	-0.786 (**0.021**)	

Table 5-3. Correlations between acoustic, aerodynamic, and electromyographic measures in individuals with nodules who are non-singers. Associated *p*-values for the Pearson's correlations are shown in parentheses. Abbreviations are as follows: T_r, rise time (s) during vowels /a/ and /i/; $E_{1\text{-}3}$, sEMG at recording locations 1 - 3 in the 500 ms prior to vowel initiation (% MVC); N_{loc}, nodule location (% vocal fold length); N_{size}, nodule size (square pixels / pixels of vocal fold length); Eff, vocal efficiency (dB SLP / cm H_2O); Flow, glottal airflow magnitude (L / s). Bold text highlights the parameters for which a correlation significant at the $p \leq 0.05$ level was found.

	T_r /a/	T_r /i/	E_1 /a/	E_1 /i/	E_2 /a/	E_2 /i/	E_3 /a/	E_3 /i/	N_{loc}	N_{size}	Eff	Flow
T_r /a/												
T_r /i/	0.060 (0.869)											
E_1 /a/	-0.008 (0.983)	0.538 (0.109)										
E_1 /i/	-0.188 (0.603)	0.588 (0.074)	0.941 (**<0.001**)									
E_2 /a/	-0.277 (0.438)	0.079 (0.828)	0.591 (0.072)	0.549 (0.100)								
E_2 /i/	-0.306 (0.389)	-0.194 (0.591)	0.104 (0.775)	0.154 (0.671)	0.771 (**0.009**)							
E_3 /a/	0.306 (0.424)	0.296 (0.440)	-0.132 (0.734)	-0.241 (0.532)	0.252 (0.513)	0.370 (0.327)						
E_3 /i/	0.140 (0.720)	0.057 (0.884)	-0.332 (0.382)	-0.413 (0.270)	0.074 (0.849)	0.440 (0.236)	0.859 (**0.003**)					
N_{loc}	-0.500 (0.141)	0.234 (0.515)	0.031 (0.932)	0.142 (0.695)	0.453 (0.188)	0.623 (0.054)	0.255 (0.508)	0.388 (0.302)				
N_{size}	-0.375 (0.286)	-0.005 (0.989)	-0.455 (0.186)	-0.453 (0.189)	-0.037 (0.920)	0.311 (0.381)	0.557 (0.119)	0.783 (**0.013**)	0.544 (0.104)			
Eff	-0.216 (0.549)	-0.164 (0.650)	-0.038 (0.917)	0.149 (0.681)	-0.137 (0.705)	-0.238 (0.508)	-0.667 (**0.050**)	-0.753 (**0.019**)	-0.233 (0.517)	-0.625 (0.053)		
Flow	-0.026 (0.943)	0.752 (**0.012**)	0.323 (0.363)	0.497 (0.143)	-0.095 (0.794)	0.002 (0.996)	0.095 (0.807)	0.095 (0.807)	0.299 (0.401)	0.088 (0.809)	-0.140 (0.699)	

Table 5-4. Correlations between acoustic, aerodynamic, and electromyographic measures in individuals with nodules who are singers. Associated *p*-values for the Pearson's correlations are shown in parentheses. Abbreviations are as follows: T_r, rise time (s) during vowels /a/ and /i/; E_{1-3}, sEMG at recording locations 1 - 3 in the 500 ms prior to vowel initiation (% MVC); N_{loc}, nodule location (% vocal fold length); N_{size}, nodule size (square pixels / pixels of vocal fold length); Eff, vocal efficiency (dB SLP / cm H_2O); Flow, glottal airflow magnitude (L / s). Bold text highlights the parameters for which a correlation significant at the $p \leq 0.05$ level was found.

Discussion

Anterior neck sEMG and Acoustic Rise Time do Not Differentiate Individuals with Nodules from those with Healthy Normal Voice

No differences were seen in the mean anterior neck sEMG at any of the three recording positions during the entire extent of all vocal tasks. When examined during the shorter time-scale surrounding vowel initiation and termination, some interesting trends can be observed in the anterior neck sEMG activity at all 3 recording locations (see Figure 5-2) indicating that perhaps some non-singers with nodules are recruiting neck musculature during vowel production to a greater degree than controls. However, only the anterior neck sEMG at recording position 3 prior to the vowel /a/ showed a statistically significant effect of group ($p = 0.014$), with non-singers with nodules showing higher anterior neck sEMG than controls and singers with nodules. Although no statistically significant differences were seen in the acoustic rise time of the three groups, the acoustic rise time measure for both vowels trended toward smaller values for non-singers with nodules relative to the singers with nodules and with controls (see Figure 5-4).

These results indicate that neither of these two objective measures show specificity for the presence of nodules in singers or non-singers. This is consistent with the findings of previous work to find objective measures for vocal hyperfunction by Hillman and colleagues (1989), who found that ratios of glottal resistance, vocal efficiency, and the AD-DC ratio (ratio of the alternating airflow to the constant airflow through the glottis) rarely detected vocal dysfunction. They attributed the failure of these individual measures in part to the large range of variability seen in normal controls (i.e., Holmberg, Hillman, & Perkell, 1988). This is consistent with the large degree of variability seen here in both individuals with nodules and controls (cf. Figure 5-2).

However, studies of current indicators of vocal hyperfunction suggest that individuals without voice disorders may also display symptoms of vocal hyperfunction, such as compression during endoscopy (Behrman, et al., 2003; Stager, et al., 2000; Stager, et al., 2003) and hard glottal attack (cf. Colton & Casper, 1996, pp., pg 80). Although behavior associated with vocal hyperfunction may be present in a vocally normal population, this does not necessarily exclude them as valuable measures. Vocal habits may lead to and prolong a voice disorder in one individual where another remains vocally healthy. Other factors, such as vocal hygiene, hydration, age, sex, and genetics, may influence vocal health in addition to behavioral mechanisms. For instance, certain proteins of the basement membrane zone are genetically influenced, leading some researchers to hypothesize that selected individuals may be predisposed to have particular voice disorders, such as nodules (Gray, 2000). The existence of these other factors does not suggest that vocally-abusive behaviors are not important to target and monitor during

therapy; in fact, for individuals predisposed to the formation and maintenance of nodules, behavior may be the one element amenable to modification that could lead to improved vocal health. One further possibility is that symptoms of vocal hyperfunction in vocally normal individuals will eventually result in a voice disorder.

Differences between Singers and Non-Singers with Nodules

No significant differences in airflow, vocal efficiency, nodule size, or nodule location were seen between singers and non-singers with nodules (see Figure 5-5). There were, however, notable trends for non-singers with nodules to have larger nodule size and greater airflow relative to singers. These trends are consistent with the work of Peppard et al. (1988) who found significantly higher airflow in non-singers with nodules relative to singers with nodules, and also significantly larger nodules in non-singers relative to singers (as measured with a qualitative 6-point scale).

Nodule location in the two groups ranged between 38% - 48% of vocal fold length (more anterior), with no obvious trend for a difference between singers and non-singers. This does not agree with the observations of Sedláčková (1961), who postulated that the nodules of singers were located more anteriorly than those of non-singers.

Measures of glottal airflow were consistently higher than values seen in a normal population (RANGE = 0.09 -0.2 L/s; Holmberg, et al., 1988); however, most values for glottal airflow were somewhat lower than those seen previously in two individuals with nodules, who reported with airflow values of 0.41 L/s and 0.49 L/s (Hillman, et al., 1989). Measures of vocal efficiency can be estimated for the two individuals with nodules studied by Hillman et al. (1989) as 8.5 dB SPL / cm H_2O and 8.6 dB SPL / cm H_2O, based on their reported sound pressure levels and subglottic pressures. These values compare well with the values seen in our participants.

Correlations among vowel rise time measures, anterior neck sEMG prior to vowel production, airflow, vocal efficiency, nodule location, and nodule size in all individuals with nodules (singers and non-singers) showed significant correlations between anterior neck sEMG measures, and also between airflow and nodule size. The correlations among anterior neck sEMG measures indicate that individuals who use extrinsic laryngeal musculature and SCM prior to voicing tend to do so non-specifically. Their tendency appears to be to employ muscles beneath all of the recording locations during voicing, rather than just particular areas. The significant correlation between airflow and nodule size ($R = 0.62$) is consistent with the previous finding of Peppard et al. (1988), that airflow values were significantly correlated with subjective ratings of nodule mass viewed during endoscopy ($R = 0.45$).

When correlations among measures were examined separately in non-singers and singers, there were a few interesting differences. In non-singers, the anterior neck sEMG at electrode 3 (SCM) prior to the vowel /a/ was correlated with vocal efficiency ($R = 0.82$) and airflow ($R = -0.94$). Further, airflow was correlated with nodule size and vocal efficiency. One interpretation of this set of correlations is that in non-singers, there is less efficient compensation for glottal insufficiency. Specifically, non-singers show a high correlation between airflow and nodule size ($R = 0.71$), indicating that the size of

the nodule is a major factor in glottal closure. Further, airflow is well-correlated with vocal efficiency (R = -0.79), suggesting that non-singers may be using increasingly inappropriately high subglottal pressures to achieve conversational sound pressure levels in cases of increased airflow. The fact that the anterior neck sEMG at electrode 3 (SCM) prior to the vowel /a/ was correlated with vocal efficiency (R = 0.82) and airflow (R = -0.94) suggests that less vocally impaired individuals may use more anterior neck sEMG prior to production of /a/.

In singers, significant correlations were found between the anterior neck sEMG at electrode 3 (SCM) prior to the vowel /i/ and nodule size, sEMG at electrode 3 (SCM) prior to the vowels /a/ and /i/ and vocal efficiency, and airflow and the linear rise time for /i/. Some of the differences in correlations between singers and non-singers indicate that there may be more efficient compensation for glottal insufficiency in singers. Specifically, unlike non-singers, singers showed no significant correlation between airflow and nodule size. Interestingly, a high correlation was seen between sEMG at electrode 3 (SCM) prior to the vowels /a/ and /i/ with vocal efficiency (R = -0.67 and R = -0.75, respectively), possibly suggesting that individuals with decreased vocal efficiency are using increased SCM, contrary to the pattern seen in non-singers. Further, the sEMG at electrode 3 (SCM) during the vowel /i/ was correlated with nodule size (R = 0.78), suggesting that individuals with larger nodules were more likely to attempt to compensate with the SCM. Another interesting correlation seen in singers (and not in non-singers) was the correlation between the linear rise time prior to the vowel /i/ and airflow (R = 0.75). This correlation suggests that singers who use "soft" or "easy" onset (associated with large rise times) may adapt a learned breathy style during sustained phonation, leading to increased steady-state airflow values.

