EFFECTS OF VOCAL INTENSITY AND PHYSICAL ACTIVITY LEVELS ON PHONATORY AND RESPIRATORY FUNCTION

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The vocal folds act as gatekeeper to the flow of air into and out of the lower airway. Another function of the vocal folds is that of oscillating sound source. To date, research has shown that under high respiratory drive (HRD) conditions voice is breathy, suggesting respiratory function will be favored over voice as physiologic needs increase. The problem is for physically active voice users acoustic goals are relatively fixed. This study used a "physiology of activity" paradigm within action theory to investigate the extent to which phonatory and respiratory functions may be affected by systemically varying vocal and metabolic goals. Thirty-two English-speaking females, ages 18-35 years, who were vocally untrained and recreationally active, participated in the study. Participants produced sets of seven consonant-vowel syllables, at rest, using a pre-determined pitch at a comfortable loudness and in a loud voice. Following, participants walked on a treadmill to achieve low and high workloads at established speed and grade adjustments. The same speech task was repeated, using the same vocal intensities. Order of vocal and exercise intensities were counterbalanced. In terms of phonatory function, R_{law} increased significantly more from a baseline of spontaneous voice at rest during loud voice compared to spontaneous voice, mediated by an increase in P_s. Moreover, R_{law} decreased significantly more from baseline with an increase in workload, resulting in increases in metabolic variables. The decrease in R_{law} coincided with numerical increases in airflow. Voice production, as compared to breathing, reduced Ve, interfering with gas exchange. No differences existed for metabolic variables between voice conditions. This study reflected an attempt to understand the

impact of goal-oriented behavior on phonatory and respiratory functions during HRD by manipulating vocal and metabolic goals. Consistent with prior research, voice, as opposed to breathing, resulted in airflow limitation during HRD, reducing ventilation and CO_2 clearance. Extreme respiratory perturbations lead to decreases in phonatory function to support metabolic needs. As predicted by action theory, loud voice appeared to favor phonatory function when vocal goal was specified, at least for a short duration. Conversely, spontaneous voice demonstrated deference to respiratory function when vocal goal remained unspecified.

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PREFACE

I entered my doctoral studies with little formal training in science but the questioning of how the world works has always been an inherent trait of mine. As part of actor preparation during my undergraduate education, I learned to observe human behavior in order to represent it on the stage. I reflected on what motivates people's actions as they interact with the world. I scrutinized people's body language to understand how physical gestures reflect inner dialogue. I trained my ear to listen for the different cadences of people's voice and speech to understand how oral communication reveals thoughts, feelings, and emotions. Through the study of acting, I have learned a great deal about people's behaviors. I have injected that same curiosity to know and passion to discover into voice research.

Overall, I have gained substantially from pursuing a doctorate of philosophy (PhD). Knowledge emerged, analytical skills were sharpened, and my writing was perfected. Although I had moments of doubt, I was resolved to finish. I was steadfast in accomplishing my goal of contributing to a field for which I had so much enthusiasm. Alas, the end has arrived and I am proud of the product I have produced. But the learning continues…

The PhD was not an easy process and it could not have been completed alone. For their support, I must thank a number of people who were instrumental in helping me arrive at this point. To my advisor Kittie, your creativity is limitless, and it has inspired a new generation of researchers. Thank you for teaching me copious valuable skills that I will use to carry on your

legacy through the rigorous research you have trained me to conduct. However, more than that, I must thank you for teaching me to trust myself. Through you, I am forever grateful for discovering that all people can enjoy science.

To my partners in crime, Amanda and Leah, thank you for being such incredible best friends. You challenged my thinking to help me become a stronger researcher than had I not had you as lab mates. More importantly, you comforted me when I needed support during the late nights and trying times. Nothing I could say or do could convey the amount of gratitude and respect I have for the two of you. We are Ziegillou!

To Ryan, Martin, Liz, Doug, Maria, Nicole, Chaya, and Adrianna, I am honored to belong to such an esteemed group. Some of you have provided the foundation upon which I have been able to succeed in this endeavor. Others came along for the ride and provided insight that contributed to my education. Whether past or present, we truly are a family.

To Sue, James, Jessie, John, Charles, Bill, and Connie, thank you for providing me with guidance. The knowledge you imparted to build my skills as an investigator will allow me to make important contributions to science. I truly appreciate your mentorship.

To Clark, Jackie, and the entire team at the UPMC Voice Center, thank you for including me in your family. I am grateful for the time I was able to spend in your center of excellence learning and growing as a clinician and researcher. This study could not have happened without your unwavering support and abundant resources.

To Manop, Andy, and Deborah, I want to thank you for being amazing friends. Your ability to help me keep my eye on the prize has been critical. I could not have reached this day without the fun times I had with you that were an important part of this journey.

To my mom, dad, Jason, Dale, and Mara, I am lucky to have such a caring and supportive family. Although not physically present, you are always in my life. The phone calls, emails, texts, and blog entries make me feel connected with you even when I am thousands of miles away. Thank you for extending a helping hand when I needed one.

To Phu…

We met at a time when I was entering a rather solitary part of the doctoral program. More, you came into my life at I time when I had been thrusted into a period of difficult personal issues that had the potential to further isolate me from the world. Your unwavering love and support allowed me to succeed in earning my Ph.D., but more importantly, it allowed me to continue living. This journey is ours. I love you with all my heart and soul!

1.0 INTRODUCTION: THE PHYSICALLY ACTIVE VOICE USER

> That ventilation during speech is adequate is evidenced by the case with which prolonged speech is accomplished. But the demands of speech and metabolism on the respiratory apparatus are not always compatible (Bunn & Mead, 1971, p. 872).

Estimates indicate roughly 2.5 million individuals in the U.S. are employed in jobs requiring voice use during concurrent strenuous physical activity (U.S. Census Bureau, 2012). Examples of individuals in such professions include military drill instructors, police officers, fire fighters, physical education teachers, aerobics instructors, personal trainers, athletic coaches, musical theater performers, dance teachers, and choreographers. In this document, we shall refer to individuals with simultaneous substantial physical and vocal demands as "physically active voice users" (PAVUs). Such persons push their bodies' physiologic capability to the extreme through challenging physical activities, resulting in conditions of high respiratory drive (HRD). Under such conditions, medullary respiratory activity is stimulated as blood-gas concentrations and pH levels deviate from resting levels, resulting in a substantial increase in ventilation (McArdle, Katch, & Katch, 2010). In addition, PAVUs also face high vocal demands and targeted communication goals. Although undoubtedly communication goals vary depending on the specific task, one constant among PAVUs, and germane to the investigation herein, is the need to effectively deliver an intelligible message that is easily understood by the listener. Some

observations indicate that voice in some PAVUs seems loud and strained, suggesting the possibility that these individuals may be at risk for hyperphonic voice disorders.

An additional observation is that an estimated 33.6% of adolescents (~7.5 million individuals) and 13.9% of the adult population (~8.5 million adults) in the U.S. experience poor cardiorespiratory (aerobic) fitness (Carnethon, Gulati, & Greenland, 2005). Furthermore, a segment of the population has lung diseases that compromise pulmonary function (Control & Prevention, 2012; Labirua & Lundberg, 2010). Some studies report that pulmonary comorbidities and referrals from pulmonology for otolaryngologic evaluation are common among patients who pursue management for voice problems (Best & Fakhry, 2011; S. M. Cohen, J. Kim, N. Roy, C. Asche, & M. Courey, 2012b; Cohen, Kim, Roy, & Courey, 2014; Turley & Cohen, 2010). Such deconditioned individuals are presumed to experience HRD executing even the most basic activities of daily living while still needing to communicate verbally. Any amount of voice use for individuals with poor respiratory drive may pose a problem for ventilation due to little reserve remaining in the system to accommodate further flow limitation imposed by phonatory adduction. One possible solution in that situation might be a result reported in one study that healthy individuals, who displayed a perceptually breathy vocal quality during the production of comfortable speech in HRD (Bailey & Hoit, 2002), exhibit hypophonia. Presumably, the loose vocal fold adduction allows for extra outflow of CO_2 from the lungs to satisfy the metabolic requirements of the body and facilitate a return to respiratory homeostasis.

A picture emerges suggesting that hyper- or hypophonically-induced voice impairments may be associated with physical activity (see Figure 1). Stated differently, a broad sector of the population may experience voice impairments associated with HRD, including impairments associated with either hyper- or hypophonia. The next section will focus on the development of

HYPERphonically-induced voice problems in PAVUs as a consequence of targeted voice production during simultaneous elevated levels of physical activity. From this point forward, we will not further address hypophonia as the focus of the study discussed in this document is on healthy, recreationally active individuals who would engage in professions in which PAVUs are found. Deconditioned or individuals with disease generally abandon such occupational situations due to the inability to meet the physical demands of the activity. To first address the situation of PAVUs, we review the prevalence estimates, symptoms, and risk factors of these voice problems to provide a window into their burden. Then, we will explore the physiology underlying respiration, exercise, and voice, and how the possible competition among them may lead to the hyperphonically-induced voice problems in PAVUs. Finally, we will discuss vocal motor control and frame the study hypotheses around a goal-oriented theory of motor control – action theory.

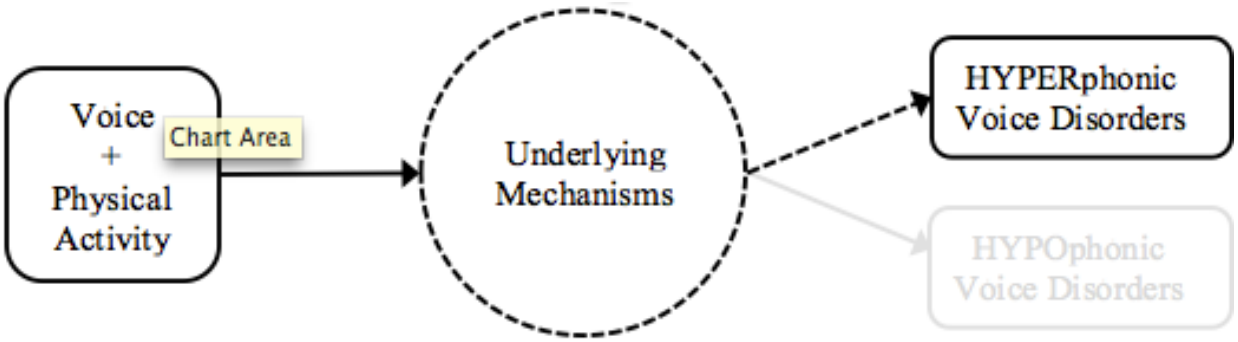

Figure 1. Conceptual schematic: Relationship between simultaneous voice use during physical activity and hyper- or hypophonic voice disorders.

1.1 PAVUS AND VOICE PROBLEMS

1.1.1 Prevalence estimates of voice problems in PAVUs

All PAVUs have one common feature: navigating the dual roles of the larynx as airflow regulator in accomplishing an imperative, biologic respiratory function to support the body's response to a physiologic stressor, and as oscillating sound source to meet a volitional, non-biologic phonatory function to communicate thoughts, ideas, and emotions. Although data are incomplete, a clear impression does emerge that such physically active individuals actually have elevated risk for phonotrauma and *hyperphonically-induced* (loud/strained phonation) voice problems (Evans, Evans, & Carvajal, 1998; Evans, Evans, Carvajal, & Perry, 1996; Heidel & Torgerson, 1993; Hoffman-Ruddy, Lehman, Crandell, Ingram, & Sapienza, 2001; Kersner, 1998; Long, Williford, Olson, & Wolfe, 1998; Mann et al., 1999; Phyland, Oates, & Greenwood, 1999; Rumbach, 2013a, 2013b; Sapir, Atias, & Shahar, 1990; E. Smith, Kirchner, Taylor, Hoffman, & Lemke, 1998; E. Smith, Lemke, Taylor, Kirchner, & Hoffman, 1998). For example, prevalence figures of self-reported voice problems in military instructors, aerobics instructors, and physical education teachers hover around 45%, 44-78% and 32%, respectively (Heidel & Torgerson, 1993; Kersner, 1998; Long et al., 1998; Rumbach, 2013a; Sapir et al., 1990; E. Smith, Kirchner, et al., 1998). Interestingly, research also reveals that aerobics instructors and musical theater performers report elevated risk of somatic injury beyond the larynx (e.g., knees, calves, shins, ankles, and feet) (Evans et al., 1998; Evans et al., 1996; Komura et al., 1992). For example, aerobics instructors are twice as likely to sustain a somatic injury than participants of aerobic dance classes (Garrick, Gillien, & Whiteside, 1986). In other words, for individuals who are required to speak while simultaneously engaging in physical activity, the physiologic demands of

physical activity may interfere with healthy vocalization and, conversely, the physiologic demands of phonation may interfere with executing movement safely.

1.1.2 Symptoms and risk factors of voice in PAVUs

Vocal symptoms frequently experienced by PAVUs include hoarseness, low pitch, vocal fatigue, laryngeal discomfort, and even voice loss (Heidel & Torgerson, 1993; Kersner, 1998; Komura et al., 1992; Long et al., 1998; Phyland et al., 1999; Rumbach, 2013a; Sapir et al., 1990). Worth noting, symptoms such as hoarseness (and to some extent voice loss) persist in aerobics instructors, for example, even *after* a given class instruction has ended (Heidel & Torgerson, 1993; Rumbach, 2013a). In fact, between 40 and 60% of aerobics instructors who report voice symptoms indicate their voice changes are chronic (Kersner, 1998; Rumbach, 2013a). This finding stands in stark contrast to reports by aerobics *participants* whose hoarseness during aerobics classes seems to be temporary, as evidenced by a marked decrease in reported hoarseness immediately following classes (Heidel & Torgerson, 1993). Persistence of voice problems in PAVUs suggests that vocal fold tissue may incur phonotraumatic changes from voice use, i.e., injury to tissue from repeated collisions of the vocal folds during phonation (Verdolini, 1999), during HRD conditions. Although some voice problems are preventable and reversible, those experienced by many PAVUs are chronic.

Several sources of such tissue changes can be identified for PAVUs: vocal dose, whole body physical stress, and environmental factors (and possibly their interaction). Regarding vocal dose, conventional wisdom attributes occupationally-induced voice disorders in general to substantial vocal loads involved in professional responsibilities, a suggestion supported in part by vocal dosimetry data (for review see Hunter & Titze, 2010; I. R. Titze, Hunter, & Svec,

2007). Not surprisingly, PAVUs are indeed at special risk of developing a voice problem as they accumulate substantial vocal doses engaging in professional or avocational duties (Kersner, 1998; Komura et al., 1992; Long et al., 1998). For example, in aerobics instructors, the risk of developing a voice problem increases with an increase in the number of weekly classes conducted (Kersner, 1998; Komura et al., 1992). Similarly, aerobics instructors who report voice loss have significantly more years of teaching experience compared to aerobics instructors without a history of voice problems (Kersner, 1998; Long et al., 1998). Along similar lines, one study documented similar amounts of total singing hours per month, including practice, across singers performing in different genres (musical theater [MT], opera [OP], and contemporary [CO]) (Phyland et al., 1999). In that study, MT singers reported the most *performance* singing hours per month (M=46.5 hours) and OP singers reported the least performance singing hours per month (M=15 hours). This finding, when considered in relation to the higher prevalence of diagnosed vocal conditions and, more specifically, of vocal fold edema (possibly a reflection of phonotrauma) in MT singers than in singers of other styles, further suggests a role of vocal load in the development of vocal fold pathology.

Regarding a whole body response to physical stress, the finding of substantial vocal load in MT singers in particular can be coupled with the observation that these singers also spend numerous hours singing while also executing physically demanding choreography (Phyland et al., 1999). In fact, Broadway and West End performers (dancers and actors) with the highest self-rated physical performance demands related to their role were almost three times more likely to have any somatic injury in their current production than performers with less physical performance demands. Moreover, performing on an inclined stage – presumably increasing physical work compared to a flat stage – increased Broadway musical dancers' and West End

actors' risk of somatic injury (Evans et al., 1998; Evans et al., 1996). Further, female dancers in Broadway and West End musicals, who, due to gender, are likely to have poorer aerobic capacity and exercise tolerance than men (Harms, 2006; Sheel & Guenette, 2008), were more likely to sustain injury than their male counterparts (Evans et al., 1998; Evans et al., 1996). Another interesting finding suggesting a connection between phonation-induced voice problems and physical performance demands is that significantly more military recruits than military drill instructors report the onset of voice symptoms pre-dating their involvement in the army and, conversely, significantly more military drill instructors than military recruits specify that their voice symptoms commenced or worsened after assuming their position as drill instructor (Sapir et al., 1990). In sum, data suggest that PAVUs not only have increased risk for a voice disorder, but also increased risk for somatic injury. At first glance, these risks seem uniquely linked to "dose" in the respective domains, but a deeper inspection of the data suggests a possible interaction such that the risk of voice problems may increase when paired with strenuous physical activity.

Regarding environmental factors, they have also been shown to play a role in the development of a voice problem in such individuals. PAVUs work in spaces that vary in size and acoustic properties, at times with high levels of ambient noise (Heidel & Torgerson, 1993; Long et al., 1998), all of which may affect voice production. Accordingly, for aerobics instructors, the self-reported need to shout out cues to participants, perhaps to overcome loud music or to be heard in large, "acoustically-dead" rooms, appears significantly greater in aerobics instructors with voice complaints compared to those without (Long et al., 1998). Despite this finding, environmental variables such as facility size, perception of room acoustics, and music volume have not been shown to significantly differentiate aerobics instructors with self-reported voice

problems from those without such problems (Long et al., 1998). Moreover, although microphone usage significantly decreases the vocal intensity of aerobic instructors' speech during class as compared to teaching without amplification (H. Koblick, 2004), self-reported microphone usage has not been shown to significantly differentiate aerobics instructors with voice problems from those without them (Long et al., 1998). The picture that emerges from inspection of these data reveals that PAVUs incur voice problems through their occupational duties, and the risk of developing a voice problem increases for females who engage in loud voice production for extended periods during work. At least for aerobics instructors, factors such as the size of the facility, the loudness of the music, the perception of room acoustics, and microphone usage do not appear to alter the risk of developing a voice disorder, again suggesting that another factor, perhaps voice use under conditions of HRD may elevate the risk of voice problems in PAVUs.

An additional, relevant point is that some physical environments, e.g., in theaters, may aggravate and elevate the risk of voice problems in some PAVUs by impacting their vocal hygiene. High temperatures, dry air, and dust and other harmful substances are common climatic conditions in theaters (Richter et al., 2002; Richter, Lohle, Maier, Kliemann, & Verdolini, 2000). Relevant to high temperatures, one response to physical activity is an increase in core body temperature. Under normal conditions, the body's thermoregulating response dissipates heat to maintain a stable temperature. Under extreme heat, the core body temperature rises excessively because cooling of the body is insufficient to offset the additional heat the body absorbs from the environment. As a consequence, individuals experience limited exercise capacity during exercise in high ambient temperatures (McArdle et al., 2010). Musical theater performers and other PAVUs who are in environments lacking humidity and temperature control are certainly at risk for overheating.