One further factor that could affect correlations in singers with nodules is their typical style of singing. While singing, the classical or operatic style is associated with lower subglottal pressures and a lower closed quotient than is the musical theater style (e.g., Bjorkner, 2008; Stone, Cleveland, Sundberg, & Prokop, 2003). Supposing that these singing tendencies carry over into speech, they could affect some of the correlations noted in this group. Most obviously, style of singing offers an alternative explanation for the lack of correlation in singers between airflow and size of nodules. Many of the singers with nodules who were musical theater singers presented with the largest nodules. Given that musical theater singers typically use a more pressed voice, this underlying relationship could have offset the expected correspondence between airflow and size of nodules, and could explain the lack of correlation between the two seen here in singers.

USE OF OBJECTIVE MEASURES IN CLINICAL ASSESSMENT OF VOCAL HYPERFUNCTION

Much of the study of vocal hyperfunction has focused on identifying specific measures that may differentiate between disordered individuals and healthy controls. However, the clinical need is not for vocal hyperfunction assessment to aid in diagnosis of voice disorders, since the primary indication of a voice disorder is that the patient feels that he or she is being limited due to his or her voice; instead there is a need for relatively non-invasive objective clinical assays of vocal hyperfunction to allow for repeated assessment

throughout voice therapy treatment in order to identify progress. Future work should monitor possible objective measures of poor vocal behaviors over the course of voice therapy through rehabilitation, to ascertain whether they are effective at identifying successful voice therapy intervention.

Conclusions

Overall, anterior neck sEMG during vocal tasks and acoustic rise time measures did not differentiate singers or non-singers with nodules from healthy controls, indicating that neither of these two proposed objective measures show specificity for the presence of nodules in singers or non-singers. When examined during the shorter time-scale surrounding vowel initiation and termination, the sEMG recorded from the SCM (recording position 3) prior to the vowel /a/ showed a statistically significant effect of group ($p = 0.014$), with non-singers with nodules showing higher sEMG than controls and singers with nodules.

No significant differences in airflow, vocal efficiency, nodule size, or nodule location were seen between singers and non-singers with nodules. Correlations among vowel rise time measures, anterior neck sEMG prior to vowel production, airflow, vocal efficiency, nodule location, and nodule size in all individuals with nodules (singers and non-singers) showed significant correlations between anterior neck sEMG measures, and also between airflow and nodule size. When correlations among measures were examined separately in non-singers and singers, some differences were seen. Some of the differences in correlations between singers and non-singers indicate that there may be more efficient compensation for glottal insufficiency in singers. Specifically, in singers, no significant correlation was seen between airflow and nodule size, whereas non-singers showed a high correlation between airflow and nodule size ($R = 0.71$), indicating that the size of the nodule was a major factor in glottal closure.

Future work is necessary to monitor possible objective measures of vocal hyperfunction over the course of voice therapy to determine their usefulness as markers of successful voice rehabilitation.

Chapter 6:

Use of Neck Strap Muscle Intermuscular Coherence as an Indicator of Vocal Hyperfunction

Intermuscular coherence in the beta band was explored as a possible indicator of vocal hyperfunction, a common condition associated with many voice disorders. Surface electromyography (sEMG) was measured from two electrodes on the anterior neck surface of 18 individuals with vocal nodules and 18 individuals with healthy normal voice. Coherence was calculated from sEMG activity gathered while participants produced both read and spontaneous speech. There was no significant effect of speech type on average coherence. Individuals with vocal nodules showed significantly lower mean coherence in the beta band (15 – 35 Hz) when compared to controls. Results suggest that bilateral EMG-EMG beta coherence in neck strap muscle during speech production shows promise as an indicator of vocal hyperfunction.

Introduction

Disorders of the glottis (the area of the vocal folds) are often caused by or accompanied by maladaptive behaviors referred to collectively as vocal hyperfunction. Vocal hyperfunction has been defined as "conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or 'imbalanced' muscular forces" (Hillman, et al., 1989), characterized by excessive laryngeal and paralaryngeal tension (Aronson, 1980; Dworkin, Meleca, & Abkarian, 2000; Koufman & Blalock, 1991b; M. D. Morrison, et al., 1983; N. Roy, et al., 1996). Despite the widespread use of the vocal hyperfunction designation, diagnosis and assessment in current clinical practice is dependent upon subjective interpretation of patient history and physical examination. There is currently no established objective measure for the detection of vocal hyperfunction.

Past attempts to develop such measures have included the investigation of acoustic and aerodynamic parameters, both individually and in combination. Although strain is likely the most common auditory-perceptual quality attributed to vocal hyperfunction, there is no known good acoustic correlate (e.g., Colton, et al., 2006). In healthy normal speakers, aerodynamic-acoustic measures have been shown to be correlated with categorical perceptual ratings of "pressed" voice, a voice quality modality associated with vocal hyperfunction (Grillo & Verdolini, 2008). Further, Hillman and colleagues (1989) investigated aerodynamic and aerodynamic-acoustic measures (ratios of glottal resistance, vocal efficiency, and the ratio of the alternating airflow to the constant airflow through the glottis), finding that they could discriminate between individuals with different manifestations of vocal hyperfunction and individuals with healthy normal voice. However, the presence of laryngeal pathology in some of the patients studied (e.g., vocal fold nodules) causes glottal insufficiency that can impact aerodynamic measures, regardless of the presence of vocal hyperfunction, making it impossible to differentiate such effects from the separate influence of vocal hyperfunction.

Common benign organic pathologies that arise on the vocal fold surface such as vocal nodules, diffuse erythema and edema, and polyps are assumed to be related to hyperfunctional behavior (vocal hyperfunction) or phonotrauma (Hillman, et al., 1990). However, in most cases seen clinically, it is unclear to what degree the organic pathology is a result of learned hyperfunctional behaviors and to what degree the vocal hyperfunction is a compensatory result of the glottal insufficiency caused by the organic pathology. Regardless, these organic pathologies are associated with vocal hyperfunction. A common manifestation of these organic pathologies is the vocal fold nodule. Clinically, a nodule is defined as a small protuberance located between the anterior and middle third of the vocal fold (Aronson, 1980; Marcotullio, et al., 2002). It is a buildup of fibrotic tissue on the surface of the vocal fold. Vocal nodules usually occur in young to mid-aged females (M. D. Morrison, et al., 1986; M. D. Morrison, et al., 1983), and also seem to be more common in young larynges (Lee & Son, 2005). Vocal hyperfunction is a common phenomenon associated with vocal nodules, with one voice

clinic reporting that 92% of cases of vocal nodules were coincident with vocal hyperfunction (M. D. Morrison, et al., 1983).

Commonly associated symptoms of vocal hyperfunction are not limited to the larynx. Many muscles in the neck that attach to the larynx and/or hyoid bone have voice and speech-related contractions due to their role in controlling the vertical position of the larynx in the neck and, to some degree, the position of the tongue. When individuals demonstrate an inappropriate degree of intrinsic laryngeal muscle contraction (hyperfunction), it is thought that they often simultaneously contract the extrinsic laryngeal muscles and other superficial neck muscles in a similar hyperfunctional or imbalanced manner (Aronson, 1980). However, with the exception of palpation-based measures (e.g., Angsuwarangsee & Morrison, 2002; e.g., Mathieson, et al., 2009), little has been done to attempt to derive a measure of vocal hyperfunction from these known symptoms.

Two past studies have attempted to use surface electromyography (sEMG) to objectively quantify neck muscle tension. Redenbaugh and Reich (1989) measured mean neck sEMG of a single anterior neck electrode in 7 individuals with healthy normal voice and 7 "hyperfunctional" individuals, finding that the individuals with disordered voice had significantly greater mean normalized neck sEMG during phonation than individuals with healthy normal voice. However, the disordered population was varied in age, sex, and clinical presentation, and data collection method was relatively rudimentary; sEMG signals were amplified, filtered, and integrated in real-time, with the integrated values displayed on-screen and recorded by hand. Hocevar-Boltezar et al. (1998) recorded sEMG from 18 pairs of differential electrodes on the face and anterior neck in 11 women with disorders associated with vocal hyperfunction (nodules, muscle tension dysphonia) with respect to 5 women with healthy normal voice. Although this study found significant differences between the mean sEMG of many electrode positions in the two groups, sEMG signals were not normalized. In order to reduce the variability due to neck surface electrode contact and participant neck mass, sEMG signals should be normalized to a reference contraction before they are compared among conditions and/or participants (Netto & Burnett, 2006), a difficult task when assessing speech musculature. Both of these studies were limited to RMS analysis, without an attempt to assess the patterns of sEMG between electrode positions.

Given the high number of degrees of freedom involved in speech motor control and the problematic nature of appropriate amplitude normalization of sEMG data, intermuscular coherence may provide a reliable objective measure of vocal hyperfunction based on the activity of the extrinsic laryngeal muscles. Although a few studies have employed physiological coherence in the study of speech and voice (Caviness, Liss, Adler, & Evidente, 2006; Denny & Smith, 1992; A. Smith & Denny, 1990), the measure has not been widely explored.

Although the rhythmic nature of muscle discharge has been appreciated for some time (see Grosse, Cassidy, & Brown, 2002 for review), one aim of recent investigations has been to determine whether patterns of physiological drives to muscle are diagnostically relevant. Use of the coherence function has been used extensively to assess the oscillatory coupling between the central nervous system and EMG by computing coherence between EMG and magnetoencephalographic (MEG) signals (e.g., Brown,

Salenius, Rothwell, & Hari, 1998; Salenius, Portin, Kajola, Salmelin, & Hari, 1997) and between EMG and electroencephalographic (EEG) signals (e.g., Mima, Matsuoka, & Hallett, 2001; e.g., Riddle & Baker, 2005). Further, coherence between multiple EMG signals can be used to measure the common presynaptic drive to motor neurons (Brown, Farmer, Halliday, Marsden, & Rosenberg, 1999).

The coherence function, written as $|R_{xy}(\lambda)|^2$, is a frequency domain measure of the linear dependency or strength of coupling between two processes – here, two time-series x(t) and y(t) as a function of the frequency, λ. The coherence function is mathematically bounded from 0 to 1, with 0 representing independence, and 1 indicating a perfect linear relationship (Halliday, et al., 1995). This function is defined by Equation 6-1, as in (Amjad, Halliday, Rosenberg, & Conway, 1997; Halliday, et al., 1995; Rosenberg, Amjad, Breeze, Brillinger, & Halliday, 1989), where d_y^T denotes the finite Fourier transform of the ℓth segment of length T (ℓ=1,..., L) of y(t) in which the dependency on ℓ has been removed by allowing T to approach infinity, and corr{a, b} indicates the correlation between a and b.

$$|R_{xy}(\lambda)|^2 = \lim_{T\to\infty} |\mathrm{corr}\{d_x^T(\lambda), d_y^T(\lambda)\}|^2$$

Equation 6-1

One commonly studied frequency band is the beta band (15 –35 Hz) which is thought to originate chiefly from the primary motor cortex (Grosse, et al., 2002). Beta band coherence is thought to represent transmission from the primary motor cortex to spinal motoneurons, with cortical-muscle interactions following a rough somatotopic map in the pimary motor cortex (Salenius, et al., 1997). Significant beta coherence has been found in trunk muscles (paraspinal and abdominal) as well as limb muscles (which have been studied more extensively), although trunk muscle coherence modulation by the CNS is weaker and may be bilateral (Murayama, Lin, Salenius, & Hari, 2001). Although efferent pathways may be the primary source of beta band coherence, several studies argue for a role of sensory feedback using evidence from cooling, anesthesia, and short-term ischaemic sensory deafferention (Fisher, Galea, Brown, & Lemon, 2002; Pohja & Salenius, 2003; Riddle & Baker, 2005) and from a deafferented individual (Kilner, Fisher, & Lemon, 2004).