Relative to irritants and dehydration, they have been recognized throughout the voice literature as contributing factors to voice problems (Koufman, 1991; Sivasankar & Leydon, 2010), and are often targeted in indirect voice therapy (Ziegler, Gillespie, & Abbott, 2010). Interestingly, studies suggests aerobic instructors do gain exposure to vocal hygiene information (Heidel & Torgerson, 1993; Rumbach, 2013a) and yet aerobic instructors with and without self-reported voice problems do not demonstrate a difference in vocal hygiene knowledge (Kersner, 1998; Long et al., 1998). To that end, research on teachers of school-aged children demonstrates that vocal hygiene alone as a treatment for voice problems does not improve the voice; however, ascribing to principles of vocal hygiene may stop progression of voice problems (Ziegler et al., 2010). Aerobic instructors frequently report adequate water consumption both in and out of class (Heidel & Torgerson, 1993). Given the large percentage of aerobic instructors reporting adequate hydration, it is no surprise that in-class water consumption has not been shown to be associated with voice problems reported by aerobic instructors (Kersner, 1998). Relevant to other factors known to affect vocal hygiene, medications, smoking, and allergies have also not been shown to be associated with voice problems reported by aerobic instructors (Heidel & Torgerson, 1993; Kersner, 1998). In sum, myriad factors may contribute to voice problems in PAVUs, including vocal dose, whole body response to physical activity, environmental factors, and vocal hygiene.

1.1.3 Consequences of voice problems in PAVUs

Not only do aerobically-challenged individuals who also have substantial vocal load appear to have an increased risk of hyperphonic voice problems compared to the general population, but also such risks have substantial, non-trivial consequences. For example, intelligibility and communicative competence may be impaired. For example, aerobics instructors with voice

complaints report difficulty generating a loud voice as a result of their voice problem (Komura et al., 1992). Nearly 25% of aerobic instructors who received a voice diagnosis in one study reported taking a leave of absence while they received treatment for their voice problem (Rumbach, 2013b). Even more problematic, a study of Broadway and West End performers found that injuries sustained by dancers and actors, the nature unspecified but including injuries to the larynx, resulted in *at least* one missed performance during the current production period (Evans et al., 1998; Evans et al., 1996). Although not statistically significant, more MT singers than OP or CO singers reported their vocal impairment was disabling, causing them to have more problems with their speaking and singing voice within the prior 12 months, and resulted in greater handicap, limiting performance opportunities over the preceding 12 month period (Phyland et al., 1999). Other data suggest that even in the general population, voice problems negatively affect quality of life (Elaine Smith, Gray, Verdolini, & Lemke, 1995), which, in some cases, may lead to psychological issues such as anxiety and depression (Mirza, Ruiz, Baum, & Staab, 2003). The issues would seem to be even further heightened in vocal performers. In fact, one study found that up to 50% of aerobic instructors who received a voice diagnosis underwent emotional distress, were socially withdrawn, and felt less work-related satisfaction (Rumbach, 2013b). The societal cost associated with having a voice problem in occupational voice users is staggering, conservatively estimated in 2001 at roughly 2.5 billion dollars annually for teachers alone (Verdolini & Ramig, 2001). More recent figures on the direct health care costs for management of laryngeal diseases and disorders in the general population estimate the annual total between $178.5 to $294.8 million (S. M. Cohen, J. Kim, N. Roy, C. Asche, & M. Courey, 2012a), which does not account for replacement personnel, workman's compensation, or change/loss of job. Indeed, PAVUs seek management for voice problems from a variety of

medical and non-medical professionals, including otolaryngologic and speech-language pathology services (Evans et al., 1998; Evans et al., 1996). Given the persistence of voice problems in PAVUs, inflated health care costs may result from ongoing visits. As with aerobics instructors and musical theater performers, military drill instructors who report voice problems suffer a vocational impact due to the interference imposed on the execution of daily duties, especially in those who reported 3 or more symptoms. The disabling impairment of voice problems in military drill instructors even causes absences from work (Sapir et al., 1990). Therefore, in some cases, public safety may be compromised as, for example, with military personnel and police (I. R. Titze, Lemke, & Montequin, 1997). In sum, substantial implications of voice problems exist in individuals who speak in HRD, not only for communication, quality of life, and economic impact, but also for human safety.

1.2 PHONOTRAUMA AND VOICE DISORDERS

1.2.1 The mechanical stress hypothesis of phonotraumatic lesions

PAVUs report having been diagnosed with types of laryngeal pathology (Heidel & Torgerson, 1993; Phyland et al., 1999; Rumbach, 2013b) that putatively result from vocal fold collision forces during repeated vocal fold vibration (Jiang & Titze, 1994; I. R. Titze, 1994). For example, 7-10% of aerobics instructors report having been diagnosed with vocal fold nodules (Heidel & Torgerson, 1993; Kersner, 1998; Rumbach, 2013b) and 28% of musical theater performers report having been diagnosed with laryngeal edema, vocal fold nodules, or polyps (Phyland et al.,

1999). A prevailing model of physical injury in general indicates the primary traumatic agent in injury is the transfer of energy in amounts exceeding local tissue thresholds (Haddon, 1963).

Phonation introduces several types of mechanical stress to vocal fold tissue. Despite differences in estimates of the relative folds of mechanical stresses in phonation, one important source of such injury is perpendicular vocal fold impact stress (force/area) (I. R. Titze, 1994). Such impact stress is of particular importance because of the theoretical relationship between resulting collision forces during vocal fold vibration and phonotrauma (VF tissue injury) (Jiang & Titze, 1994; Tao & Jiang, 2007; I. R. Titze, 1994). Relevant in terms of empirical data, Jiang and Titze (1994) completed a seminal study investigating factors contributing to increased inter-vocal fold perpendicular impact stress, using an excised hemilarynx set-up. Findings pointed to three factors directly affecting impact stress: subglottal pressure, vocal fold adduction, and vocal fold elongation (i.e., increased pitch), the latter apparently within vocal register. Furthermore, in all experimental conditions, inter-cordal impact stress was greatest at the mid-membranous vocal fold, where phonotraumatic lesions characteristically develop, compared to other locations. Traditionally, certain vocal behaviors such as a loud, pressed voice at high pitches have been thought to increase the risk of benign, phonotraumatic vocal fold lesions such as nodules (Verdolini, Rosen, & Branski, 2006). Jiang and Titze's results are consistent with these traditional clinical notions.

1.2.2 Vocal fold microarchitecture and phonotraumatic lesions

The typical site of phonotrauma is the vocal fold mucosa, a superficial band of tissue described shortly. The development of phonotraumatic lesions in this particular site can be partially understood in terms of vocal fold microarchitecture. The vocal folds are a unique, multi-layered

structure (M Hirano, Kurita, & Nakashima, 1981), which may have evolved for purposes of phonation, specifically (I.R. Titze, 2000). The outermost layer of the vocal fold, exposed to the airway lumen, consists of stratified squamous epithelial cells. The epithelium's function is to protect the more deeply-seated lamina propria. The epithelium also helps to bind the underlying tissue together as a cohesive unit (S. D. Gray, 2000). At the deep edge of the epithelium is the basement membrane zone (BMZ). Special anchoring fibers in this region, composed of collagen and fibronectin (Courey, Shohet, Scott, & Ossoff, 1996; Sakai, Keene, Morris, & Burgeson, 1986), secure the epithelial layer to underlying lamina propria (S. D. Gray, Pignatari, & Harding, 1994). Certain phonotraumatic lesions such as vocal fold nodules form as a result of disruption of anchoring fibers, and consequently, the BMZ demonstrates disorganized anchoring fibers (S. Gray & Titze, 1988), which results in gaps between cells of the BMZ and epithelial cells (Kotby, Nassar, Seif, Helal, & Saleh, 1988). Histological analysis of the BMZ reveals varying anchoring fiber densities across subjects, suggesting some individuals may be genetically predisposed to developing certain vocal fold pathologies (S. D. Gray, 2000). As a result of its superficial position, the epithelium, and by extension the basement membrane zone, are susceptible to phonotraumatic injury (S. Gray & Titze, 1988; I.R. Titze, 2000).

Beneath the epithelial layer is the tri-layered lamina propria. The superficial lamina propria (SLP), the outermost layer of the lamina propria, is also known as Reinke's space. This floppy layer contains loosely and sparsely distributed elastin and collagen fibers essentially "floating" in interstitial fluid (Hammond, Zhou, Hammond, Pawlak, & Gray, 1997). The SLP is thickest at the mid-membranous portion of the vocal fold, and contains considerable quantities of hyaluronic acid (HLA) (Hammond et al., 1997). HLA is thought to have an important influence in the biomechanics of voice production due to its ability to both allow for vibration and also to

absorb mechanical stress generated in vocal fold collisions (Ward, Thibeault, & Gray, 2002). Functionally, the epithelium and SLP form a unit called the vocal fold mucosa, which constitutes the fundamental tissue involved in vocal fold vibration (M. Hirano & Kakita, 1985). The mucosa bears most of the mechanical stress during vocal fold collision (I.R. Titze, 2000).

The next two layers of the vocal fold are the intermediate and deep layers of the lamina propria (ILP and DLP, respectively). The ILP has a large concentration of elastin fibers (Hammond, Gray, Butler, Zhou, & Hammond, 1998), which allows vocal fold tissue to resume its pre-deformed shape during and after successive cycles of vibration (S. D. Gray, 2000). The DLP is characterized by a large proportion of tightly arranged collagen fibers (Hammond, Gray, & Butler, 2000). Unlike elastin fibers, collagen fibers are minimally extensible and restrict elongation. As such, collagen fibers serve a protective function by increasing tissue stiffness and limiting tissue deformation (S. D. Gray, 2000). Together, the ILP and DLP constitute the vocal ligament. This ligament is thinnest at the midpoint of the vocal fold and thickest at its point of attachment to the thyroid cartilage anteriorly and vocal processes posteriorly. These ligamentous attachments are referred to as the anterior and posterior macula flava, respectively. The combination of collagen fibers and thickened tissue at the point of attachment makes the vocal ligament well suited for bearing much of the tensile mechanical stress that vocal fold tissue encounters during pitch increases (I. R. Titze, 1994; I.R. Titze, 2000).

The deepest layer and the bulk of the vocal fold is the thyroarytenoid (TA) muscle (I.R. Titze, 2000). TA muscle consists of striated muscle with both type I slow-twitch oxidative (medial portion) and type IIB fast-twitch glycolytic (lateral portion) muscle fibers. However, the TA muscle also has hybrid type IIX muscle fibers, which have the ability to express themselves as slow- or fast-twitch variants (J. F. Y. Hoh, 2005). Type I muscle fibers are mainly implicated

in aerobic activities. A high oxidative capacity and high resistance to fatigue characterize this muscle fiber type. In contrast, type IIB muscle fibers are mainly involved in short duration anaerobic activities. A high glycolytic capacity and low resistance to fatigue characterize this muscle fiber type. With training, type IIX muscle fibers may adopt increased oxidative or glycolytic capacity, depending on the type of activity that demands its involvement (McArdle, et al., 2010). The two innermost layers of the vocal fold, the DLP and TA muscle, constitute what is considered, for modeling purposes, the body of the vocal fold. In a generally accepted theory of vocal fold vibration, the "cover" (epithelium, SLP and ILP) propagates as a wave over the "body" of the vocal fold during phonation (M. Hirano & Kakita, 1985).

Despite structural adaptations in vocal fold tissue to accommodate mechanical stress during phonation, repeated vocal fold collisions do indeed lead to microinjury. Two temporal forms of phonotrauma are notable: acute and chronic. The clinical presentation of acute phonotrauma is edema, typically at the point of greatest impact stress, that is the mid-membranous portion of the vocal folds bilaterally. Chronic phonotrauma develops due to repeated acute vocal fold injury, that is, recurrent acute phonotraumatic (RAP) events. In some cases, protein synthesis and remodeling occur in such a way that prevents a chronic lesion from forming. In other cases, pathological response to injury occurs during the wound healing cascade (Diegelmann & Evans, 2004), which in the case of the vocal folds results in the results in benign vocal fold lesions, in the extreme case culminating in vocal fold scar (Branski, Verdolini, Sandulache, Rosen, & Hebda, 2006). Typical examples of structural pathologies of the larynx thought to result from RAP events include benign vocal fold lesions such as polyps and cysts as well as different instantiations of scar such as vocal fold nodules and fibrous masses (Branski et al., 2006).

Hyperphonia does not always result in phonotrauma. In the case of primary muscle tension dysphonia, a voice disorder with no organic structural cause, and for which a psychogenic or neurologic etiology is lacking, the larynx demonstrates increased and dyscoordinated muscle activation during phonation. The result is a strained voice (Verdolini et al., 2006). Contemporary clinical thinking is that muscle tension dysphonia occurs from poor coordination of respiration with phonation (T. J. Hixon & Putnam, 1983; Rubin, Macdonald, & Blake, 2011). Although aerobic instructors appear to mostly report diagnoses such as benign phonotraumatic lesions or other vocal fold tissue changes, they also report receiving a diagnosis of muscle tension dysphonia (Rumbach, 2013b).

To summarize, the vocal folds are a multi-layered structure that may have evolved with protective mechanisms that spare them from injury, or at the very least limit the magnitude of injury. The body responds to injury by initiating a wound healing process, which in some cases results in complete healing but in other cases leads to the formation of a lesion that affects phonation. A mechanical stress hypothesis of phonotrauma suggests that in some cases, repeated collisions of the vocal folds may ultimately lead to the development of chronic benign vocal fold lesions. Factors that increase impact stress, namely those associated with loud, strained, and high-pitch phonation, may place PAVUs at risk. However, excessive and poorly coordinated muscle activation may also result in a hyperphonically-induced voice problem such as muscle tension dysphonia. Finally, the vocal folds consist of different muscle fiber types, which may change with training and voice use. Those fiber types rely on different metabolic processes and, therefore, specific patterns of activation may vary with the unique types of activities for which they are suited.

1.3 PHYSICAL ACTIVITY

An examination of the acute physiologic response to physical activity exposes the body's metabolic needs that PAVUs compete with to achieve acoustic goals. Physical activity (i.e., any physical movement produced by contraction of skeletal muscle that substantially increases energy expenditure) stimulates somatic dynamics involving caloric expenditure, which in turn requires energy transfer (McArdle et al., 2010). In terms of our discussion here, two different classes of physical activity can be identified: leisure time physical activity and occupational physical activity (OPA). Of particular interest, OPA consists of any physical activity regardless of intensity associated with the performance of a job, usually within the time frame of an 8-hr workday (Howley, 2001). Energy demand during physical activity induces a series of somatic responses to meet metabolic needs. The body's response depends on the type, intensity, and duration of physical activity as well as the physical fitness of the individual (McArdle et al., 2010). Components of physical fitness include cardiorespiratory endurance, muscular strength and muscular endurance, flexibility, and body composition, and PAVUs may differ in their fitness within and across occupational physical activities. Of particular interest, cardiorespiratory (aerobic) fitness depends on the efficiency of the cardiorespiratory system to deliver oxygen (O_2) to active muscles (Farrell, Joyner, & Caiozzo, 2011). Exercise that is aerobic in nature involves large muscle groups in activities that result in substantial increases in heart rate and energy expenditure. Regular participation in aerobic exercise results in improvements in cardiorespiratory fitness, leading to an increase in endurance performance (McArdle, et al., 2010).

1.3.1 Physical activity and energy expenditure

Typically, the total daily energy expenditure (TDEE) of an individual is largely accounted for by resting metabolic rate (RMR), which usually contributes up to 75% of TDEE. RMR is the energy expenditure per body surface area per hour ($kCal \cdot m^{-2} \cdot h^{-1}$) measured several hours after consumption of a small meal and prior to any physical activity. RMR is consistently shown to be slightly higher than basal metabolic rate (BMR), which refers to the minimum level of energy required to sustain vital functions at rest after fasting. Females register lower BMR and RMR than males. Increased body fat in women, which metabolizes at a slower rate than lean muscle tissue, accounts for these gender differences. Regardless of body composition, physical activity is a major factor affecting energy expenditure and accounts for up to 30% of the TDEE under typical circumstances. In fact, a majority of individuals who engage in light to moderate exercise demonstrate a 10-fold increase in energy expenditure above BMR. The net energy expenditure of the performance of physical activity results from subtracting out the energy assumed to be expended during rest from the gross energy expenditure of all bodily metabolic activity, leaving the remaining energy expenditure to represent energy expended during physical activity (McArdle et al., 2010).

A classification system widely used to determine the energy required for the accomplishment of a task is the metabolic equivalent (MET). A single MET is the energy expenditure, as measured by oxygen consumption, required per unit body mass per minute at rest, which is 3.5 $ml \cdot kg^{-1} \cdot min^{-1}$ (McArdle et al., 2010). A physical activity between 3 and 6 METs is classified as moderate intensity, and any physical activity of more than 6 METs is classified as vigorous (Pate et al., 1995, as cited in Ainsworth et al., 2000). In terms of PAVUs, the intensity of their OPA tends to hover around moderate to high. Teaching an aerobics class

(class intensity level unspecified), as some PAVUs do, requires about 6.0 METs. Teaching physical education, exercise, or sports classes, as other PAVUs do, requires about 4.0 (non-sport play) to 6.5 (participate in the class) METs. Coaching, as might be expected by PAVUs in activities such as football, soccer, basketball, or baseball, requires about 4.0 METs. Dancing (ballet or modern, twist, jazz, tap, or jitterbug), as might be demanded for PAVUs who are choreographers or musical theater performers, requires about 4.8 METs (Ainsworth et al., 2000).

1.3.2 Energy substrates to support aerobic activity

Any physical activity classified as aerobic utilizes the aerobic-oxidative metabolic system as the main energy pathway. PAVUs generally engage in physical activity that, at least in part, involves the aerobic-oxidative metabolic system. This pathway requires the presence of O_2 in energy creation. Sources of energy vary depending on the type, duration, and amount of physical activity. Carbohydrates provide the main substrate for energy during this kind of physical activity, especially high-intensity or long duration exercise. Glucose derived from carbohydrates can be absorbed by the digestive system into the blood for immediate use as an energy source. However, glucose may also be stored for later use in the form of glycogen or fat. Glycogen forms from the bonding of multiple glucose molecules and is stored in the liver and muscles. The quantity of glycogen stored by the body fluctuates with the amount of dietary carbohydrates, leading those completing aerobic exercise to increase glycogen by consuming more carbohydrates. Glycogen breaks down into glucose in a process called glycolysis. This process does not require O_2, which makes glycolysis ideal in the initial stage of exercise when the demand for O_2 is greater than O_2 consumption. Consequently, O_2 needs are not adequately met, leading to an O_2 deficit. Once the acute cardiorespiratory response to exercise supplies adequate

amounts of O_2 to the working muscle, as much as 55% of hepatic glycogen stores become depleted from aerobic glycolysis during an hour of intense aerobic activity. Adequate blood glucose levels during and after physical activity limit several negative consequences of exercise. In addition to fueling active muscles, an adequate level of intramuscular glucose limits the use of protein as an energy source and, as a result, preserves muscle tissue. A sufficient amount of glucose in the body also prevents a decrease in plasma pH from an accumulation of ketones, a metabolic by-product when fat is harvested as an energy substrate. Finally, optimal central nervous system performance depends on carbohydrate consumption (McArdle et al., 2010).

Fat is another energy substrate used to perform physical activity. As with carbohydrates, the contribution of fat as an energy supplier for exercise varies depending on the intensity and duration of physical activity. Fat as an energy substrate requires O_2. During exercise, fat oxidation increases due to increased blood flow through adipose tissue. Compared to rest, the body uses three times as much fat during light and moderate intensity aerobic exercise. Whereas the body's energy needs become increasingly reliant on fat during longer durations of exercise, as exercise intensity increases, fat oxidation decreases while carbohydrates become an increasingly important energy substrate (McArdle et al., 2010).

In the first few minutes of moderately intense exercise, at which time the demand for O_2 is not met (i.e., there is so-called oxygen deficit), active muscle relies on intramuscular glycogen stores as the predominant energy source. Without the availability of O_2, glycogen will maintain its role as the main contributor of energy. However, as the intensity of aerobic exercise increases, during which time the demand for and availability of O_2 increases, blood glucose levels increase to fuel active muscles. While exercising for 20 minutes at moderate intensity, roughly half of the energy to active muscles comes from liver and muscle glycogen stores. The remainder of energy

during exercise at moderate intensity comes primarily from fat stores. As physical activity approaches maximal intensity, the primary energy substrate is carbohydrates. A different pattern of energy substrate utilization occurs with increased exercise duration. Fat becomes the main energy substrate with longer bouts of light and moderate intensity exercise (McArdle et al., 2010).

Respiratory quotient, a measure that reflects the body's utilization of fuel substrates (carbohydrate or fat), is an indicator of how the body accesses energy at rest. A related measure, respiratory exchange ratio (RER), uses the ratio of produced CO_2 to consumed O_2 expired in one breath as a proxy for estimating respiratory quotient during physical activity. During physical activity, a measurement of RER at or below 0.70 suggests that the body's predominant fuel source is fat. Conversely, a measurement of RER at or above 1.00 indicates that the body's predominant fuel source is carbohydrate. Intermediate values (i.e., $0.70 < RER < 1.00$) reflect access to a mix of fat and carbohydrates as energy substrates (McArdle et al., 2010).

Although contraction of skeletal muscle produces movements during physical activity, skeletal muscles can only accomplish movement during such activity because of a series of chemical reactions supplying them with energy. Specifically, the body stores and transfers a compound known as adenosine triphosphate (ATP) to supply working muscles with energy. The ability of an individual to accomplish a certain intensity of physical activity is dependent on the rate at which ATP can be produced. Additionally, the pathway by which the body produces ATP differs depending on the type of physical activity. In short, a catabolic chemical reaction breaks down high-energy phosphate bonds in ATP by cleaving a phosphate (Pi) and forming a new compound, adenosine diphosphate (ADP). This process also liberates large amounts of energy

that can be used to stimulate muscular contractile processes to perform mechanical work (McArdle et al., 2010).

$$\text{ATP} \rightleftharpoons \text{ADP} + \text{Pi} + \text{Energy}$$

Equation 1. Chemical equation for conversion of adenosine triphosphate to adenosine diphosphate

ATP is limited in muscle tissue and only supplies energy for a few seconds of intense exercise. However, sustained forms of aerobic exercise such as running rely on a continual and efficient replenishment of ATP. Anaerobic glycolysis, which does not require O_2, yields limited production of ATP. In contrast, oxidative phosphorylation, which requires O_2, creates up to 32 molecules of ATP per glucose molecule. However, the aerobic-oxidative energy pathway relies on the delivery of adequate amounts of O_2 by the cardiorespiratory systems to produce ATP. An individual's aerobic capacity depends on maximal O_2 consumption (VO_{2max}), which is the largest amount of O_2 an individual can acquire and utilize to produce ATP aerobically. Although rapid glycolysis does not use O_2 to form ATP, the process of ATP production through glycolysis is often considered a preliminary step in aerobic ATP production. One by-product of glycolysis, pyruvate, can become involved in ATP production if oxygen is present in the cell. With greater exercise intensity, hydrogen atoms flow to pyruvate to form a new compound, lactate. This process involves the electron transport chain, which is the final pathway in aerobic metabolism. As a consequence, lactate levels rise as exercise intensity increases, which may limit performance of physical activity. Lactate accumulation in steady-state submaximal aerobic exercise below 55 to 60% of VO_{2max} is generally small. However, when exercise exceeds this steady-state level, lactate removal in the muscle lags behind lactate accumulation. The onset of

blood lactate accumulation occurs when lactate formation exceeds lactate clearance, at which point blood lactate increases. The accumulation of blood lactate causes a decrease in pH and may lead to fatigue (McArdle et al., 2010). A concern relative to lactate formation and clearance is that, for some PAVUs, layering muscular contraction required for phonation on top of whole body physical activity may result in earlier onset of blood lactate accumulation, thereby leading to increased ventilation and fatigue with which phonatory goals must compete.

1.3.3 Acute responses to aerobic exercise

1.3.3.1 Cardiovascular responses

The acute response of the cardiovascular system to exercise serves to increase O_2 transport to working muscle tissue and remove metabolic waste, transport nutrients to tissues, and regulate body temperature. During physical activity cardiac output ($L \cdot min^{-1}$) rises substantially. This increase occurs from an increase in heart rate and stroke volume output (the amount of blood that is squeezed out of the heart with each contraction). Stroke volume eventually tapers off and further increases in cardiac output result from continued increases in heart rate. In addition to changes in the volume of blood that circulates, peripheral blood flow changes to meet metabolic demands of working muscle tissue. Vasodilation of skeletal muscles involved in physical activity increases blood flow through active muscles. In addition, blood flow changes from normal circulation patterns at rest. The diversion of blood flow to active muscle occurs in part because of vasoconstriction of tissue not involved in exercise. The vasoconstriction of blood vessels in the viscera facilitates a redistribution of blood flow to the active muscles. In addition, systolic blood pressure increases during aerobic exercise mainly due to greater ventricular contraction of the heart in the systole phase (McArdle et al., 2010).

Increased blood flow is not the only mechanism for supplying working muscle tissue with adequate amounts of O_2. The majority of O_2 in the body is bound to hemoglobin found in red blood cells rather than dissolved in blood plasma. As such, hemoglobin increases the blood's capacity to carry O_2. Women have, on average, 5% to 10% less hemoglobin than men, which accounts for gender differences observed in aerobic capacity between men and women. During intense aerobic exercise, hemoglobin unloads large amounts of O_2 to working muscle tissue. O_2 extraction by working muscles is possible because of greater O_2 dissociation from hemoglobin at lower tissue O_2 partial pressures, lower pH values and higher temperatures. As a result, the difference in O_2 content between arterial and venous blood becomes greater during exercise compared to rest. This situation is known as arteriovenous-oxygen difference (a-vO_2 difference), and is another mechanism that accounts for the improved aerobic capacity of working muscle during exercise. Those two mechanisms, maximum cardiac output and maximum a-vO_2 difference, establish VO_{2max} (McArdle et al., 2010).

1.3.3.2 Respiratory responses

Of particular relevance for PAVUs who are faced with possibly competing respiratory and phonatory goals, is the pulmonary system's acute physiologic response to exercise, which leads to an increase in the movement of air in and out of the body, also known as high respiratory drive (HRD). This response facilitates increased gas exchange between the environment and the body and plays a role in the regulation of acid-base balance. With the initiation of exercise, minute ventilation ($L \cdot min^{-1}$), the product of breathing frequency and tidal volume, increases rapidly, also known as hyperpnea. The initial increase in ventilation arises due to locomotor-linked stimuli, namely central command (feedforward) and muscle receptors (feedback). Then, ventilation rises steadily to closely match the body's metabolic needs in a feedback mechanism, and almost

directly in proportion to the increase in carbon dioxide production. Eventually, the ventilatory response plateaus (McArdle et al., 2010).

Minute ventilation increases linearly with exercise intensity up to 50-75% of VO_{2max}, at which time the rise in ventilation increases disproportionately. The point at which the increase in ventilation shifts categorically is called the ventilatory threshold. This shift is believed to result from lactic acid accumulation, which increases the hydrogen ion (H^+) concentration in the blood and induces an increase in ventilation. The increased ventilatory response during exercise is important because this response is a mechanism by which the body maintains arterial gas partial pressures and pH levels at a dynamic constancy (homeostasis), which helps limit fatigue and prevents syncope and coma. Specifically, increased ventilation helps eliminate the buildup of carbon dioxide (CO_2) and H^+ and restores acid-base balance. The maintenance of acid-base balance is accomplished with the transport of CO_2 molecules to the lungs primarily as plasma bicarbonate (HCO_3) (McArdle et al., 2010).

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3$$

Equation 2. Chemical equation for formation of carbonic acid.

Bicarbonate ion bonds with hydrogen ions to form carbonic acid (H_2CO_3). The bicarbonate buffer carries the hydrogen ion in the form of carbonic acid to the pulmonary capillaries, where it is reconverted into CO_2 (leaving water [H_2O] in the blood) that can diffuse into the alveoli, and, finally, CO_2 is released into the environment upon exhalation (McArdle et al., 2010).

Light intensity aerobic exercise produces a small O_2 deficit and quickly results in steady-rate O_2 consumption in part because of the response of the circulatory and respiratory systems. Moderate-to-intense aerobic exercise requires more time to achieve steady-rate O_2 consumption.

The increased duration creates a larger oxygen deficit. Oxygen debt or excess post-exercise oxygen consumption (EPOC) refers to the additional O_2 consumption required during recovery above the resting levels of O_2 consumption. EPOC occurs due to the elevated aerobic metabolism that occurs after exercise cessation.

1.3.4 Training-related changes in cardiorespiratory fitness

Endurance training causes an increase in VO_{2max} during aerobic exercise and decreases the time required to attain steady-rate. VO_{2max} improvements in such cases stem from cardiovascular changes. Specifically, endurance training results in an increase in stroke volume as well as increased a-vO_2 difference. In addition, increases in VO_{2max} result from greater muscle blood flow due to an increase in capillary density in muscle tissue. Further increases in VO_{2max} arise from increased mitochondria and oxidative enzymes, particularly in type I slow-twitch oxidative fibers. The changes that lead to improvement in VO_{2max} also result in a faster rise in O_2 uptake at the beginning of exercise. Such increase lessens the O_2 deficit and decreases time to reach steady-state O_2 consumption. This response helps to spare muscle glycogen and blood glucose, which reduces the formation of lactate, and therefore, limits fatigue. In addition, increased exercise-induced blood flow through the liver improves lactate clearance and decreases the amount of lactate in the blood. The limited accumulation of lactate in the blood helps to maintain blood pH. Accordingly, lactate threshold is shifted with training, which helps to explain training-related decreases (McArdle et al., 2010).

Ballet and contemporary dancers have different cardiorespiratory demands because of the physiologic requirements of those different dance styles (Angioi, Metsios, Koutedakis, & Wyon, 2009). A review of professional classical ballet dancers reveals that professional dancers

underperform on aerobic fitness tests (Angioi, Metsios, Twitchett, Koutedakis, & Wyon, 2009; Twitchett, Koutedakis, & Wyon, 2009), which may explain high injury rates (80% injury occurrence) among professional dancers (Wyon et al., 2007). The poor physical conditioning implied by these reports creates a situation in which professional ballet dancers lack reserve when skill decreases due to fatigue. Interestingly, soloists demonstrate greater VO_{2max} than non-soloist members of ballet companies. Furthermore, professional ballet dancers exhibit a lower aerobic capacity than university level ballet dancers. Despite the difference between professional and university level ballet dancers, professional ballet dancers demonstrate lower peak blood lactate levels than university level ballet dancers, which could imply professional ballet dancers move in a highly efficient manner (Twitchett et al., 2009). Nonetheless, one study of professional dancers revealed their ventilatory threshold, thought to occur at the onset of blood lactate accumulation, is similar to thresholds for *nonendurance* athletes (Wyon et al., 2007). Not surprisingly, aerobic and strength training in modern dance students results in improvements in physical fitness parameters such as VO_{2max} and in the technical elements of dance, which dance teachers with professional dance experience judged by assessing the execution of a sequence of movements in accordance with a pre-determined scoring rubric (Koutedakis et al., 2007).

Aerobics instructors demonstrate significantly different physiologic responses in teaching an aerobics routine than aerobics participants. Specifically, although instructors generally have significantly more experience with aerobics classes than participants (6.60 years versus 3.13 years, respectively), instructors exhibit significantly faster average heart rates in the first half of a 30-minute aerobics class and achieve a significantly greater fold of maximum heart rate in the second half of class, than participants. Furthermore, whereas average heart rates rise among instructors—although not significantly—in teaching the second half of a 30-minute aerobics

routine compared to the first half, average heart rates of participants are significantly lower in the second half of an aerobics routine compared to the first half. Both instructors and students exhibit significantly greater average blood lactate levels in the second half of a 30-minute aerobics routine than in the first half. The physiologic response of instructors to a 30-minute aerobics routine, in terms of VO_{2max}, is surprisingly similar across instructors and students. However, elevation of average heart rates and blood lactate levels of instructors in the second half of a 30-minute aerobics routine indicates instructors undergo physiologic stress from physical activity despite their regular participation, suggesting that perhaps instructors require additional endurance training above and beyond what they get while teaching to improve their aerobic fitness and avert such physiologic consequences (Sekulic, Rausavljevi, & Zenic, 2006). That is, for some PAVUs, more training of the cardiovascular and respiratory systems to improve their aerobic fitness level may decrease the acute cardiorespiratory response to physical activity and lead to less competition between respiratory and phonatory functions.

1.4 RESPIRATION

1.4.1 Anatomy of the airway

The respiratory system consists of a series of passageways that permit the movement of air into and out of the lungs, a process called ventilation. The larynx is a transitional organ of respiration, separating the upper from the lower airway. The anatomical division occurs at the level of the true vocal folds, which play an important role in regulating airflow. The lower airway consists of a series of tubes that continually decrease in diameter. Although increasingly smaller in

diameter, the cross-sectional area of the lower airway increases considerably as the number of tubes increases with each division, which effectively reduces the contribution of the narrow, elastic lung tissues to resistance. In fact, the passageways of the upper airway contribute more to resistance than those of the lower airway, providing a total of 25 to 40 percent of the total resistance to airflow (Levitzky, 1995).

An important physiologic distinction is seen at the point of the terminal and respiratory bronchioles. All of the structures above the respiratory bronchioles belong to the conducting airway, which is responsible for moving air into and out of the body. In contrast, the respiratory airway, which consists of the respiratory bronchioles, alveolar ducts, and, finally, alveolar sacs, contains structures for gas exchange (Levitzky, 1995). The large rapid increase in cross-sectional surface area that results with each bifurcation from the small bronchi onward, as well as the abundance of alveoli, facilitates gas exchange (Hlastala & Berger, 2001).

The lung tissue, which contains sacs called alveoli, is the primary site of gas exchange. The tiny alveoli consist of a single layer of thin, squamous epithelial cells called type I alveolar cells. Individual alveoli connect to one another through an interlocking network of elastic and connective fibers called alveolar septa. In addition to the pull from gravitational forces, these shared walls provide the lung tissue with support to resist collapse due to surface tension and smooth muscle contraction. Beds of pulmonary capillaries engulf the alveoli and, together with holes between alveoli, facilitate efficient gas exchange. Despite gravitational forces that distend the lungs and the presence of the surface tension reducing fluid called surfactant, the lungs tend toward a state of collapse (Levitzky, 1995).

Lung tissue is rather passive in ventilation. The thorax (rib cage), which consists of the thoracic portion of the vertebral column, twelve pairs of costal bone and cartilage, the sternum,

the clavicle, and muscle, acts as a mechanism to move air between the lungs and the external environment. Two thin, airtight membranes, the visceral and parietal pleura, link the lungs and rib cage so that they act as a unit. The visceral pleura cover the lungs and the parietal pleura line the inner surface of the rib cage. Each membrane contains a thin layer of liquid that couples the lungs to the rib cage. Two opposing forces at the end of the expiratory phase of quiet breathing, i.e., the lungs' tendency to collapse and the rib cage's tendency to expand, create a slightly negative pressure within the space between the two pleura. Negative intrapleural pressure is the mechanism that links the lungs to the rib cage, facilitating expansion of the lungs when forces act on the thorax and cause the thorax to enlarge (Levitzky, 1995).

1.4.2 Physiology of ventilation

Movement of air into the body always occurs due to active forces. The lung-thorax unit is in a state of equilibrium at the end of tidal (quiet/resting) expiration, in which alveolar pressure is equal to atmospheric pressure due to the sum of passive recoil forces in the lungs and rib cage. In this state, no air will flow into the lungs without the addition of muscular force acting on the lung-thorax unit. Primary muscles of inspiration, which include the diaphragm and external intercostal muscles, contract to enlarge the lung-thorax unit, resulting in a pressure gradient such that lung pressure is smaller than atmospheric pressure. Boyle's Law, which states that volume (V) is inversely related to pressure (P), explains the drop in alveolar pressure as the lungs increase in size.

$$P \propto \frac{1}{V}$$

Equation 3. Boyle's Law.

Accordingly, the pressure inside the lungs becomes lower than atmospheric pressure. When the negative pressure gradient overcomes the resistance to air flow from the upper airways, air moves from the external environment into the lungs (Hlastala & Berger, 2001). During inspiration of larger volumes of air as occurs during HRD, further increases in intrathoracic volume occur as the scalene and sternocleidomastoid muscles contract. In terms of expiration, expulsion of air during quiet breathing results from passive recoil forces. Inspiratory muscles relax and the lung-thorax unit returns to a pre-inflated resting state. Lung tissue recoil forces decrease thoracic dimensions and cause alveolar pressure to increase. Accordingly, alveolar pressure becomes greater than atmospheric pressure and a pressure gradient causes air to flow out of the lungs (Neumann, 2010).

In contrast to the situation during rest breathing, during HRD expiration is also an active process. The interosseous fibers of the internal intercostal muscles contract to exert a downward and inward force on the ribs. Transverse thoracic muscle fibers contract and pull down the upper ribs to which they are attached. The abdominal muscles also contract during active expiration. The recruitment of these muscles causes the lower ribs to move downward (Neumann, 2010). In addition, contraction of abdominal muscles increases intra-abdominal pressure and forces the diaphragm upward (T. J. Hixon, Mead, & Goldman, 1976). Combined, these actions cause increased expiratory airflow, which results in an increase in air volumes flowing out of the lungs compared to rest breathing (Neumann, 2010).

1.4.3 External and internal respiration

Air entering the body contains several types of gases. Nitrogen, oxygen (O_2), carbon dioxide (CO_2), and argon are the most abundant. Inhaled O_2 diffuses across the alveolar-capillary wall within the lungs into the bloodstream. Blood that leaves the lungs is now highly oxygenated. The oxygenated blood returns to the heart through the pulmonary veins. The blood, rich with O_2, is then distributed to somatic tissues through systemic circulation to support aerobic metabolism. Regarding CO_2, it diffuses out of the bloodstream and into the lungs. Once in the lungs, it leaves the body through exhalation. This process of gas exchange between the lungs and bloodstream is external respiration (Hlastala & Berger, 2001). On the other hand, the diffusion of gases into and out of cells is known as internal respiration. This process facilitates aerobic metabolism because of the presence of O_2 in the cell after it separates from hemoglobin and diffuses across the cell membrane. Likewise, CO_2 produced from chemical reactions leaves the muscle cell by diffusing across the cell membrane and into the bloodstream, where it circulates the body mostly as bicarbonate in the serum of the blood (McArdle et al., 2010)

Dalton's law of partial pressures states that the total pressure (P_{total}) of a gas mixture is equal to the sum of the partial pressures (p_i) of the mixture's constituent gases.

$$P_{total} = \sum_{i=1}^{n} p_i$$

Equation 4. Dalton's Law of Partial Pressures.