Intermuscular coherence in the beta range is thought to arise from common presynaptic motorneuron drive (Farmer, Bremner, Halliday, Rosenberg, & Stephens, 1993; Kilner, et al., 1999). Brown and colleagues validated the idea that beta band intermusuclar coherence is qualitatively similar to beta band corticomuscular coherence, testing individuals with cortical myoclonus (Brown, et al., 1999). However, unlike corticomuscular coherence, intermuscular coherence methods will reflect all oscillatory presynaptic drives to spinal motoneurons, not just those of cortical origin.

This study marks the first one of its kind in ascertaining normal bilateral EMG-EMG coherence in neck strap muscle during speech production, as well as comparing that activity between healthy normal speakers and individuals with a vocal hyperfunction. This parameter may be useful as a marker of vocal hyperfunction for use as a clinical tool.

Methods

PARTICIPANTS

Participants were 18 adult females diagnosed with vocal fold nodules prior to any therapeutic intervention (mean age = 26.1 years, SD = 10.7 years) and 18 female volunteers with healthy normal voice (mean age = 24.2 years, SD = 3.1 years). The diagnosis of vocal fold nodules in disordered individuals was based on visual examination using digital videoendoscopy with stroboscopy by a team comprised of a laryngologist and one or more certified speech-language pathologists. None of these participants had a history of any other voice disorder (e.g., vocal fold paralysis, laryngeal cancer). The individuals with healthy normal voice were volunteers with no complaints related to their voice who had no abnormal pathology of the larynx as observed during standard digital videoendoscopy with stroboscopy. Informed consent was obtained from all participants in compliance with the institutional review board of the Massachusetts General Hospital.

TASKS

Simultaneous neck sEMG and acoustic signals from a lavalier microphone (Sennheiser MKE2-P-K, Wedemark, Germany) were filtered and recorded digitally with Delsys ™ (Boston, Massachusetts) hardware (Bagnoli Desktop System) and software (EMGworks 3.3) with a sampling frequency of 20 kHz. The EMG recordings in this study were taken in view of current European standards (Hermens, et al., 1999). Participants' necks were prepared for electrode placement by cleaning the neck surface with an alcohol pad and "peeling" with tape to reduce electrode-skin impedance, noise, DC voltages, and motion artifacts. The neck sEMG was recorded using two 2-channel Bagnoli systems (DelSys ™ Inc.) with two double differential electrodes placed parallel to the underlying muscle fibers. Double differential electrodes were utilized to increase spatial selectivity and to prevent electrical cross-talk between the two electrodes. Electrode 1 was placed superficial to fibers of the thyrohyoid, omohyoid, and sternohyoid muscles. Electrode 2 was placed on the contralateral side superficial to the cricothyroid and sternohyoid muscles; however, based on examination of the sEMG during pitch glides, it is unlikely that any activation from the (deeper) cricothyroid was detected. The Delsys ™ 3.1 double differential surface electrodes consist of three 10-mm silver bars with inter-electrode distances of 10-mm. Electrode 1 was centered about 1 cm lateral to the neck midline, as far superior as was possible without impeding the jaw opening of the participant. Electrode 2 was centered on the gap between the cricoid and thyroid cartilages of the larynx, and centered at 1 cm lateral to the midline contralateral to Electrode 1. A schematic indicating the locations of these electrodes is shown in Figure 6-1. A ground electrode was placed on the superior aspect of the participant's left

shoulder. The EMG recordings were pre-amplified and filtered using Delsys™ Bagnoli systems set to a gain of 1000 and a band-pass filter with roll-off frequencies of 20 Hz and 450 Hz.

The recording procedure consisted of a brief vocal assessment of the participant including both read and spontaneous running speech. The read passage was the first paragraph of the Rainbow Passage, (Fairbanks, 1960). Spontaneous speech was elicited in response to a prompt from the experimenter to describe their voice issues (in the nodule group), their travel to the facility for the experiment, their job, or a recent trip or holiday experience (e.g., "Can you tell me about your voice issues?"). Recordings were monitored in real-time for signal integrity, ensuring that no recordings included movement artifact or microphonic signals from voice production.

ANALYSIS

Audio signals were examined offline by using visual inspection and by listening to the audio signal to determine periods of speech production. Speech time for analysis was chosen manually from approximately 1 s before and after continuous speech, and avoiding non-speech activity such as laughing or coughing. Read passages were of mean length 31 s (R = 26 – 54 s), while spontaneous speech samples used for analysis were of more variable length (MEAN = 29, R = 8 – 93 s).

The EMG signals were full-wave rectified and any DC offset was removed from each read and spontaneous speech sample of each participant. Coherence and phase estimates were calculated over a sliding 16,384 point (~820 ms) Hamming window with a 16,384 point FFT, using 50% overlap, mimicking the methods used in Halliday et al. (1995), using custom software written in MATLAB® (Mathworks Inc., Natick, MA). For each speech sample, a 5% significance level for coherence was determined based on sample length (e.g., Halliday, et al., 1995). These values were catalogued to better reference average coherence values. A two-factor ANOVA analysis of the 5% significance level was performed by group and speech task (read and spontaneous), and showed no effect of group ($p = 0.93$), and a significant effect of speech task ($p = 0.001$), which was not surprising given the varied lengths of the spontaneous speech samples. For read speech samples, the 5% significance levels averaged 0.040 with SD = 0.005; spontaneous speech samples had 5% significance levels averaging 0.058 with SD = 0.029.

The mean of the root-mean-squared values of sEMG collected from both electrodes was computed in 1 s windows (no overlap) using custom MATLAB® (Mathworks Inc., Natick, MA) software for the entire length of the two speech tasks (read and spontaneous), as well as during a period (10 – 20 seconds) of silent resting in which no obvious sEMG activity was apparent. For each electrode, the ratio between the mean RMS during the two speech tasks and the mean RMS during the rest period was calculated as an estimate of the signal-to-noise ratio (SNR). For each participant, the absolute difference between the SNR for each electrode was determined. Pearson's correlations between these absolute differences in SNR and the coherence values during read and spontaneous speech to assess whether noise was a factor in differences in

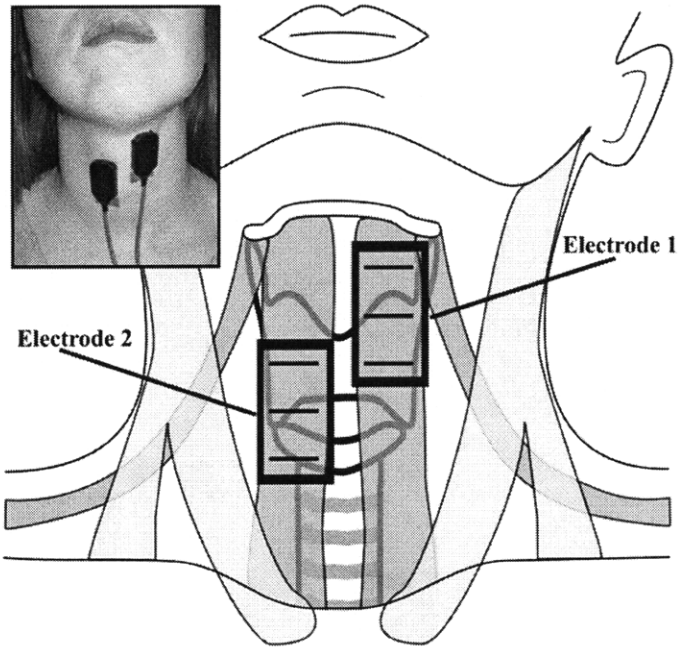


Figure 6-1. A schematic of the anterior neck with the locations of double differential sEMG locations.

coherence. Further, absolute differences in SNR in the two groups were compared using Student's t-tests,

Tests on the null hypothesis that the beta coherence (15 – 35 Hz) would be of the same level in the nodule and control groups were performed on average coherence values over the frequency range as well as Tanh-1 transformed values to account for the possibility of unstable variance (e.g., Rosenberg, et al., 1989). Statistical testing was performed by analysis of variance (ANOVA) and Student's t-tests using Minitab® Statistical Software (Minitab Inc., State College, PA).

Results

The mean coherence spectra for each group (speech task data pooled) are shown in Figure 6-2. Mean coherence was relatively high over beta and low gamma (30 – 60 Hz) frequencies for all groups relative to the 5% significance values for the speech samples compared to previous reports of bilateral intermuscular coherence for speech tasks measured in respiratory muscles and the masseter (A. Smith & Denny, 1990). No obvious peaks were seen in any frequency range. A two-factor ANOVA analysis of the average beta band coherence (15 – 35 Hz) by group and speech task (read and spontaneous) showed a statistically significant effect of group ($p = 0.001$), but no effect of speech task ($p = 0.54$); an ANOVA analysis on tanh-1 transformed values produced nearly identical results. Mean beta coherence for nodules participants was 0.14 (SD = 0.13), whereas control participants had a mean average beta coherence of 0.26 (SD = 0.16). Individual values and boxplots of the average beta coherence by group (speech task data pooled) are shown in Figure 3.

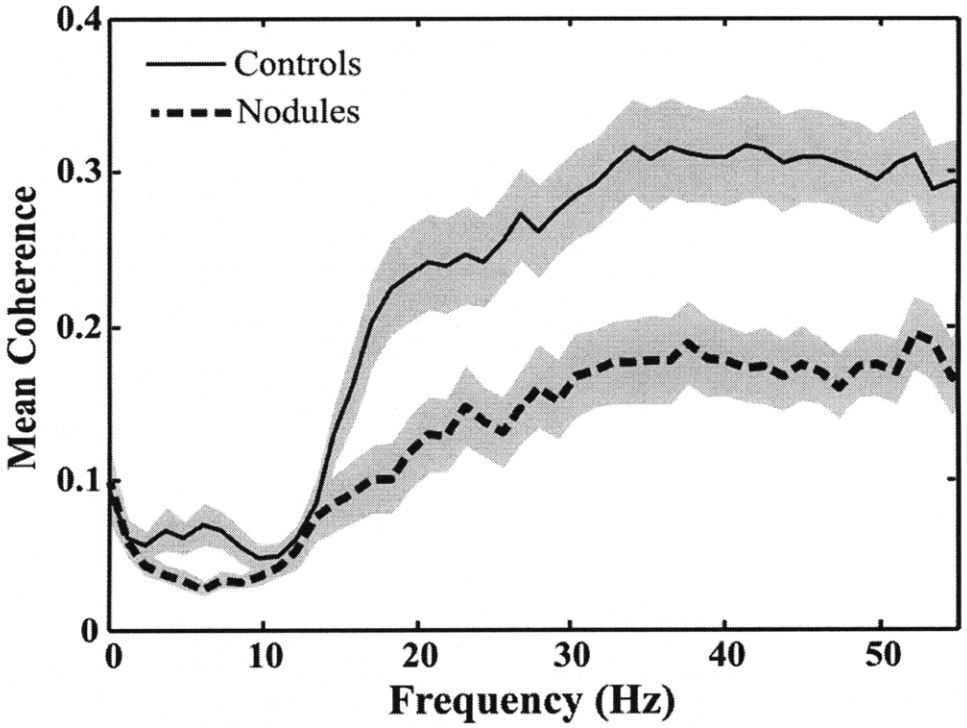


Figure 6-2. Mean coherence spectra for the two participant groups. The solid black line refers to the healthy normal controls ("Controls") and the dashed black line to the individuals with vocal nodules ("Nodules"). Grey shading indicates standard error of each group by frequency.

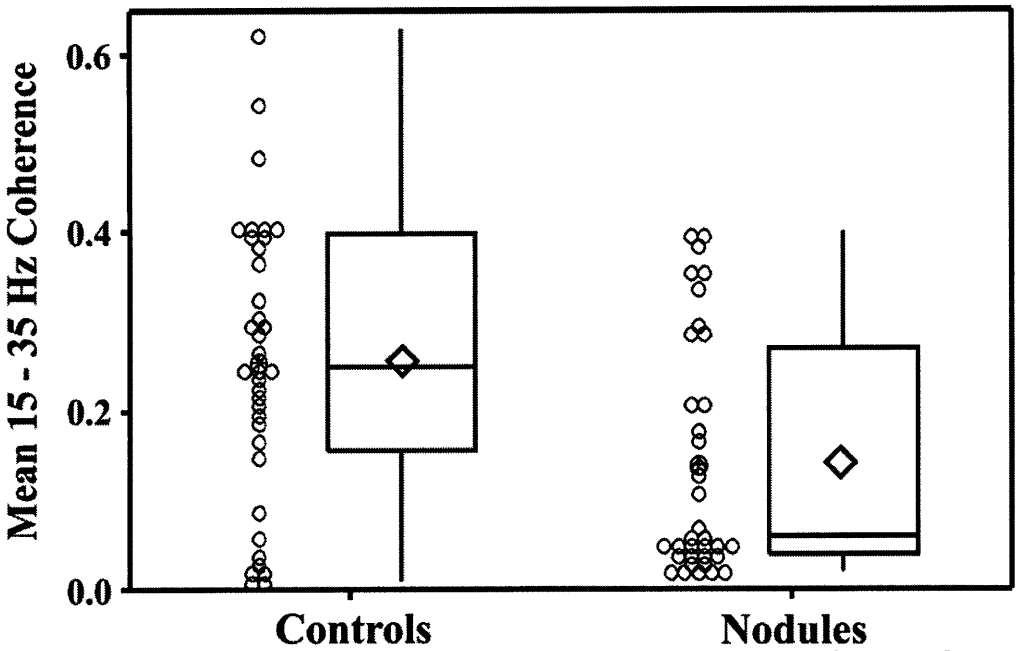


Figure 6-3. Individual values and boxplots of the mean beta coherence by group. "Controls" refers to healthy normal controls and "Nodules" to individuals with vocal nodules. Horizontal box lines indicate the lower and upper quartiles of the data, with the center line marking the data median. Diamonds show the location of the data means. Vertical whiskers extend from the boxes to the minimum and maximum values of each dataset. Individual values of coherence values are shown to the left of the boxplots with circles. ANOVA found a statistically significant difference ($p = 0.001$) between the mean beta coherence in the control and nodules groups ($p_{adj} < 0.001$).

A Student's t-test between the absolute differences in SNR in the nodule group relative to the control group did not show a significant difference (p = 0.98, 2-sided, df = 19). The average absolute difference in SNR was 9.8 (STD = 7.4) in the control group and 9.9 (STD = 25.7) in the nodules group. Neither the Pearson's correlation between the absolute differences in SNR and the coherence values during read speech (R = 0.15) nor between the absolute differences in SNR and the coherence values during spontaneous speech (R = 0.01) were statistically significant (p > 0.05).

Discussion

Oscillatory coupling in the beta band between the two neck strap muscle recording locations is relatively high for both read and spontaneous speech in healthy normal speakers, as measured by bilateral EMG-EMG coherence compared to bilateral intermuscular coherence measured in respiratory muscles and the masseter during speech tasks (A. Smith & Denny, 1990). Comparison with coherence measured in non-speech systems is difficult given the unique properties of the speech motor control system (see Kent, 2004 for review). The speech tasks used for this study are representative of the typical function of the individuals, but have little in common with the simple press and hold tasks used in previous coherence studies carried out in limbs.

Bilateral EMG-EMG Beta Band Coherence is Reduced in Individuals with Nodules Relative to Healthy Controls

In contrast to the relatively high oscillatory coupling between neck strap muscle and motor cortex seen here in healthy normal speakers, individuals with vocal nodules have reduced beta band EMG-EMG coherence for both read and spontaneous speech. This reduced coherence could be a function of a difference between the two populations in electrophysiological noise, differences in measured sEMG cross-talk, or a true difference in neural bilateral coupling. The two groups did not differ significantly in terms of the absolute differences in SNR, nor were the absolute differences in SNR significantly correlated with coherence values during read speech ($R = 0.15$) or spontaneous speech ($R = 0.01$). These findings indicate that the significant difference in coherence between the two groups was not due to a variance in SNR. Using sEMG always carries the risk of cross-talk between electrodes (e.g., Farina, Merletti, & Stegeman, 2004), which could increase coherence values. However, all participants were recorded with double differential electrodes to significantly decrease this risk (De Luca & Merletti, 1988; van Vugt & van Dijk, 2001); furthermore, it is unlikely that individuals with healthy normal voice should be any more vulnerable to crosstalk than those with vocal nodules. Reduced coherence in the individuals with vocal nodules could also correspond to reduced bilateral neural coupling, which would be consistent with the clinical impression that vocal hyperfunction may be caused by excessive and/or 'imbalanced' muscular forces (Hillman, et al., 1989). Based on the present work, the source of reduced bilateral neural coupling requires some speculation, but could implicate degraded sensory feedback, lack of cortical oversight caused by reduced attention, or overexertion.

Loss/weakness of sensory feedback leads to loss or weakness of the beta drive (e.g, Fisher, et al., 2002; Kilner, et al., 2004; e.g, Pohja & Salenius, 2003). It is possible that individuals with vocal hyperfunction have degraded sensory feedback. Altered or inappropriate sensory feedback might explain the habit of individuals with vocal

hyperfunction to use "too much" or the wrong combination of muscle activity without noticing. In fact, modification of sensory feedback has also been used to treat therapy-resistant vocal hyperfunction. Dworkin and colleagues (2000) applied topical lidocaine to three individuals causing near-immediate resolution of their functional dysphonia. To our knowledge, sensory feedback has not yet been studied systematically in individuals with vocal hyperfunction. This work provides further justification for such study.

Beta band coherence also appears to be modulated by the precision required for a task and the amount of attention used. Kristeva-Feige and colleagues saw a reduction in the beta range EEG-EMG coherences during an isometric constant force task when the task required less precision and also when a high precision task was performed while the subject divided his or her attention from the motor task by doing mental arithmetic (Kristeva-Feige, Fritsch, Timmer, & Lucking, 2002). Beta coherence also increases with visuo-motor learning, with increases not necessarily related to task performance (Perez, Lundbye-Jensen, & Nielsen, 2006). Kristeva-Feige et al. postulate that beta drive increases with learning or task precision may be due to tighter cortical control. If so, this suggests that individuals with vocal hyperfunction may have reduced cortical control over their vocal anatomy.

Individuals with vocal hyperfunction may be overusing their neck strap muscles for long periods of time, resulting in general fatigue of the vocal system, which could in turn modulate the intermuscular coherence. The effect of fatigue on beta coherence is still not completely clear. Tecchio and colleagues measured hand EMG-MEG coherence before and after a fatiguing motor task in fourteen individuals, finding increased beta coherence post-fatigue (Tecchio, et al., 2006). However, more recently, Yang et al. (2008) measured EMG-EEG coherence during the first and second halves of a fatiguing task in nine individuals, finding that the beta coherence was reduced during the second half of the task.

In their study of sEMG of the masseter, Smith and Denny (1990) found that the coherence between right and left muscles showed a large degree of intersubject variability for speech tasks when compared to the more consistent coherence patterns during chewing and jaw clenching. Determining the clinical utility of neck strap muscle coherence in individuals with voice disorders is dependent upon more complete understanding of the possible inter- and intrasubject variability of this speech-related coherence in the healthy normal population, which could vary widely as a function of time and participant. Normative studies over days and weeks are necessary to more fully understand this promising measure.

Speech type has No Effect on Bilateral EMG-EMG Beta Band Coherence

Whether the speech sample was read or spontaneous did not affect the mean beta coherence. While Fitch (Fitch, 1990) did not show significant differences in the average fundamental frequency of spontaneous and read speech in healthy normal participants, speech type may have an effect on speech respiration. Specifically, respiratory variables such as syllables per breath group show larger differences between healthy individuals

with normal voice and individuals with vocal nodules when measured using spontaneous speech tasks than with read speech (Iwarsson & Sundberg, 1999). Our investigation, however, did not show a significant difference between these two speech tasks, demonstrating that the differences among groups seen in read speech were replicated in the recordings of spontaneous speech. This may indicate that the degraded coherence in the participants with vocal hyperfunction is a resilient feature of their speech production, rather than a mere by-product of compensation techniques or attention that may differ with changes in linguistic planning between read and spontaneous speech.

SUMMARY AND INDICATIONS FOR FUTURE WORK

This is the first work to assess normal bilateral EMG-EMG coherence in neck strap muscle during speech production, as well as to compare that activity between healthy normal speakers and individuals with vocal nodules. In individuals with healthy normal voice, mean coherence was relatively high over in the beta band (MEAN = 0.26) for both speech types. Individuals with vocal nodules showed significantly lower mean coherence in the beta band (MEAN = 0.14) when compared to controls. There was no significant effect of speech type on average coherence. Results are consistent with previous hypotheses describing vocal hyperfunction as the use of 'imbalanced' muscular forces, and suggest that bilateral EMG-EMG beta coherence in neck strap muscle during speech production shows promise as an indicator of vocal hyperfunction for use as a clinical tool.