According to this law, gas molecules behave independently of other gases in the mixture. The pressure that an individual gas exerts as part of the total gas mixture is the partial pressure of that

gas. Gases diffuse into and out of the bloodstream as a result of a pressure gradient between the lungs and the bloodstream or the bloodstream and muscle cells.

Henry's law states that the amount of gas that can dissolve in a liquid is directly proportional to the partial pressure of that gas above the liquid (p) such as blood.

$$p = k_H c$$

Equation 5. Henry's Law.

where p is partial pressure of the solute in the gas above the liquid, c is the concentration of the solute, and k_H is a constant with dimensions pressure divided by concentration.

This gas solubility law refers only to the amount of gas that is dissolved at equilibrium when the partial pressures of a molecule in the gas and liquid phase are equal. Of note, the law is subject to temperature changes. At high temperatures, the solubility coefficient is low and less gas dissolves into a liquid such as blood (blood temperature keeps this coefficient low and helps explain the need for hemoglobin in transporting O_2 to working tissues).

A final important gas law is Fick's First Law of Diffusion. This law describes the behavior of gas molecules as they move across a membrane such as the capillary wall due to a concentration gradient of partial pressures.

$$J = -D\frac{\partial \emptyset}{\partial x}$$

Equation 6. Fick's First Law of Diffusion.

where J is the diffusion rate, D is diffusion coefficient, ∅ is the concentration, and ɤ is the length of the diffusion path. According to Fick's Law, the rate of diffusion increases as the cross-sectional diffusion area and the concentration driving force across the membrane increase. Conversely, the rate of diffusion decreases as the membrane thickness increases (Hlastala & Berger, 2001).

1.4.4 Neural control mechanisms of breathing

Breathing is regulated by specialized control mechanisms. These mechanisms tightly match ventilation patterns to arterial O_2 and CO_2 partial pressures. Rhythmic, automatic breathing characteristics of quiet (at rest) ventilation are regulated by neural circuitry linking central nervous system and respiratory muscle functions (Levitzky, 1995). It is generally accepted that the rostral ventrolateral medulla governs quiet automatic breathing. Within the medullary respiratory center, the pre-Botzinger complex initiates inspiration by transmitting a signal via bulbospinal pathways to motoneurons of the inspiratory muscles (Butler, 2007). Those inspiratory neurons most likely serve to activate the phrenic nerve, which governs diaphragmatic activity (Levitzky, 1995). During quiet breathing, expiration typically occurs when the medulla halts inspiratory firing commands. Another area in the medullary respiratory center, rostral to the pre-Bötzinger complex, is active in expiration involving increased respiratory drive. The two areas, the pre-Bötzinger complex and the area rostral to the pre-Bötzinger complex, are collectively known as the ventral respiratory group, and work together to maintain rhythmic respiration (Butler, 2007).

Although rhythm generation is an essential function of the medullary respiratory center, the firing pattern of inspiratory neurons may vary depending on feedback from chemoreceptors,

baroreceptors, and mechanoreceptors. These receptors communicate with the dorsal respiratory group in the medullary respiratory center to regulate respiratory rate. For example, during exercise, central and peripheral chemoreceptors sense increases in arterial CO_2 partial pressures, and such information is transmitted via afferent pathways back to the medulla to stimulate respiratory drive. Afferent fibers of the ninth (glossopharyngeus) and tenth (vagus) cranial nerves carry information about gas partial pressures and pH from the carotid and aortic arterial chemoreceptors as well as arterial blood pressure from carotid and aortic baroreceptors. Accordingly, ventilatory compensations for alterations in homeostasis occur from the integration of intrinsic rhythm pattern generation of the medullary respiratory center with input from chemoreceptors (Levitzky, 1995).

Control of breathing alters as a result of reflex mechanisms in the lungs, cardiovascular system, and other areas of the body including muscles and tendons. The pharyngeal dilator reflex is triggered by negative pressure in the upper airway. Receptors in the nose, mouth, and upper airways transmit afferent information via trigeminal, glossopharyngeal, and vagus nerves, causing a contraction in the pharyngeal dilator muscles. Another class of reflexes, originating in the cardiovascular system, influence ventilatory behavior via arterial chemoreceptors and arterial baroreceptors. Arterial chemoreceptors in the carotid arteries and aorta monitor the local chemical environment. Afferent information from these receptors stimulates ventilation via a negative feedback loop to tightly match alveolar ventilation and gas partial pressures. More specifically, arterial chemoreceptors respond to a decrease in O_2 partial pressures, an increase in CO_2 partial pressures, and a decrease in blood pH. CO_2 partial pressures probably exert the greatest influence on the respiratory control center's activity. However, given the close relationship between CO_2 and hydrogen ion concentration, a change in pH also exerts a strong

influence on ventilation. Elevated CO_2 partial pressures result in greater tidal volumes and increased breathing frequency. Peripheral arterial chemoreceptors have the capacity to influence breath-by-breath ventilatory behavior in response to arterial blood conditions. An increase in CO_2 partial pressures, hydrogen ion concentration, or both can also be detected in cerebral spinal fluid by central chemoreceptors located in the medulla. In addition to stimulation of ventilation, afferent information from arterial chemoreceptors also leads to bronchoconstriction, dilates the upper airway, and increases blood pressure. Finally, in the initial moments of physical activity, when stimulated, proprioceptors in muscles, tendons, and joints typically result in hyperpnea (increased ventilation). Information from stimulation of these receptors ascends to the medullary respiratory center via the spinal cord (Levitzky, 1995). Breath-by-breath analysis by a metabolic analyzer allows for non-invasive measurement of oxygen consumption by muscle tissue to support aerobic metabolism, carbon dioxide production from metabolic processes that results in the production of ATP, and the concentration of exhaled carbon dioxide that peaks at the end of the breath also known as end-tidal carbon dioxide.

Higher brain centers during special acts of breathing such as those that occur during speech transmit signals that the medulla integrates to influence respiratory activity. The integration of information from cortical areas permits an interruption of the normal rhythmic breathing pattern generation. However, automatic pathways and voluntary pathways may control breathing independently. Evidence in humans suggests that direct corticospinal pathways to inspiratory muscles may exist, thus circumventing the medullary respiratory center. In fact, the diaphragm has cortical representation in primary motor cortex. If this dual control system does in fact exist, final integration of cortical and bulbar neuronal signals would occur at the level of the motor neuron. Timing and strength of the final signal reaching respiratory muscle motor neurons

would ultimately depend on medullary and cortical inputs as well as other input via networks of interneurons (Butler, 2007).

1.4.5 The role of the larynx in airflow regulation

As noted, the vocal folds, housed within the larynx, function as a gateway in the regulation of respiratory homeostasis (i.e., maintenance of optimum blood-gas concentrations). Laryngeal configuration varies for different functions. Vocal fold dynamics do not reflect a simple dichotomous open or closed arrangement. Intrinsic laryngeal muscles (muscles having both origin and insertion within the larynx) activate at varying time points in the respiratory cycle and to different levels to accomplish highly specific adjustments. The coordinated involvement of specific intrinsic laryngeal muscles during different phases of the respiratory cycle highlights the important role these muscles play in actively regulating airflow (T. Brancatisano, Collett, & Engel, 1983). The movement of the vocal folds depends on the phase of the respiratory cycle (T. Brancatisano, Collett, et al., 1983; England, Bartlett, & Daubenspeck, 1982). Through most of the inspiratory phase in quiet breathing, the vocal folds remain in an abducted (separated) position. In fact, once the expiratory phase nears completion, before the onset of inspiration, the vocal folds move laterally to assume an abducted position. This movement widens the glottic aperture (the space between the vocal folds) in anticipation of the inspiratory phase of the respiratory cycle (England, Bartlett, & Daubenspeck, 1982). Peak opening of the glottis occurs at mid-inspiration (T. Brancatisano, Collett, et al., 1983). The open position of the vocal folds decreases laryngeal resistance during inspiration. Accordingly, airflow reaches peak values as laryngeal resistance decreases around the middle of the inspiratory phase of the respiratory cycle. Regardless of lung volume, the glottic aperture is greater during inspiration than expiration in

normal individuals (T. Brancatisano, Dodd, & Engel, 1983). As such, airway resistance is significantly lower during inspiration than expiration (England, Bartlett, & Daubenspeck, 1982). In fact, two intrinsic laryngeal muscles that cause the vocal folds to close and increase laryngeal resistance – TA and IA – demonstrate complete cessation or at least a substantial decrease in activity during inspiration of large tidal breaths (Kuna, Insalaco, & Villeponteaux, 1991; Kuna, Insalaco, & Woodson, 1988).

During inhalation, the posterior cricoarytenoid (PCA) exhibits increased phasic activity that corresponds with a widening of the glottis. However, the onset of PCA activity, congruent with the onset of vocal fold abduction, occurs prior to initiation of inspiratory flow (T. P. Brancatisano, Dodd, & Engel, 1984). Another intrinsic laryngeal muscle, the cricothyroid (CT), also demonstrates phasic activity during inhalation. Like the PCA, the CT has a progressive rise in activity near the end of expiration, peaking around mid-inspiration. Again, the onset of increased CT activity precedes inspiratory flow (Wheatley, Brancatisano, & Engel, 1991). Although the PCA is the only intrinsic laryngeal muscle that, by itself, can abduct the vocal folds (Ludlow, 2005), a rise in CT activity preceding inspiration causes increased vocal fold tension and prevents them from "falling" into the airway due to large inspiratory pressures. Without concurrent activation of the PCA, CT activation would function to elongate the vocal folds and this action would bring the vocal folds closer together. However, the combined activity of the PCA and CT widens the glottic aperture and decreases laryngeal resistance (Woodson, 1990).

During expiration, synergistic activation of multiple intrinsic laryngeal muscles causes the vocal folds to move toward midline and narrow the glottis. Toward the end of the inspiratory phase of the respiratory cycle in quiet breathing the vocal folds move medially and narrow the glottic aperture. This movement precedes expiratory flow. The vocal folds remain in a narrowed

configuration for the majority of the expiratory phase of the respiratory cycle. The size of the glottic aperture during exhalation is positively correlated with expiratory airflow during quiet tidal breathing such that greater expiratory flow occurs with a larger glottic aperture. However, compared with inspiration, the glottic aperture is smaller during expiration and, accordingly, airway resistance is increased (England, Bartlett, & Daubenspeck, 1982). The glottic aperture narrows even more at high lung volumes (T. Brancatisano, Dodd, et al., 1983). The tight coupling of glottic aperture and lung volume demonstrates that exhalation during quiet breathing is not at all passive. Rather, a narrowed laryngeal configuration increases airway resistance or "brakes" air flow to slow lung emptying (T. Brancatisano, Collett, et al., 1983).

Specific muscular activity in expiration is as follows. Glottic aperture decreases during expiration due to a combination of increased activity of intrinsic laryngeal muscles that adduct and tense the vocal folds as well as decreased activation of the sole abductor muscle, the PCA. First, the activity of the PCA rapidly decreases, although not completely, to reduce glottic opening (T. P. Brancatisano et al., 1984). Without PCA activity the vocal folds are no longer kept in an open position and, as a result, move medially. Additionally, the CT shows a dramatic decrease in activity near the end of the inspiratory phase of the respiratory cycle (Wheatley et al., 1991). The decline in CT activity decreases vocal fold tension and allows the folds to shorten. In addition to the lower activation levels of the PCA and CT, during expiration the glottis narrows from increased activation of intrinsic laryngeal muscles that adduct (close) the vocal folds. Both the thyroarytenoid (TA) and interarytenoid (IA) muscles demonstrate dramatic increases in phasic activity near the onset of the expiratory phase of the respiratory cycle (Kuna et al., 1991; Kuna et al., 1988). IA muscle contraction draws the vocal folds toward midline and TA activation shortens and bulges the vocal folds. In addition, TA muscle contraction increases the

intrinsic tension of the vocal fold. Both muscular actions contribute to the increase in expiratory resistance. Towards the end of inhalation of larger lung volumes, the IA demonstrates substantially increased activity that continues into expiration (Kuna et al., 1991). Interestingly, the IA and TA demonstrate different discharge patterns (Kuna et al., 1991; Kuna et al., 1988). Longer expiratory times and greater expiratory resistance are associated with greater TA activation (Kuna et al., 1988). Likewise, large increases in IA activity toward the end of expiration are associated with changes in expiratory flow and expiratory time (Kuna et al., 1991).

The glottis increases in size during inspiration in HRD that results from physical activity or chemical stimulation (England & Bartlett, 1982), consequently decreasing laryngeal resistance (A. Brancatisano, Dodd, & Engel, 1991) and facilitating increased minute ventilation (see Table 1) (A. Brancatisano et al., 1991; England, Bartlett, & Knuth, 1982; Kuna, Insalaco, Villeponteaux, Vanoye, & Smickley, 1993). This increase in minute ventilation seems to occur due to increased tidal volume (England & Bartlett, 1982) and increased breathing frequency (England & Bartlett, 1982; England, Bartlett, & Knuth, 1982). During inspiration, the distance between the vocal folds is slightly greater while exercising than during rest (England & Bartlett, 1982), thus decreasing inspiratory laryngeal resistance. Although a widening of the glottic aperture occurs during inspiration, inspiratory time remains relatively stable during chemically-stimulated HRD from inspiring CO_2 (hypercapnia) compared to rest (England, Bartlett, & Knuth, 1982). However, increased glottic opening during exercise and chemically-stimulated hypercapnic (increased CO_2) conditions result in reduced expiratory time (England & Bartlett, 1982; England, Bartlett, & Knuth, 1982) and increased expiratory airflow (England, Bartlett, & Knuth, 1982). Despite a change in inspiratory time, breathing frequency increases with chemically-stimulated hypercapnic conditions (Woodson, 1990), and this change seems to be

mediated by the decrease in expiratory time (England & Bartlett, 1982; England, Bartlett, & Knuth, 1982).

Intrinsic laryngeal muscle activity during quiet breathing in conditions of HRD differs from quiet breathing in eupnea or normal drive (see Table 1). During HRD, increased CT and PCA activity occurs during both inspiration and expiration (Woodson, 1990). First, the PCA exhibits an increase in phasic activity during the inspiratory phase of the respiratory cycle, widening the glottis (A. Brancatisano et al., 1991). In addition to the change in PCA muscle activity on inspiration, tonic and phasic IA muscle activity decreases with progressive increases in CO_2 to further widen the glottic aperture (Kuna et al., 1993). The increase in glottic size results in decreased laryngeal resistance to airflow and allows larger volumes of air to move into the lungs (England, Bartlett, & Knuth, 1982). The PCA muscle also demonstrates an increase in tonic activity (during both inspiration and expiration) (A. Brancatisano et al., 1991). Accordingly, the glottis increases in size not only during the inspiratory phase of the respiratory cycle but also during the expiratory phase in HRD relative to rest (England & Bartlett, 1982). Once again, the larger glottis decreases laryngeal resistance to airflow and facilitates the movement of larger amounts of air out of the lungs during the expiratory phase of the respiratory cycle.

Table 1. Intrinsic Laryngeal Muscle Activity and Its Impact on Vocal Fold Movement and Laryngeal Resistance During HRD Quiet Breathing in Relation to Normal Drive

In Relation to Normal Drive	Muscle Activity and their Laryngeal Effects	
	Inspiration	*Expiration*
PCA	Increased	Increased
CT	Increased	Increased
IA	Decreased	Decreased
TA/LCA	Decreased	Decreased
Vocal folds	↑ glottic aperture	↑ glottic aperture
Laryngeal Resistance (in the absence of phonation)	Decreased	Decreased

Note. PCA = posterior cricoarytenoid; CT = cricothyroid; IA = interarytenoid; TA = thyroarytenoid; LCA = lateral cricoarytenoid

The sole vocal fold ABductor is not the only muscle to have a change in activity during HRD. The vocal fold ADductors actually exhibit decreases in phasic and tonic activity under such conditions (England, Bartlett, & Knuth, 1982; Kuna et al., 1993), which results in decreased laryngeal resistance and aids in ventilation. Interestingly, IA muscle activity demonstrates abrupt bursts in the transition from inspiration to expiration, which is associated with a decrease in glottic area (Kuna et al., 1993). TA muscle activity also demonstrates decreases in phasic and tonic activity with progressive increases in CO_2 levels but the decrease occurs more rapidly in tonic activity. In fact, TA muscle activity completely disappears when CO_2 levels are large. Such cessation facilitates expiration by augmenting expiratory air flow and decreasing expiratory time (Insalaco, Kuna, Cibella, & Villeponteaux, 1990).

To conclude, in both normal and high respiratory drive, co-activation of PCA and CT widens the vocal folds during inhalation to decrease laryngeal resistance and permit the flow of air into the lungs, although more extreme vocal fold opening occurs in HRD as activation levels of those two muscles increase and IA and TA/LCA activation levels decrease. In exhalation, a similar increase in PCA and CT muscle activation occurs during HRD to provide maximal

opening of the glottic aperture in HRD, and as ventilation needs increase, IA and TA/LCA muscles demonstrate diminished activation levels. These activation patterns provide maximum airway patency, creating optimum conditions for ventilation during exercise that results in larger tidal volumes, greater airflow rates, and shorter inhalatory and exhalatory times. These findings reveal that the larynx is an organ actively involved in ventilation, of which the movement of the vocal folds helps regulate airflow for effective and efficient gas exchange.

1.5 SPEECH BREATHING

Speech involves volitional acts of breathing. Several studies have examined the influence of speech on ventilation and energy expenditure (Bunn & Mead, 1971; Hoit & Lohmeier, 2000; Meanock & Nicholls, 1981; Russell, Cerny, & Stathopoulos, 1998; Warner, Waggener, & Kronauer, 1983). Individuals demonstrate a high degree of consistency over successive trials of continuous speaking in a day (Hoit & Lohmeier, 2000). Reading aloud at comfortable sound pressure level (SPL) is characterized by lower breathing frequencies and greater tidal volumes as compared to resting tidal breathing (Bunn & Mead, 1971). However, individuals increase breathing frequency, using shorter inspiratory times and greater expiratory times, when speaking loudly compared to quiet breathing (Russell et al., 1998).