Patients with hyperfunctionally related disorders such as vocal nodules are typically offered voice therapy that is designed to reduce vocal hyperfunction. Future studies monitoring bilateral EMG-EMG coherence in vocal hyperfunction patients across the course of voice therapy are needed to determine whether this measure correlated with rehabilitative outcomes. Investigations are also needed to determine the sensitivity and specificity of this measure when compared to current methods of assessment. Further scientific study is also warranted to elucidate the possible factors affecting bilateral anterior neck EMG-EMG coherence such as sensory feedback, precision, attention, and fatigue.

Summary

1. Injection laryngoplasty does not offer a suitable platform for the study of all types of vocal hyperfunction, likely due to the structural pathologies limiting vocal fold adduction in this population.

2. Current schemes for the quantification of supraglottic compression do not correspond with perceptual measures. Their use is not recommended.

3. Vocal fold kinematic estimators of muscle stiffness show promise as objective indicators of vocal hyperfunction.

4. Current neck tension palpation rating scales offer unreliable and thus invalid assessment of neck muscle tension. Furthermore, there is no evidence that they correspond with RMS anterior neck sEMG.

5. Although a select number of individuals with vocal hyperfunction may display patterns of anterior neck RMS sEMG unlike those seen in controls, RMS sEMG measures do not discriminate between individuals with vocal fold nodules and controls, nor do changes in RMS sEMG measures correspond with changes in more established estimators of vocal hyperfunction in a population of individuals undergoing injection laryngoplasty.

6. Compared with controls, individuals with vocal fold nodules show significantly reduced bilateral EMG-EMG coherence in neck strap muscles during speech.

References

Alipour, F., Berry, D. A., & Titze, I. R. (2000). A finite-element model of vocal-fold vibration. *Journal of the Acoustical Society of America, 108*(6), 3003-3012.

Alipour, F., Titze, I. R., Hunter, E., & Tayama, N. (2005). Active and passive properties of canine abduction/adduction laryngeal muscles. *Journal of Voice, 19*(3), 350-359.

Allison, G. T., Marshall, R. N., & Singer, K. P. (1993). EMG Signal Amplitude Normalization Technique in Stretch-shortening Cycle Movements. *J Electromyogr Kinesiol, 3*(4), 236-244.

Altman, K. W., Atkinson, C., & Lazarus, C. (2005). Current and emerging concepts in muscle tension dysphonia: a 30-month review. *J Voice, 19*(2), 261-267.

Amjad, A. M., Halliday, D. M., Rosenberg, J. R., & Conway, B. A. (1997). An extended difference of coherence test for comparing and combining several independent coherence estimates: theory and application to the study of motor units and physiological tremor. *J Neurosci Methods, 73*(1), 69-79.

Andrade, D. F., Heuer, R., Hockstein, N. E., Castro, E., Spiegel, J. R., & Sataloff, R. T. (2000). The frequency of hard glottal attacks in patients with muscle tension dysphonia, unilateral benign masses and bilateral benign masses. *J Voice, 14*(2), 240-246.

Andrews, M. L. (1999). *Manual of Voice Treatment* (2nd ed.). San Diego, CA: Singular Publishing Group, Inc.

Andrianopoulos, M. V., Gallivan, G. J., & Gallivan, K. H. (2000). PVCM, PVCD, EPL, and irritable larynx syndrome: what are we talking about and how do we treat it? *J Voice, 14*(4), 607-618.

Angsuwarangsee, T., & Morrison, M. (2002). Extrinsic laryngeal muscular tension in patients with voice disorders. *J Voice, 16*(3), 333-343.

Arnold, G. E. (1962). Vocal nodules and polyps: laryngeal tissue reaction to habitual hyperkinetic dysphonia. *Journal of Speech and Hearing Disorders, 27*, 205-217.

Aronson, A. E. (1980). *Clinical Voice Disorders: An Interdisciplinary Approach* (1 ed.). New York: Thieme-Stratton, Inc.

Atkinson, J., & Erickson, D. (1976, November 16-19, 1976). *The function of strap muscles in speech: pitch lowering or jaw opening.* Paper presented at the 92nd Meeting: Acoustical Society of America, San Diego, CA.

Atkinson, J. E. (1978). Correlation analysis of the physiological factors controlling fundamental voice frequency. *J Acoust Soc Am, 63*(1), 211-222.

Baer, T., Gay, T., & Niimi, S. (1976). Control of Fundamental Frequency, Intensity, and Register of Phonation. *Haskins Laboratories: Status Report on Speech Research, SR-45/46*, 175-185.

Behrman, A., Dahl, L. D., Abramson, A. L., & Schutte, H. K. (2003). Anterior-posterior and medial compression of the supraglottis: signs of nonorganic dysphonia or normal postures? *J Voice, 17*(3), 403-410.

Benecke, R., Rothwell, J. C., Dick, J. P., Day, B. L., & Marsden, C. D. (1986). Performance of simultaneous movements in patients with Parkinson's disease. *Brain, 109 (Pt 4)*, 739-757.

Bhalla, R. K., Wallis, J., Kaushik, V., & Carpentier, J. P. (2005). How we do it: adjunctive intravenous midazolam: diagnosis and treatment of therapy-resistant muscle tension dysphonia. *Clin Otolaryngol, 30*(4), 367-369.

Bizzi, E., Accornero, N., Chapple, W., & Hogan, N. (1984). Posture control and trajectory formation during arm movement. *Journal of Neuroscience, 4*(11), 2738-2744.

Bjorkner, E. (2008). Musical theater and opera singing--why so different? A study of subglottal pressure, voice source, and formant frequency characteristics. *J Voice, 22*(5), 533-540.

Boersma, P., & Weenink, D. (2008). Praat: doing phonetics by computer (Version 5.0.20): http://www.praat.org/.

Bridger, M. W., & Epstein, R. (1983). Functional voice disorders. A review of 109 patients. *J Laryngol Otol, 97*(12), 1145-1148.

Brown, P., Farmer, S. F., Halliday, D. M., Marsden, J., & Rosenberg, J. R. (1999). Coherent cortical and muscle discharge in cortical myoclonus. *Brain, 122 (Pt 3)*, 461-472.

Brown, P., Salenius, S., Rothwell, J. C., & Hari, R. (1998). Cortical correlate of the Piper rhythm in humans. *J Neurophysiol, 80*(6), 2911-2917.

Carding, P. N., & Horsley, I. A. (1992). An evaluation study of voice therapy in non-organic dysphonia. *Eur J Disord Commun, 27*(2), 137-158.

Carding, P. N., Horsley, I. A., & Docherty, G. J. (1999). A study of the effectiveness of voice therapy in the treatment of 45 patients with nonorganic dysphonia. *J Voice, 13*(1), 72-104.

Caviness, J. N., Liss, J. M., Adler, C., & Evidente, V. (2006). Analysis of high-frequency electroencephalographic-electromyographic coherence elicited by speech and oral nonspeech tasks in Parkinson's disease. *J Speech Lang Hear Res, 49*(2), 424-438.

Choi, H. S., Berke, G. S., Ye, M., & Kreiman, J. (1993). Function of the posterior cricoarytenoid muscle in phonation: in vivo laryngeal model. *Otolaryngol Head Neck Surg, 109*(6), 1043-1051.

Cohen, S. M., & Garrett, C. G. (2007). Utility of voice therapy in the management of vocal fold polyps and cysts. *Otolaryngol Head Neck Surg, 136*(5), 742-746.

Collier, R. (1974). Laryngeal Muscle Activity, Subglottal Air Pressure, and the Control of Pitch. *Haskins Laboratories: Status Report on Speech Research, SR-39/40*, 137-170.

Colton, R. H., & Casper, J. K. (1996). *Understanding Voice Problems: A Physiological Perspective for Diagnosis and Treatment* (2nd ed.). Baltimore, MD: Williams & Wilkins.

Colton, R. H., Casper, J. K., & Leonard, R. (2006). *Understanding voice problems: a physiological perspective for diagnosis and treatment* (3rd ed.). Baltimore, MD: Lippincott Williams & Wilkins.

Cooke, A., Ludlow, C. L., Hallett, N., & Selbie, W. S. (1997). Characteristics of Vocal Fold Adduction Related to Voice Onset. *Journal of Voice, 11*(1), 12-22.

Courey, M. S. (2004). Injection laryngoplasty. *Otolaryngol Clin North Am, 37*(1), 121-138.

Dailey, S. H., Kobler, J. B., Hillman, R. E., Tangrom, K., Thananart, E., Mauri, M., et al. (2005). Endoscopic measurement of vocal fold movement during adduction and abduction. *Laryngoscope, 115*(1), 178-183.

De Luca, C. J., & Merletti, R. (1988). Surface myoelectric signal cross-talk among muscles of the leg. *Electroencephalogr Clin Neurophysiol, 69*(6), 568-575.

Debruyne, F., Ostyn, F., Delaere, P., & Wellens, W. (1997). Acoustic analysis of the speaking voice after thyroidectomy. *J Voice, 11*(4), 479-482.

Debruyne, F., Ostyn, F., Delaere, P., Wellens, W., & Decoster, W. (1997). Temporary voice changes after uncomplicated thyroidectomy. *Acta Otorhinolaryngol Belg, 51*(3), 137-140.

Demmink-Geertman, L., & Dejonckere, P. H. (2002). Nonorganic habitual dysphonia and autonomic dysfunction. *Journal of Voice, 16*(4), 549-559.

Demmink-Geertman, L., & Dejonckere, P. H. (2008). Neurovegetative symptoms and complaints before and after voice therapy for nonorganic habitual dysphonia. *Journal of Voice, 22*(3), 315-325.

Denny, M., & Smith, A. (1992). Gradations in a pattern of neuromuscular activity associated with stuttering. *J Speech Hear Res, 35*(6), 1216-1229.

Dikkers, F. G., & Nikkels, P. G. (1995). Benign lesions of the vocal folds: histopathology and phonotrauma. *Ann Otol Rhinol Laryngol, 104*(9 Pt 1), 698-703.

Dikkers, F. G., & Nikkels, P. G. (1999). Lamina propria of the mucosa of benign lesions of the vocal folds. *Laryngoscope, 109*(10), 1684-1689.

Dikkers, F. G., & Schutte, H. K. (1991). Benign lesions of the vocal folds: uniformity in assessment of clinical diagnosis. *Clin Otolaryngol Allied Sci, 16*(1), 8-11.

Dworkin, J. P., Meleca, R. J., & Abkarian, G. G. (2000). Muscle tension dysphonia. *Current Opinion in Otolaryngology & Head and Neck Surgery, 8*(3), 169-173.

Dworkin, J. P., Meleca, R. J., Simpson, M. L., & Garfield, I. (2000). Use of topical lidocaine in the treatment of muscle tension dysphonia. *Journal of Voice, 14*(4), 567-574.

Erickson, D. (1977). The function of strap muscles in speech: pitch lowering or jaw opening. *Haskins Laboratories: Status Report on Speech Research, SR-49*, 97-102.

Erickson, D., Baer, T., & Harris, K. S. (1981). The Role of the Strap Muscles in Pitch Lowering. In D. M. Bless & A. J.H.A. (Eds.), *Vocal Fold Physiology*. San Diego, CA: College-Hill Press.

Estill, J., Baer, T., Honda, K., & Harris, K. S. (1983, June 5, 1983). *The control of pitch and quality. Part I and II.* Paper presented at the XIIth Symposium, Care of the Professional Voice, New York, NY.

Eustace, C. S., Stemple, J. C., & Lee, L. (1996). Objective measures of voice production in patients complaining of laryngeal fatigue. *J Voice, 10*(2), 146-154.

Faaborg-Andersen, K., & Sonninen, A. (1960). The function of the extrinsic laryngeal muscles at different pitch: an electromyographic and roentgenologic investigation. *Acta Otolaryngol, 51*, 89-93.

Fairbanks, G. (1960). *Voice and Articulation Drillbook* (2nd ed.). New York: Harper and Row.

Falla, D., Dall'Alba, P., Rainoldi, A., Merletti, R., & Jull, G. (2002). Location of innervation zones of sternocleidomastoid and scalene muscles--a basis for clinical and research electromyography applications. *Clin Neurophysiol, 113*(1), 57-63.

Fant, G. (1960). *Acoustic Theory of Speech Production*. The Hague: Mouton.

Farina, D., Merletti, R., & Disselhorst-Klug, C. (2004). Multi-channel techniques for information extraction from the surface EMG. In R. Merletti & P. Parker (Eds.), *Electromyography: Physiology, Engineering, and Noninvasive Applications* (pp. 169-203). Hoboken, NJ: John Wiley & Sons, Inc.

Farina, D., Merletti, R., & Stegeman, D. F. (2004). Biophysics of the generation of EMG signals. In R. Merletti & P. A. Parker (Eds.), *Electromyography: Physiology, Engineering, and Noninvasive Applications* (pp. 81-105). Hoboken, NJ: John Wiley & Sons, Inc.

Farina, D., & Rainoldi, A. (1999). Compensation of the effect of sub-cutaneous tissue layers on surface EMG: a simulation study. *Med Eng Phys, 21*(6-7), 487-497.

Farley, G. R. (1996). A biomechanical laryngeal model of voice F0 and glottal width control. *Journal of the Acoustical Society of America, 100*(6), 3794-3812.

Farmer, S. F., Bremner, F. D., Halliday, D. M., Rosenberg, J. R., & Stephens, J. A. (1993). The frequency content of common synaptic inputs to motoneurones studied during voluntary isometric contraction in man. *J Physiol, 470*, 127-155.

Ferrand, C. T. (2001). Speech Science: An integrated approach to theory and clinical practice.

Ferrand, C. T. (2001). *Speech Science: An Integrated Approach to Theory and Clinical Practice*. Boston, MA: Allyn & Bacon Publishers.

Fink, B. R., & Demarest, R. J. (1977). *Laryngeal Biomechanics*. Cambridge, MA: Harvard University Press.

Fink, B. R., & Demarest, R. J. (1978). Laryngeal Biomechanics.

Fisher, R. J., Galea, M. P., Brown, P., & Lemon, R. N. (2002). Digital nerve anaesthesia decreases EMG-EMG coherence in a human precision grip task. *Exp Brain Res, 145*(2), 207-214.

Fitch, J. L. (1990). Consistency of fundamental frequency and perturbation in repeated phonations of sustained vowels, reading, and connected speech. *J Speech Hear Disord, 55*(2), 360-363.

Flash, T. (1987). The control of hand equilibrium trajectories in multi-joint arm movements. *Biological Cybernetics, 57*(4-5), 257-274.

Fulton, K. S. (2007). *Vocal efficiency in trained singers vs. non-singers.* Unpublished Master of Science, Brigham Young University, Provo, UT.

Gallena, S., Smith, P. J., Zeffiro, T., & Ludlow, C. L. (2001). Effects of levodopa on laryngeal muscle activity for voice onset and offset in Parkinson disease. *J Speech Lang Hear Res, 44*(6), 1284-1299.

Goldman, S. L., Hargrave, J., Hillman, R. E., Holmberg, E. B., & Gress, C. (1996). Stress, Anxiety, Somatic Complaints, and Voice Use in Women With Vocal Nodules: Preliminary Findings. *American Journal of Speech-Language Pathology, 5*(1), 44-54.

Gray, S. D. (2000). Cellular physiology of the vocal folds. *Otolaryngol Clin North Am, 33*(4), 679-698.

Gray, S. D., Hammond, E., & Hanson, D. F. (1995). Benign pathologic responses of the larynx. *Ann Otol Rhinol Laryngol, 104*(1), 13-18.

Grillo, E. U., & Verdolini, K. (2008). Evidence for Distinguishing Pressed, Normal, Resonant, and Breathy Voice Qualities by Laryngeal Resistance and Vocal Efficiency in Vocally Trained Subjects. *J Voice, 22*(5), 546-552.

Grosse, P., Cassidy, M. J., & Brown, P. (2002). EEG-EMG, MEG-EMG and EMG-EMG frequency analysis: physiological principles and clinical applications. *Clin Neurophysiol, 113*(10), 1523-1531.

Hall, S. J. (2002). *Basic Biomechanics* (4th ed.). Boston, MA: McGraw-Hill.

Halliday, D. M., Rosenberg, J. R., Amjad, A. M., Breeze, P., Conway, B. A., & Farmer, S. F. (1995). A framework for the analysis of mixed time series/point process data--theory and application to the study of physiological tremor, single motor unit discharges and electromyograms. *Prog Biophys Mol Biol, 64*(2-3), 237-278.

Hermens, H. J., Freriks, B., Merletti, R., Stegeman, D. F., Blok, J., Gunter, R., et al. (1999). *European Recommendations for Surface ElectroMyoGraphy: Results of the SENIAM project.* Enschede: Roessingh Research and Development b.v.

Herrington-Hall, B. L., Lee, L., Stemple, J. C., Niemi, K. R., & McHone, M. M. (1988). Description of laryngeal pathologies by age, sex, and occupation in a treatment-seeking sample. *J Speech Hear Disord, 53*(1), 57-64.

Hillman, R. E., Gress, C., Hargrave, J., Walsh, M., & Bunting, G. (1990). The Efficacy of Speech-Language Pathology Intervention: Voice Disorders. *Seminars in Speech and Language, 11*(3), 297-310.

Hillman, R. E., Holmberg, E. B., Perkell, J. S., Walsh, M., & Vaughan, C. (1989). Objective assessment of vocal hyperfunction: an experimental framework and initial results. *Journal of Speech and Hearing Research, 32*(2), 373-392.

Hillman, R. E., Montgomery, W. M., & Zeitels, S. M. (1997). Current Diagnostics and Office Practice: Use of objective measures of vocal function in the multidisciplinary management of voice disorders. *Curr Opin Otolaryngol Head Neck Surg, 5*(3), 172-175.

Hirano, M. (1988). Vocal Mechanisms in Singing: Laryngological and Phoniatric Aspects. *J Voice, 2*, 51-69.

Hirano, M., Koike, Y., & von Leden, H. (1967). The sternohyoid muscle during phonation. Electromyographic studies. *Acta Otolaryngol, 64*(5), 500-507.

Hocevar-Boltezar, I., Janko, M., & Zargi, M. (1998). Role of surface EMG in diagnostics and treatment of muscle tension dysphonia. *Acta Otolaryngol, 118*(5), 739-743.

Hoffer, J. A., & Andreassen, S. (1981). Regulation of soleus muscle stiffness in premammillary cats: intrinsic and reflex components. *Journal of Neurophysiology, 45*(2), 267-285.

Hogan, N. (1984). An organizing principle for a class of voluntary movements. *Journal of Neuroscience, 4*(11), 2745-2754.

Holmberg, E. B., Hillman, R. E., Hammarberg, B., Sodersten, M., & Doyle, P. (2001). Efficacy of a behaviorally based voice therapy protocol for vocal nodules. *Journal of Voice, 15*(3), 395-412.

Holmberg, E. B., Hillman, R. E., & Perkell, J. S. (1988). Glottal airflow and transglottal air pressure measurements for male and female speakers in soft, normal, and loud voice. *J Acoust Soc Am, 84*(2), 511-529.

Honda, K., Hirai, H., Masaki, S., & Shimada, Y. (1999). Role of vertical larynx movement and cervical lordosis in F0 control. *Lang Speech, 42 (Pt 4)*, 401-411.

Hong, K. H., & Kim, Y. K. (1997). Phonatory characteristics of patients undergoing thyroidectomy without laryngeal nerve injury. *Otolaryngol Head Neck Surg, 117*(4), 399-404.

Hong, K. H., Ye, M., Kim, Y. M., Kevorkian, K. F., & Berke, G. S. (1997). The role of strap muscles in phonation--in vivo canine laryngeal model. *J Voice, 11*(1), 23-32.

Hong, K. H., Ye, M., Kim, Y. M., Kevorkian, K. F., Kreiman, J., & Berke, G. S. (1998). Functional differences between the two bellies of the cricothyroid muscle. *Otolaryngol Head Neck Surg, 118*(5), 714-722.

Hsiao, T. Y., Liu, C. M., Hsu, C. J., Lee, S. Y., & Lin, K. N. (2001). Vocal fold abnormalities in laryngeal tension-fatigue syndrome. *Journal of the Formosan Medican Association, 100*(12), 837-840.

Hsiao, T. Y., Liu, C. M., & Lin, K. N. (2002). Videostrobolaryngoscopy of mucus layer during vocal fold vibration in patients with laryngeal tension-fatigue syndrome. *Ann Otol Rhinol Laryngol, 111*(6), 537-541.

Hsiung, M. W., & Hsiao, Y. C. (2004). The characteristic features of muscle tension dysphonia before and after surgery in benign lesions of the vocal fold. *Journal for Oto-Rhino-Laryngology, Head and Neck Surgery, 66*(5), 246-254.

Hunter, E. J., Titze, I. R., & Alipour, F. (2004). A three-dimensional model of vocal fold abduction/adduction. *Journal of the Acoustical Society of America, 115*(4), 1747-1759.

Iwarsson, J., & Sundberg, J. (1999). Breathing behaviors during speech in healthy females and patients with vocal fold nodules. *Log Phon Vocol, 24*, 154-169.

Jiang, J., Stern, J., Chen, H. J., & Solomon, N. P. (2004). Vocal efficiency measurements in subjects with vocal polyps and nodules: a preliminary report. *Ann Otol Rhinol Laryngol, 113*(4), 277-282.

Jiang, J. J., Diaz, C. E., & Hanson, D. G. (1998). Finite element modeling of vocal fold vibration in normal phonation and hyperfunctional dysphonia: implications for the pathogenesis of vocal nodules. *Annals of Otology, Rhinology, & Laryngology, 107*(7), 603-610.