Resting tidal breathing occurs with minimal energy cost. Although speech breathing patterns differ from quiet breathing, typically breathing during the production of comfortable speech interferes little with the body's gas exchange needs and occurs in a manner that imposes minimal energy requirements. In fact, most studies have demonstrated that, while stationary, continuous reading (Bunn & Mead, 1971; Hoit & Lohmeier, 2000; Meanock & Nicholls, 1981)

and conversation (Warner et al., 1983) at a comfortable pitch and loudness result in only slightly more air exchange compared to quiet breathing, which results in minimal speaking-related hyperventilation. However, loud speech results in large minute ventilation values (Russell et al., 1998), and consequently, end-tidal CO_2 levels detected from expired air demonstrate a decrease from resting levels during quiet breathing (Bunn & Mead, 1971; Russell et al., 1998). This finding suggests speaking-related hyperventilation as a result of speaking loudly. In terms of gas exchange, O_2 consumption has not been found to change significantly from resting levels when speaking in a comfortable, or habitual manner (Hoit & Lohmeier, 2000; Russell et al., 1998). However, altering speech breathing from comfortable to, for instance, loud speech, leads to greater O_2 consumption and CO_2 production than quiet breathing (Russell et al., 1998). As such, any deviation from comfortable, for example, using increased loudness [+10 dB above comfortable], poses a challenge to maintaining optimal blood-gas concentrations (i.e., respiratory homeostasis). Interestingly, individuals demonstrate compensatory post-speech behaviors even in resting speech, presumably to restore respiratory homeostasis and achieve normal blood-gas concentrations. That is, following just minimal hyperventilation that occurs from speaking comfortably at rest, individuals delay inhalation entirely by holding their breath (moments of apnea) (Bunn & Mead, 1971) or decrease the rate and extend the duration of inhalation, as CO_2 levels are low (Abel, Mottau, Klubendork, & Koepchen, 1987, as cited in T.J. Hixon & Hoit, 2006).

These findings reflect the ability of speakers to override automatic ventilatory behavior that functions to tightly match gas exchange with the body's metabolic needs. Although speakers have the capacity to accomplish linguistic needs despite minor disturbances to ventilation, peripheral feedback mechanisms from chemoreceptors kick in to return blood-gas levels and

blood pH levels to baseline levels through altered ventilatory behavior after speech has ceased (Bunn & Mead, 1971). The adaptive nature of the respiratory apparatus permitting speech even with minor challenges to normal ventilation highlights a degree of accommodation. However, whereas speech in normal respiratory drive conditions results in only minor disturbances to gas exchange, speaking during HRD creates competition between metabolic needs and linguistic demands (Bailey & Hoit, 2002; Hoit, Lansing, & Perona, 2007). Studies investigating speech breathing in HRD conditions have thus far stimulated the change in ventilation chemically (Bailey & Hoit, 2002; Bunn & Mead, 1971; Hale & Patrick, 1987; Hoit et al., 2007; Meanock & Nicholls, 1981; Phillipson, McClean, Sullivan, & Zamel, 1978) or with physical activity (Baker, Hipp, & Alessio, 2008; Doust & Patrick, 1981; Meanock & Nicholls, 1981; Meckel, Rotstein, & Inbar, 2002; Otis & Clark, 1968; Rotstein, Meckel, & Inbar, 2004). Despite different methods for inducing HRD, remarkable similarities have emerged across studies. Results have shown that speech in HRD reduces minute ventilation compared to quiet breathing in HRD (Bailey & Hoit, 2002; Baker et al., 2008; Bunn & Mead, 1971; Doust & Patrick, 1981; Hale & Patrick, 1987; Hoit et al., 2007; Meckel et al., 2002; Otis & Clark, 1968) by as much as 55% of control values (Doust & Patrick, 1981). Thus, the limited ventilation that occurs in speech under HRD conditions compromises the regulation of gas partial pressures and elimination of metabolic waste produced during physical activity. In spite of the obstructive nature of speech, both minute ventilation and expiratory airflow increase in speech during HRD compared to speech in normal drive, and that response is different depending on the magnitude of the perturbation (Bailey & Hoit, 2002; Baker et al., 2008; Doust & Patrick, 1981; Hoit et al., 2007). Furthermore, whereas minute ventilation during breathing remains consistent, it increases during simultaneous exercise and speaking over time, which reveals an ongoing attempt by the body to maintain adequate gas

partial pressures by making further adjustments in ventilation (Bailey & Hoit, 2002; Baker et al., 2008; Doust & Patrick, 1981; Hoit et al., 2007). Accordingly, the ventilatory response increases with greater disturbances in partial pressures of O_2 and CO_2.

Although several studies have found that expiratory flow increases during speech in HRD as compared to speech under normal respiratory drive (Bailey & Hoit, 2002; Bunn & Mead, 1971; Doust & Patrick, 1981; Hoit et al., 2007), this increased expiratory flow is mainly linked to the addition of nonphonated expirations, frequently at the ends of phrases, to expel accumulated CO_2. Phonated expirations demonstrate only a slight increase in airflow compared to comfortable speech under normal drive. In non-phonated breathing under HRD, minute ventilation demonstrates an increase from 27 L/min to 78 L/min, while average phonated ventilation shows a smaller change with increasing exercise intensity, from 18 L/min to 30 L/min (Doust & Patrick, 1981).

Speech breathing kinematics are also affected by HRD conditions. Under such conditions, individuals initiate and terminate speech at higher lung volumes than during quiet breathing. Larger rib cage volume mediates the increase in lung volume both at initiation and termination of expiration during speech in HRD as compared to quiet breathing (Bailey & Hoit, 2002; Hoit et al., 2007). These larger than normal lung volumes may result from a change in an individual's ability to voluntarily control lung volume when CO_2 partial pressures increase (Katz-Salamon, 1986).

Individuals report more difficulty with speaking tasks as ventilation increases (Bailey & Hoit, 2002; Hoit et al., 2007; Otis & Clark, 1968; Rotstein et al., 2004). The relationship between speaking-related dyspnea and ventilatory drive seems nonlinear and, for some individuals, greater difficulty with speech production coincides with the ventilatory threshold. Those findings

suggest that some individuals demonstrate an association between perceived difficulty in producing speech and exercise intensity (Rotstein et al., 2004). Subjects report three main perceptions under such conditions: air hunger, physical exertion, and mental effort. Of those perceptions, statements about mental effort are the most frequently reported (Hoit et al., 2007). Reports of mental effort can be construed to reflect competition between behavioral and automatic respiratory control (Hoit et al., 2007; Rotstein et al., 2004).

Interestingly, people tend to maintain intelligible speech throughout a range of exercise intensities despite perceived difficulty speaking under HRD (Rotstein et al., 2004). Although speech remains comprehensible in the face of the body's response to a physiologic stressor, studies have shown that when the intensity of HRD increases, metabolic needs begin to override linguistic demands, resulting in the alteration of speech phrasing (Bailey & Hoit, 2002; Baker et al., 2008; Otis & Clark, 1968). Speech becomes truncated, characterized by a decrease in the number of syllables produced per breath, and pauses occur at locations other than sentence, clause, and phrase boundaries (Bailey & Hoit, 2002; Otis & Clark, 1968). Furthermore, alteration in linguistic strategies becomes more common as ventilatory drive increases. For example, more inhalations are initiated at nonlinguistic junctures (i.e., within phrases or clauses) (Bailey & Hoit, 2002; Hoit et al., 2007; Otis & Clark, 1968).

Speaking in HRD negatively affects the body's response to a physiologic stressor (Meckel et al., 2002). As already noted, speaking under HRD results in hypoventilation compared to non-phonated breathing in HRD. Consequently, decreased ventilation leads to increased CO_2 (hypercapnia). Furthermore, O_2 consumption and the ventilatory equivalent for O_2 consumption (minute ventilation divided by O_2 consumption) significantly decrease. In addition, blood lactate levels are significantly greater during exercise while talking as compared to

exercise alone, and this increase is greatest at lower exercise intensities (Meckel et al., 2002). Accordingly, minute ventilation rises and overshoots baseline levels during HRD quiet breathing abruptly after cessation of speaking (Doust & Patrick, 1981; Phillipson et al., 1978), suggesting the body compensates for decreased gas exchange during speaking in HRD compared to quiet breathing. Doust and Patrick (1981) found that in the first 15 seconds after termination of speech, mean ventilation was 114% of the mean control value. Substantial alterations of metabolic needs through physical activity or chemical stimulation demonstrate that tolerance to an additional disturbance such as speech may exceed the ventilatory reserve of the system. In addition, the disequilibrium and change from normal ventilatory behaviors has consequences for energy expenditure.

1.6 PHYSIOLOGY OF PHONATION

Mechanical coupling of the vocal folds to the lungs-thorax unit forms a system that generates voice. Phonatory sound generation requires approximation of the vocal folds during the exhalatory phase of the respiratory cycle (Scherer, 2006). During voice production, the vocal folds function as a complex oscillator that transforms pulmonary airflow into a laryngeal sound source, ultimately perceived as voice (I. R. Titze, 2002). During a brief period just before the opening phase, LCA and IA contract to draw the vocal folds together, closing the membranous and cartilaginous portions of the glottis, and TA activates to increase internal vocal fold tension. Intrinsic laryngeal muscle activation, in addition to viscoelastic properties of the vocal folds, causes resistance to flow that must be overcome. Under such conditions, the lungs-thorax unit contracts to increase alveolar pressure so that air molecules leave the lungs and accumulate

underneath the vocal folds. At the point when this subglottal pressure becomes large enough to overtake the resistance provided by the vocal folds, an opening phase begins in which tissue deformation occurs and the vocal folds begin to separate. According to a classic understanding, as the air escapes through the glottis, two mechanisms serve to bring the vocal folds back together. First is the elastic recoil of the vocal folds. Second, the Law of Continuity and the Bernoulli principle combine to create a reduction in inter-vocal fold pressure, even negative pressure, assisting with vocal fold closure and the cycle thus continues. This theory is called the myoelastic-aerodynamic theory of vocal fold vibration (I.R. Titze, 2000; Janwillem Van den Berg, 1958).

However, the myoelastic-aerodynamic theory does not capture an important feature of vocal fold behavior during phonation. This mechanical system is clearly capable of self-sustained oscillation, which several basic principles govern for vocal fold oscillation to result in a self-sustained state. First and foremost, self-sustained oscillation requires steady airflow. A mechanical system forms between airflow and vocal folds. As a constant energy source, continuous glottal airflow overcomes mechanical energy losses in the vocal folds, exciting the vocal fold tissue and permitting repeated back-and-forth movement. In the case of systems that are capable of self-sustained oscillation, the system requires a component of the positive net energy flow, or driving force, to occur in phase with tissue velocity. State differently, a velocity-dependent driving force supplies energy to vocal folds. Whereas in-phase the driving force supplies energy to the system, out-of-phase, the system would lose energy and extinguish vocal fold oscillation (I. R. Titze, 1988).

Achievement of self-sustained oscillation from a velocity-dependent driving force occurs in part from a changing glottal shape during two phases of the vibratory cycle, an opening and

closing phase (I. R. Titze, 1988). This model of vocal fold vibration assumes a body-cover model of the vocal folds, in which a wave through the cover propagates over the body of the vocal folds, to create alternating glottal geometry during vocal fold opening versus closing (Hirano, 1985). In a two-mass model of healthy phonation, the vocal folds vibrate as a coupled 11-mode oscillator that exhibits medial-lateral and vertical motion (I. R. Titze, 1976). Accordingly, at the outset of their outward excursion, the vocal folds are wider at the bottom than at the top, creating what is called a convergent glottis. Conversely, in a divergent glottis, the vocal folds are narrower at the bottom than at the top. The unique glottal geometry during opening and closing phases of vocal fold vibration causes different intraglottal pressure distributions. Average pressure within the glottis is greater during a convergent glottis compared to a divergent glottis. Without varying glottal area during different phases of the vibratory cycle, the Bernoulli force would be in opposition to the tissue velocity in the initial moments of the opening phase, diminishing the energy provided upon vocal fold closure (I. R. Titze, 1988).

As the vocal folds open, airflow through the glottis increases gradually. Relative to pressures, the vocal tract input pressure at the exit of the glottis increases above atmospheric to accelerate the air column in the vocal tract in tandem with accelerating glottal airflow. This acceleration moves the air column into the vocal tract in a rostral direction and corresponds to an increasing intraglottal pressure. Energy supplied to the vocal folds due to this driving pressure occurs in the direction of a positive tissue velocity during the outward movement of the vocal folds from the bottom to the top edge. In the case of a convergent glottis, a large intraglottal pressure exerts a force on the medial surface of the vocal folds during positive tissue velocities and assists in opening the vocal folds. The energy drives the vocal folds apart. The upper margin of the vocal folds takes longer to open than the lower margin, and consequently, tissue

displacement occurs ahead of glottal airflow. Therefore, peak airflow occurs after maximal tissue displacement (I. R. Titze, 1988).

The elastic component in each vocal fold creates a restoring force that causes each vocal fold to recoil toward midline. In addition to vocal fold elasticity, as the vocal folds return and the glottis is closing, the supraglottic air column continues its ascent. This forward momentum of the air column results in a lowering of vocal tract input pressure. Thus, average glottal pressure is lowered. Equally, and in the case of the divergent glottis, the lag in closing of the top margin of the vocal folds relative to the bottom lowers the intraglottal pressure, and at some point, may become negative. As a result, the driving pressure on the vocal fold tissue decreases, resulting in a diminished in-phase driving pressure with a negative tissue velocity during the inward movement of the vocal fold tissue. The amount of deceleration in flow depends on how quickly the vocal folds close but, in general, air flow through the glottis slows abruptly during the closing phase. During the closed portion of vocal fold vibration, airflow decreases to near zero in most cases (I. R. Titze, 1988). The cycle becomes repetitive, producing an average of about 110 Hz in typical speech for healthy adult men (Hollien & Shipp, 1972) and about 220 Hz in healthy adult women (Stoicheff, 1981).

The larynx in phonation acts as either a low or high impedance source for regulating glottal airflow for increasing glottal efficiency to yield the greatest vocal intensity. That is, the vocal folds provide opposition to the flow of air with the ultimate goal of providing maximum transfer of aerodynamic energy into acoustic output. In accordance with a theory of maximum power transfer, a more capacious laryngeal vestibule maximizes vocal output when matched with loose adduction of the vocal folds and low glottal resistance. Conversely, tight adduction of the vocal folds and increased glottal resistance requires a narrow laryngeal vestibule for maximizing

vocal output. Under those two conditions, impedance matching of the vocal tract and glottis converts maximum aerodynamic power into acoustic energy that radiates from the mouth (I. R. Titze, 2002).

1.6.1 Control strategies in the regulation of vocal intensity

Vocal intensity, that is the amount of acoustic energy radiated per second from the mouth into the air, per unit area, varies with alveolar pressure, vocal fold adduction, fundamental frequency, and vocal tract adjustments (I.R. Titze, 2000). Specifically, the acoustic power generated at the glottis increases as the lungs produce larger alveolar pressures (Finnegan, Luschei, & Hoffman, 2000; Stathopoulos & Sapienza, 1993). Larger lung pressures result in larger AC flows through the glottis during phonation. These pressures also result in faster vocal fold closure and an increase in speed quotient, which causes the glottal airflow waveform to skew more, resulting in an increase in high harmonics in the acoustic signal (I.R. Titze, 2000). Specifically, change in glottal source power is related to the amount of excess lung pressure over phonation threshold pressure (the minimum pressure required to initiate and sustain vocal fold vibration). The glottal source power increases by 8-9 dB for every doubling of excess lung pressure over threshold. Of that gain, a 6 dB increase in glottal source power after controlling for fundamental frequency, open quotient, and speed quotient comes from a doubling of excess pressure over threshold (I. R. Titze & Sundberg, 1992).

Regarding adduction, the acoustic power generated at the glottis changes with the degree of arytenoid and presumably membranous vocal fold adduction, as measured by the distance between the vocal processes at phonation onset (Berry et al., 2001; Stathopoulos & Sapienza, 1993). An optimal voicing pattern relevant for a wide range of both healthy individuals and those

with vocal pathology is defined as a pattern that optimizes the ratio of voice output intensity divided by vocal fold impact intensity. For typical phonation conditions, this objective is achieved when the vocal processes are barely touching or barely separated (approximately 0.6-0.7 mm between the vocal processes) (Berry et al., 2001). Especially in utterances involving some degree of semi-occluded vocal tract, the corresponding perception is “resonant voice,” which involves anterior oral vibratory sensations in the context of perceived phonatory “ease.” This voicing pattern is widely used as a training target for much speech and singing, as well as much voice therapy for a wide range of pathologies, due to its ability to generate relatively strong output intensities while at the same time minimizing pathogenic inter-vocal fold impact stresses (Berry et al., 2001; Stemple & Fry, 2009; Verdolini, Druker, Palmer, & Samawi, 1998; Verdolini-Marston, Burke, Lessac, Glaze, & Caldwell, 1995).

Other voicing patterns include pressed and breathy, which are the perceptual correlates to extremes in laryngeal adduction. The loose adduction associated with breathy voice results in increased phonatory airflow and a perceptually breathier voice compared to normal. In addition, maximum flow declination rate and glottal source power decrease. Conversely, tight adduction results in airflow decreases and a decrease in glottal source power. Perceptually, the voice sounds pressed (I.R. Titze, 2000). In both breathy and pressed voice, as adduction deviates from an optimal configuration, vocal intensity decreases, if all other factors are kept constant (e.g., fundamental frequency and subglottic pressure) (Berry et al., 2001).

Glottal source power is also dependent on fundamental frequency. After controlling for other factors, for every doubling of fundamental frequency at a constant pressure value, glottal source power increases 6 dB. Therefore, increasing fundamental frequency from 100 Hz to 200 Hz would result in an increase in glottal source power of 6 dB Titze & (I.R. Titze, 2000).

The last mechanism available to increase vocal intensity involves tuning of the vocal tract, also known as formant tuning. The vocal tract acts like an amplifier in boosting the acoustic power (i.e., tuning) of source harmonic frequencies that correspond with formants of the vocal tract, producing a higher overall vocal intensity and a perceptually louder voice (Carlsson & Sundberg, 1992; Johan Sundberg, 1994). Frequencies close to one of the vocal tract's formants receive an intensity boost. Conversely, acoustic energy of harmonic frequencies diminishes if the source harmonic frequency aligns poorly with formants. Furthermore, the vocal tract tends to preferentially boost acoustic energy of higher frequencies, in general, due to their proximity to the first two formant frequencies (I.R. Titze, 2000). Consequently, the vocal tract provides a 10-15 dB boost in energy (I. R. Titze & Sundberg, 1992). However, classical singers and actors regularly employ formant tuning by altering the vocal tract to perfectly align a harmonic with a formant, which in the case of singers allows them to overcome the intensity of an orchestra, especially because the so-called "singer's formant" generally occurs in the range of about 3kHz, which corresponds to a relative "hole" in the orchestral spectrum (J. Sundberg, 1977). Formant tuning becomes especially important at high frequencies in which the spacing between harmonics widens compared to low frequencies. In cases of widely spaced harmonics, or when the formant frequency is lower than the fundamental frequency, the source does not receive an energy boost. To avoid that problem, especially present in sopranos, the position of articulators changes to affect the first two formants so they coincide with partials of the fundamental frequency or the fundamental frequency itself (J. Sundberg, 1973, 1977; I.R. Titze, 2000). This unique skill of singers increases vocal intensity by an additional 3-5 dB at higher fundamental frequencies (I. R. Titze & Sundberg, 1992). To conclude, individuals have a variety of methods for increasing vocal intensity, some of which minimize impact stress and preserve

vocal fold tissue health, as is the case with slightly abducted vocal folds in resonant voice or with the use of formant tuning.