Johns, M. M. (2003). Update on the etiology, diagnosis, and treatment of vocal fold nodules, polyps, and cysts. *Curr Opin Otolaryngol Head Neck Surg, 11*(6), 456-461.

Jones, D. L., Murry, T., & Rosen, C. A. (1998). The Rold of Physical Therapy in the Multidisciplinary Treatment of Muscle Tension Dysphonia: A Case Report. *Journal of Medical Speech-Language Pathology, 6*(1), 41-47.

Kelchner, L. N., Brehm, S. B., Weinrich, B., Middendorf, J., Dealarcon, A., Levin, L., et al. (2009). Perceptual Evaluation of Severe Pediatric Voice Disorders: Rater Reliability Using the Consensus Auditory Perceptual Evaluation of Voice. *J Voice*.

Kempster, G. B., Gerratt, B. R., Verdolini Abbott, K., Barkmeier-Kraemer, J., & Hillman, R. E. (2009). Consensus Auditory-Perceptual Evaluation of Voice: Development of a Standardized Clinical Protocol. *Am J Speech Lang Pathol, 18*, 124-132.

Kent, R. D. (2004). The uniqueness of speech among motor systems. *Clin Linguist Phon, 18*(6-8), 495-505.

Kenyon, E. (1927). Relation of oral articulative movements of speech and of extrinsic laryngeal musculature in general to function of vocal cord. *Arch Otolaryngol, 5*, 481-501.

Kilner, J. M., Baker, S. N., Salenius, S., Jousmaki, V., Hari, R., & Lemon, R. N. (1999). Task-dependent modulation of 15-30 Hz coherence between rectified EMGs from human hand and forearm muscles. *J Physiol, 516 (Pt 2)*, 559-570.

Kilner, J. M., Fisher, R. J., & Lemon, R. N. (2004). Coupling of oscillatory activity between muscles is strikingly reduced in a deafferented subject compared with normal controls. *J Neurophysiol, 92*(2), 790-796.

Kim, M. J., Hunter, E. J., & Titze, I. R. (2004). Comparison of human, canine, and ovine laryngeal dimensions. *Annals of Otology, Rhinology, & Laryngology, 113*(1), 60-68.

Kinzl, J., Biebl, W., & Rauchegger, H. (1988). Functional aphonia: psychosomatic aspects of diagnosis and therapy. *Folia Phoniatr (Basel), 40*(3), 131-137.

Konrad, H. R., Rattenborg, C. C., Kain, M. L., Barton, M. D., Logan, W. J., & Holaday, D. A. (1984). Opening and closing mechanisms of the larynx. *Otolaryngol Head Neck Surg, 92*(4), 402-405.

Kooijman, P. G., de Jong, F. I., Oudes, M. J., Huinck, W., van Acht, H., & Graamans, K. (2005). Muscular tension and body posture in relation to voice handicap and voice quality in teachers with persistent voice complaints. *Folia Phoniatr Logop, 57*(3), 134-147.

Koufman, J. A., & Blalock, P. D. (1982). Classification and approach to patients with functional voice disorders. *Ann Otol Rhinol Laryngol, 91*(4 Pt 1), 372-377.

Koufman, J. A., & Blalock, P. D. (1991a). Functional voice disorders. *Otolaryngol Clin North Am, 24*(5), 1059-1073.

Koufman, J. A., & Blalock, P. D. (1991b). Functional voice disorders. *Otolaryngologic Clinics of North America, 24*(5), 1059-1073.

Krauss, B., & Green, S. M. (2000). Sedation and analgesia for procedures in children. *N Engl J Med, 342*(13), 938-945.

Kristeva-Feige, R., Fritsch, C., Timmer, J., & Lucking, C. H. (2002). Effects of attention and precision of exerted force on beta range EEG-EMG synchronization during a maintained motor contraction task. *Clin Neurophysiol, 113*(1), 124-131.

Laukkanen, A. M., Leppanen, K., Tyrmi, J., & Vilkman, E. (2005). Immediate effects of 'voice massage' treatment on the speaking voice of healthy subjects. *Folia Phoniatr Logop, 57*(3), 163-172.

Lee, E. K., & Son, Y. I. (2005). Muscle tension dysphonia in children: Voice characteristics and outcome of voice therapy. *Int J Pediatr Otorhinolaryngol, 69*(7), 911-917.

Lieberman, J. (1998). Principles and Techniques of Manual Therapy: Applications in the Management of Dysphonia. In H. T, H. S, R. J & D. M. Howard (Eds.), *The Voice Clinic Handbook* (pp. 91-138). London: Whurr Publishers Ltd.

Linville, S. E. (1992). Glottal gap configurations in two age groups of women. *J Speech Hear Res, 35*(6), 1209-1215.

Marcotullio, D., Magliulo, G., Pietrunti, S., & Suriano, M. (2002). Exudative laryngeal diseases of Reinke's space: a clinicohistopathological framing. *Journal of Otolaryngology, 31*(6), 376-380.

Mathieson, L., Hirani, S. P., Epstein, R., Baken, R. J., Wood, G., & Rubin, J. S. (2009). Laryngeal Manual Therapy: A Preliminary Study to Examine its Treatment Effects in the Management of Muscle Tension Dysphonia. *Journal of Voice, 23*(3), 353-366.

McIvor, N. P., Flint, D. J., Gillibrand, J., & Morton, R. P. (2000). Thyroid surgery and voice-related outcomes. *Aust N Z J Surg, 70*(3), 179-183.

McMahon, T. A. (1984). *Muscles, Reflexes and Locomotion.* Princeton: Princeton University Press.

Merletti, R., & Hermens, H. J. (2004). Detection and conditioning of the surface EMG signal. In R. Merletti & P. Parker (Eds.), *Electromyography: Physiology, Engineering and Non-Invasive Applications* (pp. 107-131). Hoboken: Wiley-IEEE Press.

Mesuda, Y., Oridate, N., Nishizawa, N., Mori, M., Dohsaka, Y., Furuta, Y., et al. (2007). [Three cases of muscle tension dysphonia improved using proton pump inhibitors]. *Nippon Jibiinkoka Gakkai Kaiho, 110*(5), 416-419.

Mima, T., Matsuoka, T., & Hallett, M. (2001). Information flow from the sensorimotor cortex to muscle in humans. *Clin Neurophysiol, 112*(1), 122-126.

Minnigerode, B. (1967). Untersuchungen der extralaryngealen Muskulatur für den Phonationsakt unter besonderer Berücksichtigung des Musculus cricipharyngeus. *Arch Klin Exp Ohren-Hasen u Kehlkopfheilk, 188*, 604-623.

Moore, D. S., & McGabe, G. P. (1998). *Introduction to the Practice of Statistics* (3rd ed.). New York: W.H. Freeman and Company.

Morrison, M. (1997a, March 2-7 1997). *A pathophysiological model for dysphonia.* Paper presented at the XVI World Congress of Otorhinolaryngology Head and Neck Surgery, Sydney, Australia.

Morrison, M. (1997b). Pattern recognition in muscle misuse voice disorders: how I do it. *J Voice, 11*(1), 108-114.

Morrison, M., Rammage, L., & Emami, A. J. (1999). The irritable larynx syndrome. *J Voice, 13*(3), 447-455.

Morrison, M. D., Nichol, H., & Rammage, L. A. (1986). Diagnostic criteria in functional dysphonia. *Laryngoscope, 96*(1), 1-8.

Morrison, M. D., & Rammage, L. A. (1993). Muscle misuse voice disorders: description and classification. *Acta Oto-Laryngologica, 113*(3), 428-434.

Morrison, M. D., Rammage, L. A., Belisle, G. M., Pullan, C. B., & Nichol, H. (1983). Muscular tension dysphonia. *Journal of Otolaryngology, 12*(5), 302-306.

Munhall, K. G., & Ostry, D. J. (1983). Ultrasonic measurement of laryngeal kinematics. In I. Titze & R. Scherer (Eds.), *Vocal fold physiology: biomechanics, acoustics and phonatory control* (pp. 145-162). Denver: Denver Center for the Performing Arts.

Murayama, N., Lin, Y. Y., Salenius, S., & Hari, R. (2001). Oscillatory interaction between human motor cortex and trunk muscles during isometric contraction. *Neuroimage, 14*(5), 1206-1213.

Netto, K. J., & Burnett, A. F. (2006). Reliability of normalisation methods for EMG analysis of neck muscles. *Work, 26*(2), 123-130.

Niimi, S., Horiguchi, S., & Kobayashi, N. (1991). F0 raising role of the sternothyroid muscle: an electromyographic study of two tenors. In J. Gauffin & B. Hammarberg (Eds.), *Vocal Fold Physiology* (pp. 183-188). San Diego: Singular Publishing.

Ohala, J., & Hirose, H. (1970). The function of the sternohyoid muscle in speech. *Annual Bulletin, Research Institute of Logopedics and Phoniatrics, 4*, 41-44.

Orlikoff, R. F., Watson, B., Baken, R. J., & Deliyski, D. (2006). *Quantifying Vocal Attack Using a Glottographic Esitmate of Vocal-Fold Approximation Time.* Paper presented at the 7th International Conference on Advances in Quantitative Laryngology Voice and Speech Research, Groningen, the Netherlands.

Parker, R. I., Mahan, R. A., Giugliano, D., & Parker, M. M. (1997). Efficacy and safety of intravenous midazolam and ketamine as sedation for therapeutic and diagnostic procedures in children. *Pediatrics, 99*(3), 427-431.

Peppard, R. C., Bless, D. M., & Milenkovic, P. (1988). Comparison of Young Adult Singers and Nonsingers with Vocal Nodules. *J Voice, 2*(3), 250-260.

Perez, M. A., Lundbye-Jensen, J., & Nielsen, J. B. (2006). Changes in corticospinal drive to spinal motoneurones following visuo-motor skill learning in humans. *J Physiol, 573*(Pt 3), 843-855.

Perrier, P., Loevenbruck, H., & Payan, Y. (1996). Control of tongue movements in speech: the Equilibrium Point Hypothesis perspective. *Journal of Phonetics, 24*, 53-75.

Peters, H. F., Boves, L., & van Dielen, I. C. (1986). Perceptual judgment of abruptness of voice onset in vowels as a function of the amplitude envelope. *J Speech Hear Disord, 51*(4), 299-308.

Pohja, M., & Salenius, S. (2003). Modulation of cortex-muscle oscillatory interaction by ischaemia-induced deafferentation. *Neuroreport, 14*(3), 321-324.

Pontes, P., Kyrillos, L., Behlau, M., De Biase, N., & Pontes, A. (2002). Vocal nodules and laryngeal morphology. *Journal of Voice, 16*(3), 408-414.

Ramig, L. O., & Verdolini, K. (1998). Treatment efficacy: voice disorders. *J Speech Lang Hear Res, 41*(1), S101-116.

Rammage, L. A., Nichol, H., & Morrison, M. M. (1987). The Psychopathology of Voice Disorders. *Human Communication Canada, 11*(4), 21-25.

Rammage, L. A., Peppard, R. C., & Bless, D. M. (1992). Aerodynamic, Laryngoscopic, and Perceptual-Acoustic Characteristics in Dysphonic Females with Posterior Glottal Chinks: A Retrospective Study. *J Voice, 6*(1), 64-78.

Redenbaugh, M. A., & Reich, A. R. (1989). Surface EMG and related measures in normal and vocally hyperfunctional speakers. *Journal of Speech and Hearing Disorders, 54*(1), 68-73.

Reynere, J. (1974). The Timbre and Pitch Range Variations Following Thyroidectomy. *Brazilian Journal of Otorhinolaryngology, 40*(2), 173-176.

Riddle, C. N., & Baker, S. N. (2005). Manipulation of peripheral neural feedback loops alters human corticomuscular coherence. *J Physiol, 566*(Pt 2), 625-639.

Roeleveld, K., Stegeman, D. F., Vingerhoets, H. M., & Van Oosterom, A. (1997). Motor unit potential contribution to surface electromyography. *Acta Physiol Scand, 160*(2), 175-183.

Rosenberg, J. R., Amjad, A. M., Breeze, P., Brillinger, D. R., & Halliday, D. M. (1989). The Fourier approach to the identification of functional coupling between neuronal spike trains. *Prog Biophys Mol Biol, 53*(1), 1-31.

Roubeau, B., Chevrie-Muller, C., & Lacau Saint Guily, J. (1997). Electromyographic activity of strap and cricothyroid muscles in pitch change. *Acta Otolaryngol, 117*(3), 459-464.

Roy, N. (2003). Functional dysphonia. *Current Opinion in Otolaryngology & Head and Neck Surgery, 11*(3), 144-148.

Roy, N., & Bless, D. M. (1998). Manual circumlaryngeal techniques in the assessment and treatment of voice disorders. *Current Opinion in Otolaryngology & Head and Neck Surgery, 6*, 151-155.

Roy, N., Bless, D. M., & Heisey, D. (2000). Personality and voice disorders: a multitrait-multidisorder analysis. *journal of Voice, 14*(4), 521-548.

Roy, N., Bless, D. M., Heisey, D., & Ford, C. N. (1997). Manual circumlaryngeal therapy for functional dysphonia: an evaluation of short- and long-term treatment outcomes. *J Voice, 11*(3), 321-331.

Roy, N., & Ferguson, N. (2001). Formant frequency changes following manual cicumlaryngeal therapy for functional dysphonia: Evidence of laryngeal lowering? *Journal of Medical Speech-Language Pathology, 9*(3), 169-175.

Roy, N., Ford, C. N., & Bless, D. M. (1996). Muscle tension dysphonia and spasmodic dysphonia: the role of manual laryngeal tension reduction in diagnosis and management. *Annals of Otology, Rhinology, & Laryngology, 105*(11), 851-856.

Roy, N., & Leeper, H. A. (1993). Effects of the manual laryngeal musculoskeletal tension reduction technique as a treatment for functional voice disorders: perceptual and acoustic measures. *J Voice, 7*(3), 242-249.

Roy, N., Nissen, S. L., Dromey, C., & Sapir, S. (2009). Articulatory changes in muscle tension dysphonia: evidence of vowel space expansion following manual circumlaryngeal therapy. *J Commun Disord, 42*(2), 124-135.

Roy, N., Smith, M. E., Allen, B., & Merrill, R. M. (2007). Adductor spasmodic dysphonia versus muscle tension dysphonia: examining the diagnostic value of recurrent laryngeal nerve lidocaine block. *Ann Otol Rhinol Laryngol, 116*(3), 161-168.

Rubin, A. D., Shah, A., Moyer, C. A., & Johns, M. M. (2009). The effect of topical anesthesia on vocal fold motion. *Journal of Voice, 23*(1), 128-131.

Salenius, S., Portin, K., Kajola, M., Salmelin, R., & Hari, R. (1997). Cortical control of human motoneuron firing during isometric contraction. *J Neurophysiol, 77*(6), 3401-3405.

Sander, E. K., & Ripich, D. E. (1983). Vocal fatigue. *Ann Otol Rhinol Laryngol, 92*(2 Pt 1), 141-145.

Sanguineti, V., Laboissiere, R., & Ostry, D. J. (1998). A dynamic biomechanical model for neural control of speech production. *Journal of the Acoustical Society of America, 103*(3), 1615-1627.

Sedláčková, E. (1961). Profesionální hlasové poruchy (Professional Voice Disturbances). *Journal, 14*, 237-242. Czech. (as translated by Dr. Petr Jordan).

Shipp, T. (1975). Vertical laryngeal position during continuous and discrete vocal frequency change. *J Speech Hear Res, 18*(4), 707-718.

Shipp, T., & Izdebski, K. (1975). Letter: Vocal frequency and vertical larynx positioning by singers and nonsingers. *J Acoust Soc Am, 58*(5), 1104-1106.

Simada, Z., & Hirose, H. (1970). The Function of the Laryngeal Muscles in respect to the Word Accent Distinction. *Annual Bulletin, Research Institute of Logopedics and Phoniatrics, 4*, 27-40.

Smith, A., & Denny, M. (1990). High-frequency oscillations as indicators of neural control mechanisms in human respiration, mastication, and speech. *J Neurophysiol, 63*(4), 745-758.

Smith, M. E., Ramig, L. O., Dromey, C., Perez, K. S., & Samandari, R. (1995). Intensive voice treatment in Parkinson disease: laryngostroboscopic findings. *J Voice, 9*(4), 453-459.

Sonninen, A. (1968). The external frame function in the control of pitch in the human voice. *Annals of the New York Academy of Science, 155*, 68-90.

Stager, S. V., Bielamowicz, S. A., Regnell, J. R., Gupta, A., & Barkmeier, J. M. (2000). Supraglottic activity: evidence of vocal hyperfunction or laryngeal articulation? *J Speech Lang Hear Res, 43*(1), 229-238.

Stager, S. V., Neubert, R., Miller, S., Regnell, J. R., & Bielamowicz, S. A. (2003). Incidence of supraglottic activity in males and females: a preliminary report. *Journal of Voice, 17*(3), 395-402.

Stepp, C. E., Heaton, J. T., Braden, M. N., Jetté, M. E., Stadelman-Cohen, T. K., & Hillman, R. E. (submitted). Comparison of neck tension palpation rating systems with surface electromyographic and acoustic measures in vocal hyperfunction.

Stevens, K. N. (2000). *Acoustic Phonetics*. Cambridge, MA: MIT Press.

Stojadinovic, A., Shaha, A. R., Orlikoff, R. F., Nissan, A., Kornak, M. F., Singh, B., et al. (2002). Prospective functional voice assessment in patients undergoing thyroid surgery. *Ann Surg, 236*(6), 823-832.

Stone, R. E., Jr., Cleveland, T. F., Sundberg, P. J., & Prokop, J. (2003). Aerodynamic and acoustical measures of speech, operatic, and Broadway vocal styles in a professional female singer. *J Voice, 17*(3), 283-297.

Story, B. H., & Titze, I. R. (1995). Voice Simulation With A Body-Cover Model Of The Vocal Folds. *Journal Of The Acoustical Society Of America, 97*(2), 1249-1260.

Sturman, M. M., Vaillancourt, D. E., Metman, L. V., Sierens, D. K., Bakay, R. A., & Corcos, D. M. (2007). Deep brain stimulation and medication for parkinsonian tremor during secondary tasks. *Movement Disorders, 22*(8), 1157-1163.

Tecchio, F., Porcaro, C., Zappasodi, F., Pesenti, A., Ercolani, M., & Rossini, P. M. (2006). Cortical short-term fatigue effects assessed via rhythmic brain-muscle coherence. *Exp Brain Res, 174*(1), 144-151.

Thibeault, S. L., Gray, S. D., Li, W., Ford, C. N., Smith, M. E., & Davis, R. K. (2002). Genotypic and phenotypic expression of vocal fold polyps and Reinke's edema: a preliminary study. *Ann Otol Rhinol Laryngol, 111*(4), 302-309.

Titze, I. R. (1989). Physiologic and acoustic differences between male and female voices. *Journal of the Acoustical Society of America, 85*(4), 1699-1707.

Titze, I. R. (1994a). Mechanical stress in phonation. *J Voice, 8*(2), 99-105.

Titze, I. R. (1994b). *Principles of Voice Production.* Englewood Cliffs, NJ: Prentice Hall, Inc.

Titze, I. R., & Hunter, E. J. (2007). A two-dimensional biomechanical model of vocal fold posturing. *Journal of the Acoustical Society of America, 121*(4), 2254-2260.

Tucker, H. M. (1987). The Larynx.

Van Lierde, K. M., Claeys, S., De Bodt, M., & van Cauwenberge, P. (2006). Long-Term Outcome of Hyperfunctional Voice Disorders Based on a Multiparameter Approach. *J Voice.*

van Vugt, J. P., & van Dijk, J. G. (2001). A convenient method to reduce crosstalk in surface EMG. Cobb Award-winning article, 2001. *Clin Neurophysiol, 112*(4), 583-592.

Vennard, W., Hirano, M., & Fritzell, B. (1971). The extrinsic laryngeal muscles. *NATS Bull*, 22-30.

Verdolini, K., Rosen, C. A., & Branski, R. C. (Eds.). (2006). *Classification Manual for Voice Disorders - I.* Mahwah, NJ: Lawrence Erlbaum Associates.

Vertigan, A. E., Gibson, P. G., Theodoros, D. G., Winkworth, A. L., Borgas, T., & Reid, C. (2006). Involuntary glottal closure during inspiration in muscle tension dysphonia. *Laryngoscope, 116*(4), 643-649.

Vilkman, E., Sonninen, A., Hurme, P., & Korkko, P. (1996). External laryngeal frame function in voice production revisited: a review. *J Voice, 10*(1), 78-92.

Wuyts, F. L., De Bodt, M. S., & Van de Heyning, P. H. (1999). Is the reliability of a visual analog scale higher than an ordinal scale? An experiment with the GRBAS scale for the perceptual evaluation of dysphonia. *J Voice, 13*(4), 508-517.

Yang, J. F., & Winter, D. A. (1983). Electromyography reliability in maximal and submaximal isometric contractions. *Arch Phys Med Rehabil, 64*(9), 417-420.

Yang, Q., Fang, Y., Sun, C., Siemionow, V., Ranganathan, V., Khoshknabi, D., et al. (2008). Weakening of Functional Corticomuscular Coupling during Muscle Fatigue. *Brain Research.*

Zenker, W., & Zenker, A. (1960). Ūber die Regelung der Stimmlippenspannung durch von aussen eingreifende Mechanismen. *Folia Phoniatr (Basel), 12*, 1-36.