1.6.2 Laryngeal airway resistance during phonation

The respiratory and laryngeal systems have a dynamic interaction during voice production. Clearly, the two systems play key roles in initiating and sustaining vocal fold oscillation (I. R. Titze, 1988) but they also possess a shared function in regulating vocal intensity (Finnegan et al., 2000; Thomas J Hixon, Goldman, & Mead, 1973; Stathopoulos & Sapienza, 1993). Of clinical relevance, aerodynamic forces and laryngeal configuration contribute to increases in mechanical stress that have the potential to negatively impact vocal fold tissue health (Titze, 1994). Naturally, addressing their interconnectedness seems reasonable in order to understand the collaboration of the two systems in voice production and to the physical health of the vocal folds.

Laryngeal airway resistance (R_{law}), a recently proposed control parameter of vocal function (Gillespie, 2013; T.J. Hixon, Weismer, & Hoit, 2008), reflects that synergistic relationship between the respiratory and laryngeal systems. Stated differently, thoracic and laryngeal kinematics demonstrate interdependence as they regulate the flow of air through the glottis during phonation. As previously explained, during voicing the respiratory system increases alveolar pressures to drive air through the glottis. Airflow is generated as a result of a pressure gradient created by discontinuity across pressure below as compared to above the vocal folds (ambient pressure), or translaryngeal pressure. Slightly before the creation of this gradient, the vocal folds adduct and provide resistance, to regulate the flow of air through the glottis. Such flow is called translaryngeal airflow. R_{law} is the ratio of translaryngeal pressure to translaryngeal airflow (Baken & Orlikoff, 2000). Thus, it is an appropriate measure of respiratory and laryngeal

coordination and provides an understanding of the coupling between those two mechanical systems. In combining both subglottal pressure and airflow components, R_{law} also reflects the sum of muscle activation patterns of the respiratory and laryngeal subsystems thereby permitting an understanding of vocal motor control. Under the assumption that voice motor control is integrative and interactive, looking at the individual components of the combined system provides a false impression about the way in which the respiratory and laryngeal systems coordinate in phonation. Disconnecting them leads to an artificial distinction in functions as they relate to voice production, as both systems are necessary for self-sustained oscillation. Accordingly, the study of R_{law} offers the opportunity to gain an understanding of motor control of respiration for metabolic breathing versus for voice production.

Despite the conceptual centrality of these constructs and thus their empirical verification, especially transglottal pressure is not necessarily straightforward to measure. Direct measures of it involve a tracheal puncture, the positioning of an intraglottal transducer, or the use of an esophageal balloon, all of which are obviously invasive and thus unattractive for research purposes (Bouhuys, Proctor, & Mead, 1966; Isshiki, 1964; J. Van Den Berg, 1956). Smitheran and Hixon (1981) proposed an approach that has received wide acceptance as a non-invasive technique for estimating R_{law}. Briefly, individuals produced seven repetitions of the consonant-vowel combination /pi:/ on one breath using normal pitch, loudness, and voice quality for naturalistic speech. Syllable production occurred at a rate of 1.5 syllables per second to limit the possibility that slower rates cause intermittent velopharyngeal opening. Holmberg and colleagues (1987) further showed that execution of the speech task must adhere to two assumptions about the manner and rate of production to minimize variation for obtaining reliable results. Importantly, vocal effort needs to remain constant and the syllables connected throughout the

utterance, so that presumably, pressure measured during /p/, which is voiceless, reflects pressure that is occurring during voice segments of the utterance. Additionally, individuals need to execute the productions at a slow rate of 1.4-1.5 syllables per second to ensure reliable measurements. Although not tested, Holmberg and colleagues (1987) recommend five syllables to consistently obtain adequate pressure peaks, whereas Smitheran and Hixon (1981) suggest seven.

Regarding the calculation of R_{law}, estimated subglottal pressure (P_s) is calculated by measuring the intraoral pressure during production of the voiceless, bilabial plosive in the consonant-vowel syllable /pi/ over the seven repetitions, excluding the first and last pressure peaks (Smitheran & Hixon, 1981). This approach to measuring P_s circumvents the need of previously noted invasive and uncomfortable techniques such as tracheal puncture (Isshiki, 1964), passing a transducer through the glottis (Kitzing & Lofqvist, 1975), or by swallowing an esophageal balloon (Bouhuys et al., 1966; J. Van Den Berg, 1956). The logic is essentially that during lip closure for /p/, pressure in the oral cavity is equal to pressure in the lower airway. Three factors permit pressure equalization in the system during production of /p/: (1) the lips shutter close to seal off the oral cavity from the environment, (2) the velum raises to close off the velopharyngeal port so no air leaks out of the nasal passageways, and (3) the larynx opens so that given sufficient time, pressure within the oral cavity equilibrates with the pressure in the lower airways. Thus, measurement of peak oral pressure on /p/ provides a reasonable estimate of P_s during phonation (Kitajima & Fujita, 1990; Lofqvist, Carlborg, & Kitzing, 1982). By contrast, production of a vowel upon release of the /p/ permits the measurement of phonatory airflow. During vowel production, the vocal folds vibrate, thereby regulating airflow, and the oral cavity opens to allow a pneumotach attached to a mask enclosing the face to capture translaryngeal

airflow. Importantly, the nasal cavity continues to remain closed during vowel production with the velum in a raised position. These valving adjustments during the closed and released phases of a consonant-vowel combination create a situation allowing for the capture of near-simultaneous translaryngeal pressure and translaryngeal airflow required to calculate R_{law}.

In their validation study, Smitheran and Hixon (1981) determined that /pi/ was the most suitable speech stimulus for measuring R_{law}. That consonant-vowel combination adhered to underlying theoretical constraints that pressure equalizes during /p/ due to complete closure of the respiratory system and transglottal airflow is measurable during the vowel portion, both due to a combination of oral, velopharyngeal, and laryngeal valving adjustments. Additionally, execution of a consonant-vowel syllable string did not provide any difficulty for participants and was deemed an easy take to complete. Importantly, the use of a syllable had similarities to conversational speech and yet maintained a controlled phonetic environment that permitted the measurement of R_{law}. Relative to its component phonemes, /p/ was selected for a number of reasons. First, as already stated, transglottal pressure equalizes a few moments after closure. Next, interference would be minimal compared to typical speech production with the insertion of a pressure-sensing probe into the oral cavity, such that articulatory gestures would be fairly typical. Furthermore, insertion of only a few inches of the probe into the oral cavity is less likely to capture saliva thereby maintaining the internal validity of the measurement of P_s. Also, occlusion of the lips is easily visualized to confirm an adequate seal and the lips permit the tightest possible seal in comparison to other structures in the oral cavity that participate in production of alternative stop-plosives. Finally, the procedure has wide clinical utility for measurement of transglottal pressures in adults and even children, as it is an early emerging phoneme. As for the vowel, the velopharynx closes tightly during the production of /i/, and the

forward elevation of the tongue on /i/ in conjunction with /p/, localizes all articulatory action to the same area of the oral cavity thereby simplifying the task. Finally, consonant-vowel articulatory gestures are minimal reducing movement artifact and minimizing measurement error of translaryngeal flow. Subsequent studies argue that the use of open vowels such as /a/ or /æ/ yield similar values of R_{law} or its component parts compared to /i/. Open vowels produce average transglottal pressures assumed to equal intra-oral pressures due to negligible supraglottal pressures of /a/ or /æ/ compared to more constricted vowels such as /i/. Furthermore, an assumption is that airflow through the glottis is equal to oral airflow during open vowels (E. Holmberg, 1980; E. B. Holmberg, Hillman, & Perkell, 1988).

Findings from the measurement of male participants using the non-invasive equipment set-up and the /pi/ tasks resulted in R_{law} values that approximated previously documented R_{law} values in separate studies that employed invasive techniques (Smitheran & Hixon, 1981). In addition to establishing validity, subsequent studies investigated the reliability of R_{law} measurement using a non-invasive approach within and across days (Wilson & Leeper, 1992). Although recent data suggest that over time participants significantly improve their performance of phonation threshold pressure (PTP) (Dastalfo, 2011), the minimum amount of P_s required to initiate and sustain vocal fold oscillation, R_{law} values in comfortable and loud phonation demonstrate consistency within speakers (Wilson & Leeper, 1992), except for in extreme circumstances to be discussed shortly. When intra-speaker variation within and across session occurs, standardized scores demonstrate less than two standard deviations in variance and variability seems highly related to changes in vocal intensity, for which researchers can control in the experimental design or post hoc, statistically, during data analysis (E. B. Holmberg, Hillman, Perkell, & Gress, 1994). Therefore, researchers have established that R_{law} is a valid and

reliable measurement tool that assesses the coupled functions of the respiratory and laryngeal systems.

Researchers have asked about the effect of varying CO_2 partial pressures, vocal intensities, voice qualities, and pitches on R_{law} and its component parts. Findings have been complex and are summarized in Table 2. To date, only one study has investigated R_{law} during phonation under varying capnic conditions that induce HRD. Gillespie (2013) had female participants inspire CO_2 and produce a consonant-vowel combination at a comfortable, participant-determined pitch and loudness. Despite a statistically significant increase in minute ventilation from eupnea, minute ventilation that matches metabolic needs, to hypercapnia, Gillespie failed to find a significant difference in R_{law} – which was not constrained by any instructions or other experimental maneuvers – between eupnea and hypercapnia. Descriptively, mean R_{law} was slightly higher in hypercapnia (M = 52.3, SD = 30.7) compared to eupnea (M = 50.3, SD = 28.4). Additionally, the differences in component parts of R_{law}, estimated P_s and airflow, were not statistically significant between eupneic and hypercapnic conditions. Mean estimated P_s was slightly greater in hypercapnia (M = 7.97, SD = 1.86) than compared with eupnea (M = 7.65, SD = 2.01). Unlike P_s, mean airflow was numerically greater in eupnea (M = 0.182, SD = 0.076) compared to hypercapnia (M = 0.176, SD = 0.058). Regarding acoustic variables, neither fundamental frequency nor vocal intensity were significantly different across eupneic and hypercapnic conditions. However, many data points were missing for acoustic data. The authors identified microphone placement, ambient noise, and low vocal intensities as reasons for the missing data.

Despite the failure to detect significant differences in R_{law} or its component parts across conditions, in the noted study exhaled CO_2 varied due to a change in laryngeal function induced

by the hypercapnic condition. Mean end-tidal carbon dioxide levels during phonated segments were significantly greater in hypercapnia (M = 51.84, SD = 2.52) compared to eupnea (M = 35.38, SD = 4.10). Comparing phonated to non-phonated steady state, end-tidal carbon dioxide was significantly greater in phonated segments compared to non-phonated segments during both eupnea (non-phonated: M = 32.72, SD = 2.72) and hypercapnia (non-phonated: M = 50.16, SD = 1.50). The foregoing results suggest that the larynx during phonation has an obstructive effect on exchange of CO_2, which becomes magnified under hypercapnic conditions. Despite the limitation phonation places on gas exchange, R_{law} exhibits robustness against challenges to homeostasis, at least for a short duration and in healthy individuals.

A limitation in that study was the lack of control for vocal intensity. Previous studies show substantial changes in R_{law} during phonation as a result of varying vocal intensity (E. B. Holmberg et al., 1988; Leeper & Graves, 1984; Wilson & Leeper, 1992). In the study by Leeper and Graves (1984) on consistency of R_{law}, preliminary data from one participant, in which the experimenters held vocal intensity constant, demonstrated a reduction in the range of R_{law} values and the standard deviation compared to an uncontrolled condition. The authors concluded that vocal intensity is an important control variable in the collection of R_{law} data. A follow-up study by Wilson and Leeper (1992) with a larger sample size investigated changes in R_{law} due to changes in vocal intensity levels (25^{th}, 50^{th}, and 75^{th} percentile of the participant's vocal intensity range) in men and women. In both sexes, increases in vocal intensity increased mean R_{law}. However, the standard deviation values also increased, although the effect of SPL on R_{law} variability was greater in women compared to men. In yet another study, Holmberg and colleagues (1988) asked male and female participants to produce three vocal effort levels: soft, normal, and loud voice. Again, both males and females with no vocal training demonstrated

significantly increased R_{law} values as well as P_s and airflow values from normal to loud voice. In sum, vocal intensity influences R_{law} and its component parts by creating larger R_{law} values and greater variability at higher vocal intensities.

Beyond vocal intensity, R_{law} has been shown to change with voice quality. Grillo and Verdolini (2008) had 13 vocally trained female participants produce /pi/ in pressed, normal, resonant, and breathy voice qualities while controlling for pitch and loudness. All comparisons of R_{law} values except for those between normal and resonant voice showed statistically significant differences. R_{law} was greatest in pressed voice and lowest in breathy voice, with normal and resonant voice producing intermediate values. An earlier study by Holmberg (1980) demonstrated similar findings descriptively in four males trained as phoneticians with skill in controlling vocal pitch, loudness, and quality. As Grillo and Verdolini (2008), pressed voice produced the largest R_{law} values and breathy voice produced the smallest values. However, unlike results reported by Grillo and Verdolini (2008), the study by Holmberg (1980) offered further investigation of the interaction of pitch, loudness, and voice quality on laryngeal airway resistance. Whereas R_{law} increased as intensity increased in normal fundamental frequency and normal voice quality, with the greatest contribution coming from P_s, breathy voice quality caused R_{law} to decrease with increased intensity, primarily from an increase in airflow. Accordingly, P_s, airflow, or a combination of both variables can change with alterations in voice quality and vocal intensity at comfortable fundamental frequency.

Unlike vocal intensity and voice quality, changes in fundamental frequency demonstrate less influence on R_{law} and its component parts. In a preliminary study, Holmberg (1980) found no systematic relationship between R_{law} and three fundamental frequency levels (low, normal, high). Again, in a larger study utilizing both sexes, Holmberg and colleagues (1989) found

equivocal R_{law} results in terms of the impact of fundamental frequency. R_{law} did not differ significantly between normal and high pitch for either men or women. However, as vocal intensity was free to vary, both sexes produced significantly higher vocal intensities in high pitch compared to normal. Even after adjusting for vocal intensity, no changes in the results occurred. As for its component parts, although men demonstrated significant differences in both P_s and airflow between normal and high pitch conditions in the unadjusted means, again adjusting for vocal intensity eliminated those differences. Women demonstrated significant changes only in P_s in the unadjusted means, but again after including vocal intensity as a covariate these changes were no longer significant. The authors concluded that changes in R_{law} and its component parts correlate strongly with vocal intensity but not with fundamental frequency.

Taken together, the results of the above studies suggest that R_{law} maintains relative constancy under some conditions but fluctuates greatly under others. Most notably, the changes in the component parts of R_{law}, P_s and airflow, which represent the product of thoracic and laryngeal kinematics, illustrate how various aerodynamic events impact R_{law}. Relative to previously studied variables, vocal intensity and voice quality seem to impact R_{law} most, while fundamental frequency and short-term disturbances in CO_2 partial pressures impose less influence on R_{law} values. With respect to vocal intensity at a comfortable fundamental frequency and typical voice quality, P_s, and in terms of physiology, thoracic kinematics, appears to drive an increase in R_{law} as sound pressure level increases. The contribution of P_s and airflow to R_{law} demonstrate a more complex series of kinematic and aerodynamic events when considering voice quality. In sum, those variables that exert substantial influence, vocal intensity and voice quality, are variables that have a direct relationship with the very aerodynamic events that impact R_{law}.

Table 2. Effects of Carbon Dioxide/Hypercapnia, Vocal Intensity, Voice Quality, and Fundamental Frequency on Laryngeal Airway Resistance and Its Component Parts of Subglottal Pressure and Airflow

Relative to normal…	R_{law}	P_s	Airflow
Hypercapnia	Slightly increased	Increased	Decreased
Loud voice	Increased*	Increased*	Slightly increased
Pressed voice	Increased*	Increased	Decreased
Breathy voice	Decreased*	Decreased	Increased
High pitch	No systematic change	Increased, both sexes (*females only)	Increased, both sexes (*males only)

Note. R_{law} = laryngeal airway resistance; P_s = subglottal pressure

*denotes finding was statistically significant

1.6.3 Neural control mechanisms of phonation

Under the assumption that respiratory and phonatory functions compete, a consideration of neural control of phonation may provide insight on neural mechanisms that permit PAVUs to override automatic breathing neural control. As noted, the vocal folds interrupt the outward flow of air in a metered fashion. Stated differently, phonation involves yet another act of breathing that, unlike the continuous flow of air during quiet breathing, divides the exhalatory phase of the respiratory cycle into pulses of air in a precise and finely tuned manner. In fact, between 70-80 muscles from respiratory, laryngeal, pharyngeal, palatal, and orofacial groups come under neural integration to produce human speech (Davis, Zhang, Winkworth, & Bandler, 1996). Given the volitional nature of this type of breathing during intentional acts of communication, the neural control mechanisms of phonation have evolved to include the integration of cortical and subcortical structures in addition to the typical brainstem structures involved in metabolic or non-volitional breathing and other reflexive respiratory responses (Blitzer, Brin, & Ramig, 2009) (Blitzer et al., 2009). For example, the most complex form of vocalization, which occurs in

human speech, owes itself to intentional communication learned over time. This form comes under voluntary control and relies on higher-level structures (Jürgens, 2002).

At the lower-most, peripheral level of the nervous system, a distributed pool of motoneurons give rise to the nerves that innervate the various muscles involved in vocalization. Specific to the internal muscles of the larynx, the vagus nerve, which controls the intrinsic laryngeal muscles, arises from the nucleus ambiguus in the caudal medulla oblongata (Jürgens, 2009). From the vagus nerve, the external branch of the superior laryngeal nerve provides motor innervation to the cricothyroid and the recurrent laryngeal nerve provides motor innervation to all other intrinsic laryngeal muscles. That the branch of the vagus nerve controlling the muscles of the pharynx and intrinsic laryngeal muscles originates in the caudal medulla oblongata suggests that this nerve evolved from one involved in the control of accessory muscles for respiration to one that is also involved in vocalization (Blitzer et al., 2009).

Regarding innervation of respiratory muscles involved in phonation, both abdominal and intercostal muscles increase activation during phonation. Relative to abdominal muscle groups, internal and external oblique musculature demonstrate activation prior to the transverse and rectus abdominus. Moreover, the oblique and transverse muscles display an activation pattern that reflects the amplitude of phonation. The abdominal muscles are innervated by motoneurons that arise from the thoracic and upper lumbar spinal cord. Together with the abdominal muscles, both external and internal intercostal muscles activate during phonation, although with regard to external intercostal muscles, only the caudal portion activates during phonation and exerts an expiratory function. Intercostal and subcostal nerves innervate the thoracic muscles and arise from the cervical and thoracic portions of the spinal cord (Jürgens, 2002).

As might be expected, phonation requires the coordination of different neural structures and interconnection of a large network of motoneuron pools. In fact, different control mechanisms of the intrinsic laryngeal muscles seem to come into play even for different types of vocalization (not just different types of breathing). Interestingly, whereas the pattern of TA and PCA muscle activation in laughter as a response to verbal humor is highly reliable and relatively invariant, production of speech syllables results in a person-specific pattern of laryngeal muscle activation (Ludlow, 2011). The implication of such findings is that laughter may involve activation of primitive structures such as the peri-acquaductual gray (PAG) located in the midbrain of the brainstem, whereas speech production may require unique, individualized muscle activation patterns to accomplish learned and volitional use of laryngeal muscles. Strong suggestions exist for reciprocal interaction or a heterarchical – as opposed to hierarchical – organization of voice motor control (Jürgens, 2002, 2009). Although neuroanatomical studies fail to provide abundant evidence of direct interconnections among phonatory motoneuron pools, tracer injection studies reveal a joining together of phonatory motoneuron pools through premotor areas, which connect to all pools (Jürgens, 2002).

Within the brainstem, studies have found that an important and direct role of the reticular formation is voice motor control. A study of monkeys that provided stimulation through injection of the neurotransmitter glutamate found activation of a region of the medullary reticular formation that directly connects with phonatory motoneuron pools. Another study of monkeys that delivered electrical stimulation to the medullary reticular formation as well as other regions of the brainstem such as the lateral pons demonstrated alteration in the acoustic output of vocalization. Dysphonia even results after surgically induced lesions to the medullary reticular formation (Jürgens, 2002). Unlike the PAG to be discussed next, the reticular formation also

shows influence over fundamental frequency and amplitude of phonation (Jürgens, 2009). Thus, clearly the reticular formation provides a direct role in activation of phonatory neurons in the production of voice (Jürgens, 2002), although together reticular formation and phonatory motoneurons alone are not sufficient to produce vocalization (Jürgens, 2009).

Returning to the PAG, primitive and agonistic emotional or involuntary vocalization results from activation of this structure, whether by electrical stimulation or through injection of neurotransmitters, not just in humans but also across mammalian species (Jürgens, 2002; Schulz, Varga, Jeffires, Ludlow, & Braun, 2005). Conversely, bilateral destruction of the PAG leads to complete loss of vocalization, that is, mutism (Davis et al., 1996). Given its ubiquitous presence across species, the PAG is an essential structure for vocalization (Jürgens, 2002). Despite its clear importance in vocalization, the specific role of the PAG is not completely understood. Rather than playing a role in vocal motor coordination, the PAG may have more of a gating function as PAG activation occurs before and not during vocalization. Spontaneous firing patterns of the PAG as well as evidence from studies involving electrical stimulation of the region suggest that rather than changing the patterning of the acoustic structure of a vocalization, the PAG is responsible for the initiation and intensity of vocalizations that occur in response to painful stimuli (Jürgens, 2009). That being said, the output of the PAG does not appear to communicate directly with phonatory motoneuron pools but rather sends output to other structures in the brainstem that directly connect with them such as the reticular formation (Jürgens, 2002). Interestingly, suggestions exist that the PAG plays some sort of role as a mediator between perception and production. As opposed to some PAG neurons that fire immediately before onset of vocalization, many PAG neurons fire long before the start of vocalization and continue to fire after the animal has been exposed to other animals'

vocalizations. In contrast, PAG neurons do not fire in response to others' vocalizations when a response is not required (Jürgens, 2009).

Indeed, humans possess an ability to control vocalization that is different than lower-order mammalians. The volitional and active control of breathing for learned, propositional phonation during speech in humans involves a broader, more complex cortically-mediated network of neural pathways compared to the pathways involve in the primitive and agonistic emotional or involuntary vocalization of other mammalian species or infants, which involve activation of a PAG-mediated neural pathway (Ludlow, 2011). Two cortical areas exhibit involvement in volitional, as opposed to stimulated, control of vocal initiation. Whereas electrical stimulation of the anterior cingulate cortex elicits vocalization in both humans and non-human mammals, evidence suggests that activation of supplementary motor area (SMA) produces vocalization in humans only. Additionally, SMA is active in speech and singing, and limited speech tasks result in activation of anterior cingulate cortex. Anterior cingulate cortex does activate during silent, volitional emotional tasks such as thinking about emotional states. Furthermore, although induced lesion studies of anterior cingulate cortex and SMA are lacking in humans, injury to those areas results in transcortical motor aphasia characterized by limited spontaneous utterances and monotone speech. In socially isolated squirrel monkeys, destruction of the anterior cingulate cortex eliminates spontaneous calls but maintains calls in response to vocal calls uttered by other squirrel monkeys. In contrast, SMA lesions in squirrel and rhesus monkeys do not abolish spontaneously produced vocalizations, although the number of spontaneous productions diminishes with destruction of the pre-SMA region. Taken together, this evidence suggests that both anterior cingulate cortex and SMA play a role in initiation of vocalization, but SMA involvement is primarily for initiation of learned vocal motor patterns and

anterior cingulate cortex is mainly involved with initiation of innate vocal motor patterns (Jürgens, 2002).

As suggested above, two cortically mediated pathways seem to exist: one from the anterior cingulate cortex and one from the motor cortex, including SMA. The first neural pathway has projections from anterior cingulate cortex to PAG, which ultimately projects to the reticular formation. Given the structures involved, this limbic pathway seems to control the readiness to produce non-verbal, emotional vocalizations (Jurgens & Zwirner, 1996). For example, production of a pain cry by an actor as opposed to the reflexive pain cry response to a real harm likely involves activation of anterior cingulate cortex (Jürgens, 2009). Another pathway that controls acoustic patterning of learned, propositional vocalization can be traced from the motor cortex to the reticular formation in the lower brainstem. At the highest level of this pathway, the inferior motor cortex contains neurons that represent various muscles that control vocalization. Stimulation of that area produces bilateral vocal fold adduction. Bilateral damage to the motor cortex results in an inability to speak or sing due to total loss of voluntary fine motor vocal control. Nonverbal, emotional vocalizations representing innate vocal patterns remain intact with such lesions (Jürgens, 2009). Such evidence leads one to conclude that motor cortex involvement is mandatory for learned, propositional vocalizations but unnecessary for innate, non-propositional vocalizations. In accordance with such conclusions, pharmacological inactivation of the PAG abolishes initiation of vocal fold movement upon stimulation of the anterior cingulate cortex but does not affect initiation of vocal fold movement when the motor cortex is electrically stimulated, suggesting a direct pathway from motor cortex to reticular formation, bypassing the PAG (Jürgens, 2009). In contrast, the reticular formation is the only region capable of blocking vocal fold movement elicited by the motor cortex, as evidenced by a

lack of vocal fold response with electrical stimulation of the motor cortex following pharmacologic blockade of the reticular formation (Jürgens, 2009). Together, these findings suggest a role of the reticular nuclei as a relay from the motor cortex to the nucleus ambiguus for learned vocalizations (Blitzer et al, 2009). The intersection of learned, propositional vocalization and automatic breathing at the medullary reticular formation sets the stage for a possible competition between phonatory and respiratory functions.

1.6.3.1 Action systems: A goal-oriented theory of motor control

Until this point, the suggestion has been made that PAVUs are at increased risk for hyperphonic voice problems due to possibly competing respiratory and phonatory functions. During increased levels of physical activity, the medullary respiratory center receives information about changing blood-gas concentrations and pH levels, and responds by increasing ventilation to meet the body's metabolic needs and restore respiratory homeostasis. This acute hyperpneic respiratory response causes an increase in airway patency, which in part occurs from decreased laryngeal resistance, to accommodate large movements of air into and out of the airway. At the same time, under these HRD conditions faced by PAVUs, the phonatory system sends signals from the laryngeal motor cortex to the intrinsic laryngeal muscles, by way of the medullary reticular formation to produce learned, propositional vocalization. The response of the larynx is activation of muscles of adduction, which increases laryngeal airway resistance for the production of voice. As such, the vagus nerve, arising from the nucleus ambiguus and which controls the internal muscles of the larynx, is tasked with relaying integrated information about both respiratory and phonatory needs to the vocal folds. In other words, when PAVUs need more air the vocal folds open, but this action occurs at the same time PAVUs need to phonate, which requires approximated vocal folds. The problem with this dual role of the larynx is that PAVUs function

under possibly competing phonatory and respiratory goals. The discussion of action theory, which follows shortly, states that physiology of activity depends on functional goals. Under different initial conditions in which an action occurs, context-dependent tuning of coordinative structures such as the respiratory-laryngeal system takes place in attainment of an action goal. This motor equivalence is possible because of heterarchical, or integrated, neural control that allows for attainment of action goals under varying, even extreme, initial conditions. Therefore, action theory provides a framework for understanding the possibly competing respiratory and phonatory goals that are relevant to PAVUs.

The flexibility in and adaptation of the human respiratory system is indisputable when considering the number of functions in which it participates, for example, in the present case, gas exchange (e.g., exercise) and vocalization (e.g., communication in speech and song). Certainly, motor control of the respiratory system, including the larynx, must be complex in considering the number of degrees of freedom, which within a mechanical system are the number of independent actions that are possible for each dimension of the phase space (Pennestri, Cavacece, & Vita, 2005). One description of the chest wall suggests at least three degrees of freedom. Those actions include displacements of the rib cage, displacement of the abdominal wall, and axial displacements caused by extension of the spine and rotation of the pelvis (J. C. Smith & Mead, 1986). Degrees of freedom for the phonatory system seem equally complicated. Three degrees of freedom exist for the movement of the arytenoid cartilages, consisting of sliding in the medial-lateral direction for vocal fold adduction and abduction and rocking in the anterior-posterior direction for forward and backward motion, with an additional, but limited rotational movement (Titze, 2000;(Sellars & Sellars, 1983; von LEDEN & Moore, 1961). Additionally, the thyroid cartilage tilts about the cricoid cartilage for yet another degree of freedom for the laryngeal

framework (Titze, 2000). Moreover, in a two-mass model of voice production, Titze (I. R. Titze, 1976) identified two primary translational degrees of freedom of the vocal folds in the production of healthy phonation with a coupled 11-mode oscillator, which are medial-lateral motion and vertical motion. Clearly, more complicated is the degrees of freedom problem when one considers additional components of each of those separate systems such as individual muscles and joints.

Bernstein (1967) addressed this issue by questioning the central mechanism's ability to control individual motor units or even individual muscles involved in a complex, coordinated act due to the large number of degrees of freedom. His approach was to suggest that muscles work together synergistically in coordinative structures. Such structures have fewer degrees of freedom than the degrees of freedom involved in the summary set of individual mechanical elements of the muscle system. The synergistic activity of different muscle systems, rather than individual muscles, tendons, and joints, reflects the functioning of coordinative structures, and these coupled muscle systems execute an action that is functionally specific. In short, coordinative structures provide a solution to the degrees of freedom problem by allowing for flexibility in movement patterns in changing muscle systems while limiting the possible degrees of freedom (Bernstein, 1967). Coordinative structures operate in this somewhat constrained manner to perform an activity (Fowler, Rubin, Remez, & Turvey, 1980).

Related, Lashley (K. S. Lashley, 1930, 1933) proposed the concept of motor equivalence in which an action goal becomes realized in different circumstances through families of movements as opposed to individual movements that are invariant. Stated differently, muscular compensations occur so that a variety of movement patterns allow for the accomplishment of movement goals under different circumstances. For example, in cases of injury, individuals

demonstrate a remarkable capacity to spontaneously produce compensations or adaptive reorganization such that the end behavior is not chaotic, as one might expect with sensory or motor deficits. In a classic report (K. Lashley, 1917), Lashley described a patient with a gunshot wound of the lumbar spinal cord resulting in sensory deprivation. Despite such deprivation that clearly compromised proprioception in the left leg, the patient demonstrated an ability to lift the affected leg with remarkable accuracy. Specifically, the patient maintained an ability to control the range of leg movement, control the rate and range of movement independently, and to alternate between extension and flexion in a rhythmic manner. Lashley concluded that movement of an organ such as the leg can occur without complete sensory innervation due to neural sprouting from other action systems, proposing the control mechanism may reside either in the central nervous system or in other structures in the body (Lashley, 1917). His findings suggest that regardless of the condition of various anatomical structures, the nervous system demonstrates ability to self-regulate independent of the structural components, resulting in a degree of plasticity that permits a readjustment of behavior to normal, or near normal, capacity. To that end, structures certainly influence one another, but at the same time, the activity of certain structures may divorce themselves from others and act independently (Lashley, 1930).

Such motor equivalence (Turvey, 1977) explains how a person is able to reach and grasp a glass regardless of how far the glass is from the individual, or in the case of voice production, phonate under a variety of perturbations such as HRD. All equivalent families of movements operate within the modifiable properties of a coordinative structure involved in that action. The organization of functionally nested coordinative structures delineates the range of the equivalence family of movements. Each higher nesting level broadens the equivalent family of movements from the lower level nested within, and alterations in lower levels define higher

levels (Fowler et al., 1980). The entire system involved in an action works in an organized fashion with a goal directed by the upper levels of the central nervous system but with adjustments in lower levels of the nervous system such as at the level of the spinal cord or the peripheral nerves themselves. Through this integration of nervous system function that permits equivalent classes of movements, an organism can achieve a movement goal in which experience is lacking and learning has never taken place. This heterarchical or interactive, as opposed to hierarchical or top-down organization of the motor control system provides an explanation for how adjustments or compensations can occur to accomplish an action goal depending on the initial conditions. Lower levels use information about structures in higher levels, providing a strategy that allows lower levels to approximate with increased precision the intended action. Despite variations in movement patterns, an action is recognized as that action because of its similarities to other patterns of movement that contain similar goals (Turvey, 1977).

As Fowler and colleagues (1980) state, "As we have indicated, under conditions in which the value of the variable is fixed, the remaining variables assume values that preserve the equation of constraint (p. 401)." The implication of this statement in terms of motor control is that the effect of a perturbation to a system should be negligible in terms of goal accomplishment, allowing for the other components in the coordinative structure to attain the requisite values (p. 401). Relative to PAVUs, as a result of active compensatory adjustments, the respiratory-phonatory control system responsible for vocal intensity, for example, seems to adapt to current conditions by employing strategies that suggest vocal intensity may be invariant only when the aim is for it to be invariant. Therefore, the executive provides a control for an "equivalence class of permissible, potential organizations." In other words, the goal of the action plan may remain invariant (i.e., loud voice), but the action plan is a set of evolving processes,

and in all of the action plan's phases of change, these processes tailor themselves to the current kinematic and environmental conditions. Such a view would suggest that the central mechanism controls the interaction of lower centers that are capable, through the coordinative systems that they govern, of producing a coordinated movement pattern in a relatively autonomous fashion. Activation and tuning of coordinative structures are distinct and controlled separately. Tuning is not part of the action plan, but rather, is set based on other structures after the action plan has been established. Consequently, the central mechanism requires the storage of less information given that the tuning occurs elsewhere and lower action levels are not in need of highly detailed instructions (Turvey, 1977).

A theory of action, proposed here as a way to understand voice motor control in conditions of HRD, suggests that actions are classified in terms of function and consist of three important rules: (1) elements of action, which are groups of muscles, are a subset of the set of elements that enter into any rule for coordinated activity, (2) an executive system manages coordinated activity across muscles to the end of obtaining a given movement outcome, and (3) elements entering into action are constrained by current circumstances of this system (Turvey, 1977). Action systems' units, which do not serve a central control mechanism, consist of postures, the way in which an organism exists within its surrounding environment, and movements, in which organisms move between two postures. Postures are arranged in a stacked fashion to allow for parts of the body to change their orientation with respect to another part of the body as well as the with respect to the environment. Movements bridge postures, and general postures of the whole body or part of the body are retained during movements (Reed, 1982).

Action theory has roots in the work of Bernstein (1967) who questioned the extent of central mechanisms in motor control. He noted an ambiguous relationship between the central

command for an action and the end movement. One reason for this equivocal relationship is that the muscular contraction responding to a central command interacts with extant forces that influence the movement and, consequently, leads to a variety of movement patterns for any given initial command from the central mechanism. This equivocality also comes from the synapsing of neurons onto interneurons before they synapse with motoneurons, thus creating an indirect, rather than direct, innervation pathway. This type of nervous system organization means that multiple sources of afferent and efferent information converge at interneuronal pools to alter the state of the original central command, which presumably is about a goal, and not about specifics in terms of how to accomplish that goal. Simply stated, the central command for action represents only one input to the effector, among other types of input (Fowler et al., 1980; Turvey, 1977). Consequently, the central mechanism acts more like a community leader who organizes rather than a dictator who controls (Fowler et al., 1980). Studies have confirmed Bernstein's predictions of context-specific attunement of motor control (Reed, 1982). The speech production literature is replete with examples of immediate or nearly immediate compensations to a perturbation that interfere with normal articulation (Fowler et al., 1980). Context-conditioned variability in action cannot occur exclusively from pre-programmed open loop central processes. A change in the relationship between effector and the rest of the body causes an alteration of the initial conditions and disturbs the movement. Physical dynamics of the effector system respond to influential proprioceptive afferent information suggesting the importance of input-output loops rather than input-to-central feedback only. In other words, the integrated nervous system consists of input-output loops that reflect a complex, interactive structure. This organization of the motor system permits adaptability, which allows an organism to exhibit context-conditioned variability

in response to the changing and unanticipated environment in which it lives in order to achieve a goal, which is, if not, the central point in action theory (Reed, 1982).

In line with an integrated organization of neural control, postures and movements come under the control of perceptual systems. Data suggest, as Gibson recognized, that there could be "an output to perceptual systems and an input from motor systems (Reed, 1982)." In other words, perception and action are part of the same process. Postures involve proprio- and extero-ceptive processes. That is, the organism relates stimuli from within as well as from the external environment. Action systems learn to act through a change in perceptual variables that control movements and postures, not a change in stimulus-response associations. The integration of perception into the activities of the organism so that the behavior of the organism comes from a perceived significance of the environmental situation permits such systems to serve a plethora of functions. Borrowing from Gibson, Reed (1982) explains that agents, or the organism that performs an action, recognizes that a resource in the environment – such as an object or event - presents an opportunity upon which the agent can act. These objects or events are known as affordances. Once perceiving the utility of an object as actionable, the agent recognizes that to execute the action the agent will need to generate certain postures and movements to carry out a specific action. Thus, perceptual systems guide action systems and the component parts of postures and movements in the realization of affordances (Reed, 1982).

Accordingly, action theory builds on the term Bernstein (1967) coined, "physiology of activity," which contrasts conceptually with a physiology of reaction. Unlike the latter, "physiology of activity" is a theoretical approach that assumes an organism acts purposely, rather than reacts only, and actions in which the organism engages help the organism to equilibrate to its constantly changing environment. These actions undergo scrutiny so the organism may better

adapt, allowing it to meet the needs and goals as it navigates the environment. The perceptual systems actively evaluate and action systems provide the adaptive movement to navigate its environment. As evaluators, the perceptual systems sample ongoing stimulation, and seldom is the organism a passive receiver of stimulation. Similarly, most organisms control their actions, and rarely do actions occur without some adaptation. The "physiology of activity" accounts for variability in latencies, intensity of activity, and coordinative muscular patterns depending on the functional relevance of the situation.

Jürgens (2009) also proposes that the voice motor control system is under heterarchical rather than hierarchical control. Accordingly, the speaker has a specified goal to achieve but execution of that goal, which depends on the initial conditions, occurs through cross-talk between the different levels of the motor control system. Ultimately, the speaker draws from the unique movement patterns of a specific equivalence class to carry out the goal-oriented action and the activation patterns realized represent the synergistic interaction of various muscles so as to reduce the degrees of freedom. In the case of voice, and more specifically, voice production during physical activity but with the explicit goal of loud voice, the respiratory system, including the larynx, should demonstrate families of coupled configurations that result in a voice that achieves the target subglottal pressure but that also produces a signal that is clear enough to be understood easily. Likewise, in the same voice example, the respiratory system should attend to the metabolic needs of the body, providing an added action goal to accomplish adequate gas exchange. Therefore, the situation arises in which the individual has competing action goals. However, system constraints dictate how much each action goal can exert itself. That voice production occurs across a range of phonatory glottal configurations and physical activity requires energy production, which can be supplied through different substrates and by different

metabolic pathways, permits flexibility in the system to accomplish different action goals at the same time.

1.6.4 Vocal function in conditions of high respiratory drive

Studies investigating speech breathing behavior have observed that speech is perceived louder and exhibits higher vocal intensities, is higher in pitch, and is tremulous under HRD than under most other conditions (Otis & Clark, 1968). In addition, an informal assessment of voice quality in HRD suggested that the voice sounds breathier than normal with increased ventilatory drive (Bailey & Hoit, 2002). Several studies have investigated specific acoustic properties of voice during HRD (see Table 3) and have found that the voice reacts to rather than acts in conditions of HRD when phonation controls are not instituted (Otis & Clark, 1968). A study of 44 active duty U.S. army drill instructors (39 males, 5 females) with no known history of laryngeal surgery or laryngeal pathology, who were followed over five consecutive days while engaging in typical strenuous occupational voice use, failed to demonstrate any changes in acoustic measures of jitter and shimmer across time. However, as time progressed, a subset of drill instructors (n = 5) exhibited increasingly abnormal jitter values (short-term variability in fundamental frequency) (Mann et al., 1999).

Another study investigated vocal parameters in six female certified aerobics instructors during HRD in a commercial fitness studio (~1,000 sq. ft.). Instructors denied any previous diagnosis of vocal fold pathology. However, three instructors reported a voice problem. During the study, instructors conducted a 30-minute aerobics training session. They were specifically advised to “conduct aerobic instruction as they would in a typical session with ‘warm-up, aerobic intensity, and ending’ commands.” Instructors listened to music through headphones to

encourage a naturalistic demonstration. Pre- and post-instruction, voice and electroglottographic recordings were obtained. Audio recordings were obtained during aerobic instruction as well. Results indicated that aerobics instructors with self-reported voice problems phonated longer (*M*=52.79 percent phonation time) and with a greater vocal intensity (*M* = 40.50 dB SPL at microphone-to-mouth distance of 15 cm) than those without voice problems (*M*=30.87 percent phonation time; *M* = 34.05 SPL at microphone-to-mouth distance of 15 cm) across all instructional periods, including warm-up, intense phase, and ending commands. All instructors, regardless of vocal status, spoke with significantly higher average fundamental frequencies and greater frequency variation during all aerobic instruction periods than during reading and conversation. Importantly, electroglottography failed to reveal differences in open quotient or speed quotient between the two groups or, more importantly, between two pre-post instruction time points. The implication of these findings is that vocal patterns were similar across groups, which were differentiated only in terms of phonation time and intensity (Wolfe, Long, Youngblood, Williford, & Olson, 2002).

One final study (unpublished master's thesis) investigated the effects of concurrent aerobic exercise and speech in a group of 10 aerobics instructors (8 female, 2 male). All of the subjects had at least one year of aerobic teaching experience and taught a minimum of 2 one-hour classes per week. The subjects were tested under four speech conditions: (1) no exercise and no amplification, (2) no exercise, using amplification, (3) exercise without amplification, and (4) exercise with amplification. In addition, subjects were tested in all four conditions in two environments that included the aerobics studio (with music) and a laboratory. Acoustic measures demonstrated significant differences across the conditions. In the aerobics studio environment, instructors demonstrated significantly greater vocal intensities during sustained /a/ in the no-

exercise, no-amplification condition than in the other three conditions. In addition, vocal intensity was significantly greater in the exercise, no-amplification condition than in the exercise plus amplification condition. In the aerobics studio environment while producing a functional phrase ("keep up the energy guys"), aerobics instructors had significantly greater vocal intensities in unamplified as compared to amplified conditions. In the lab environment, vocal intensities for sustained /a/ and functional phrase were significantly lower in amplification as compared with no-amplification conditions. Exercise was not a factor in mediating statistically significant changes in vocal intensity in the lab environment. Additionally, during functional phrases in the lab, instructors produced significantly greater vocal intensities in no-amplification as compared with amplification conditions (H. M. Koblick, 2004). Stated differently, in both environments, instructors felt they did not have to shout as loudly when using amplification. Contrary to findings in the study by Otis and Clark (1968), in which vocal intensity increased with exercise, in the present study exercise inhibited vocal intensity. The lack of significantly different vocal intensity as a result of exercise condition in the lab environment could be due to a difference in room size or the lack of similar exercise intensities between the classroom and the lab. In the aerobics class environment, instructors produced significantly higher fundamental frequency in all conditions except conditions 1 (no exercise, no amplification) and condition 4 (exercise, amplification). Fundamental frequency was highest in the condition with exercise and no amplification and lowest in the condition of no exercise with amplification. Furthermore, aerobic instructors produced greater pitch variation as measured by standard deviations of fundamental frequency in conditions with exercise (H. M. Koblick, 2004).

Regarding laryngeal function in the same study, instructors exhibited a significantly lower harmonic-to-noise ratio in the no-exercise, amplification condition then with exercise and

no amplification. Although exercise did not cause a significant difference in harmonic-to-noise ratio, numerically, noise increased as physical demands increased. Furthermore, jitter values increased the exercise plus amplification condition than in the no-exercise, no-amplification condition as well as in the no-exercise, plus amplification condition. Additionally, shimmer values increased in exercise plus amplification condition compared to the no-exercise, no amplification conditions (H. M. Koblick, 2004).

Table 3. Research on Voice and Aerobic Activity

Reference	Subjects	Tasks	Time points	Measures	Results
Mann, McClean, Gurevich-Uvena, Barkmeier, McKenzie-Garner, Paffrath, & Patow, 1999	42 active-duty U.S. Army drill sergeants • 37 males, 5 females • Age: *M* = 32 yrs (range, 25-37 yrs) • 15 smokers • Excluded for known laryngeal pathology or surgery	• Sustained /a/ at comfortable pitch and loudness (acoustic) • Quiet breathing, sustained /i/ at comfortable pitch and loudness, high- and low-pitched /i/, loud /i/, soft /i/, and ascending glide on /i/ (visual-perceptual pre-post only)	• Day1: baseline (preceding start of 6-week training period) • Day 2-6: before commencing daily training of new military recruits (all visits at same time of day)	• Acoustic - jitter - shimmer • Visual-perceptual - vocal fold edge irregularity - amplitude of excursion - mucosal wave - phase symmetry - edema - erythema Inter-rater reliability: *r* = 0.76 Intra-rater reliability: *r* = 0.76, *r* = 0.77	• Acoustic - jitter: NS - shimmer: NS - *trend* of worsening perturbation values over time • Visual-perceptual - ↑VF edge irregularity: p = .0001 - ↓amplitude of excursion: p = .0106 - ↓mucosal wave: p < .009 - phase symmetry: NS - ↑edema: p = .00012 - ↑erythema: p = .0003 • Correlation b/n acoustic & V-P: NS
Wolfe, Long, Youngblood, Williford, & Olson, 2002	6 certified female aerobics instructors • 3 w/ self-reported voice problems (VP) • 3 no voice problems (NVP)	• Sustained /a/ - comfortable loudness (CL) - minimum loudness (ML) • Pitch range • Conversational speech • Oral reading	• 30-minute instruction of aerobic exercise in ~1000 sq. ft. studio - Pre-instruction - Post-instruction	• Acoustic (connected speech tasks, pitch range) - Average fundamental frequency (F_0) - F_0 SD - % talking time - Vocal intensity (25' mic-to-mouth) • Acoustic (/a/)	• Acoustic (connected speech, tasks, pitch range) - F_0: instruction > conversation or reading (p = 0.001); group: NS; task X group: NS - F_0 SD: same as F_0 - % talking time: VP > NVP (p = .0001); period of instruction: NS; group X period: VP

Table 3 (continued)

Reference	Subjects	Tasks	Time points	Measures	Results
	• Age: *M* = 32 yrs (range, 26-35 yrs) • nonsmokers	• Aerobic instruction (beginning, middle, end of session)		- Jitter - Shimmer - Harmonic-to-noise ratio (HNR) - Periodicity • Electroglottography - open quotient (OQ) - speed quotient (SQ) Measurement reliability: /a/: *r* = 0.98 (p < .001); connected speech: *r* = 0.92 (p < .001)	instruct longer at end of session (p = 0.053) - Vocal intensity: VP > NVP (p = .0001); period: NS; group X period: NS • Acoustic (/a/) - Jitter: VP > NVP (p = .027); loudness: NS; time: NS - Shimmer: ML > CL (p = .015); group: NS; time: NS - HNR: group, time, loudness: NS - Periodicity: NVP > VP (p = .049); CL > ML (p = .001); time: NS • Electroglottography - OQ: group, time, loudness: NS - SQ: CL > ML (p = .001); group: NS; time: NS
Koblick, 2004 (unpublished masters thesis)	10 aerobics instructors • 8 females, 2 males • Age: range, 18-45 yrs • 1 yr teaching experience	• Sustained /a/ • Functional phrase (“Keep up the energy guys!”) • Aerobics instruction	• (1) no exercise, no amplification, (2) no exercise, using amplification, (3) exercise without	• Acoustic - Vocal intensity - Average fundamental frequency (F_0) - F_0 SD - Noise-to-harmonic ratio (NHR)	• Acoustic - Vocal intensity: *in studio* (/a/): 1 > 2, 3, 4; 3 > 4; *in lab* (/a/): sig for each pair except 1, 3 and 2, 4; 1, 3 > 2, 4; *in studio* (phrase): sig for each pair except 2, 4; 1,

Table 3 (continued)

Reference	Subjects	Tasks	Time points	Measures	Results
	minimum • 2 one-hour classes/wk • Pulmonary function WNL • Laryngeal exam: four w/ laryngeal pathology pre-post instruction		amplification, (4) exercise with amplification - laboratory - aerobics studio	- Jitter % - Shimmer % • Subject-perceptual - Dyspnea ratings (laboratory, aerobics studio)	3 > 2, 4; *in lab* (phrase): sig for each pair except 1, 3 & 2, 4; 1, 3 > 2, 4 - F_0: *in lab* (/a/): sig for each pair except 1, 4; 3 (highest), 2 (lowest) - F_0 SD: *in lab* (/a/): sig for each pair except 1, 2 & 3, 4; 1, 3 (highest) - NHR: *in lab* (/a/): 4 > 2 - Jitter %: *in lab* (/a/): 4 > 1; 4 > 2 - Shimmer %: *in lab* (/a/): 4 > 1 • Subject-perceptual - Dyspnea: *in studio*: sig for each pair except 1, 2; 3, 4 > 1, 2; *in lab*: sig for each pair except 1, 2; 3, 4 > 1, 2

1.7 GAPS AND STATEMENT OF PURPOSE

A picture emerges that PAVUs are tasked with accomplishing phonatory goals under the backdrop of extreme respiratory perturbations. At the center of this issue are opposing laryngeal configurations required for metabolic breathing versus phonation (i.e., patent airway for breathing versus approximated vocal folds for voicing). A conceptual starting point in approaching the issue of the intersection of motor control and hard-wired physiology of respiration and phonation in PAVUs is the suggestion that during phonation under HRD, phonatory and respiratory functions may compete. Previous studies lay the groundwork for preliminary understanding of interactions between metabolic breathing and voice. Presumably, the increase in airflow and the breathy voice observed occur with relatively abducted vocal folds. The implication of previous studies – which did not constrain participants' acoustic goals – is that to handle metabolic requirements for adequate gas exchange under conditions of altered blood-gas levels during simultaneous speech and breathing in HRD, metabolic functions will be favored over voice as physiologic needs increase (Bailey & Hoit, 2002; Bunn & Mead, 1971; Doust & Patrick, 1981; Hoit et al., 2007; Otis & Clark, 1968). However, these results leave unanswered the question of whether intentional communication with specified acoustic targets would result in similar increases in airflow, again favoring metabolic need while sacrificing acoustic goals.

The relevance of this issue is that for PAVUs (e.g., law enforcement, aerobics instructors, physical education teachers, etc.), communication is *intentional* and the vocal task is *not* free to vary. Acoustic goals are relatively fixed – the individual must produce loud speech due to task/employment demands (e.g., vocal intensity of 95-103 dB SPL at 12") (Hoffman-Ruddy et

al., 2001). Loud voice – and, presumably, in the case of the bright quality and increased high-frequency energy associated with the "belt" voice in musical theater (Bourne & Garnier, 2012; DeLeo LeBorgne, Lee, Stemple, & Bush, 2010; Stone, Cleveland, Sundberg, & Prokop, 2003) – allows the acoustic signal to have discriminative power to ensure sufficient contrast from interfering signals and noise to facilitate perceptual distinctiveness for the listener. Loud voice in general and "belt" voice in particular are generally thought to be associated with *in*creased VF adduction. Consequently, exhalatory airflow in speech should be impeded and blood-gas levels simultaneously negatively impacted from decreased ventilation.

Due to their lack of control of acoustic goal, prior studies shed little light on this situation. The present series addresses the aforementioned gaps by investigating the extent to which *phonatory and respiratory* functions may be affected by systemically constraining vocal and metabolic (physical) demands of a task. The overarching goal is to determine if there is a trade-off, such that under some HRD conditions, metabolic needs will be favored over vocal demands, and under other conditions, phonatory functions will be favored over respiratory homeostasis (see Figure 2). Of particular interest for the present research are changes in phonatory function under conditions of HRD, in which millions of Americans engage on a regular basis while also speaking. The clinical and theoretical significance has to do with (1) the importance of improved understanding of mechanisms of voice disorders, which affect a huge proportion of the population across the lifespan, often with substantial implications for quality-of-life, economic consequences, and even public safety (Seth M. Cohen, Jaewhan Kim, Nelson Roy, Carl Asche, & Mark Courey, 2012; Jacobson et al., 1997; Verdolini & Ramig, 2001) Jacobson et al., 1997), and (2) the expansion of investigations on voice into a novel domain of phonatory behavior in conditions of HRD towards a better understanding of voice motor control.

The findings from the present research will guide future research in both normal and abnormal voice and cardiorespiratory function, ultimately leading to large-scale clinical studies.

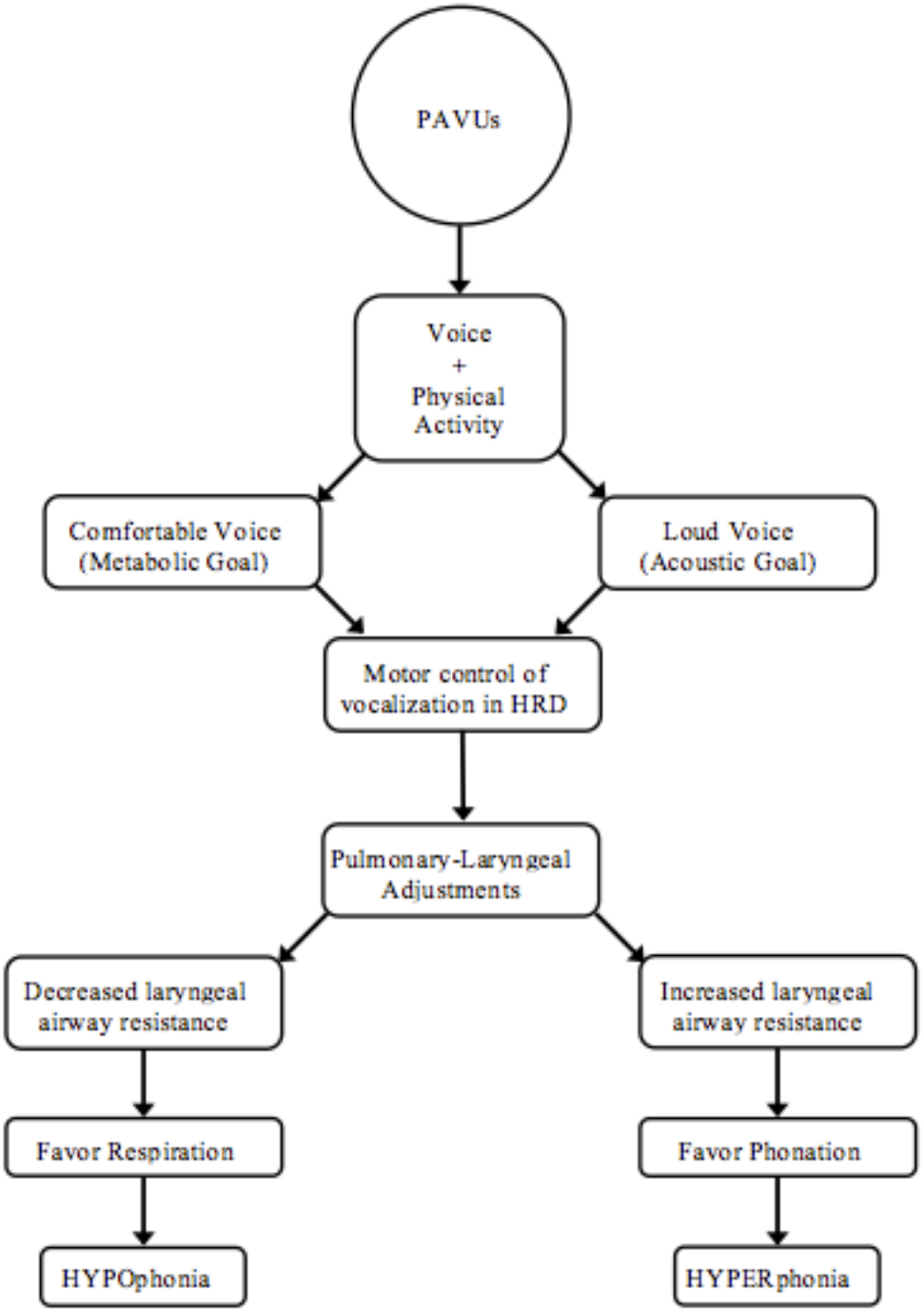

Figure 2. Conceptual schematic: Pathway delineating motor control of comfortable versus loud phonation during concurrent voice use and physical activity and the relationship with hyper- or hypophonia.

1.8 SPECIFIC AIMS AND HYPOTHESES

1.8.1 Specific aim 1 (SA1)

Investigate the influence of (a) vocal goal and (b) physical activity level on ***phonatory*** function. The primary outcome variable was laryngeal airway resistance (R_{law}), calculated as the ratio of estimated subglottal pressure to mean airflow, which together reflect coordinated respiratory-laryngeal function, during production of a 7-syllable train of /pae:/. The hypothesis for Specific Aim 1 was that adjustments in R_{law} would be linked to the speaker's acoustic goal (see Figure 3). Specifically, R_{law} would reflect "deference" to an intrinsic, biologic respiratory goal in the spontaneous voice condition, thereby showing a reduction in R_{law} as workload increased. Conversely, R_{law} would reflect "deference" to the phonatory goal in the loud voice condition, which was acoustically constrained (in difference to the spontaneous voice condition), even as workload increased.

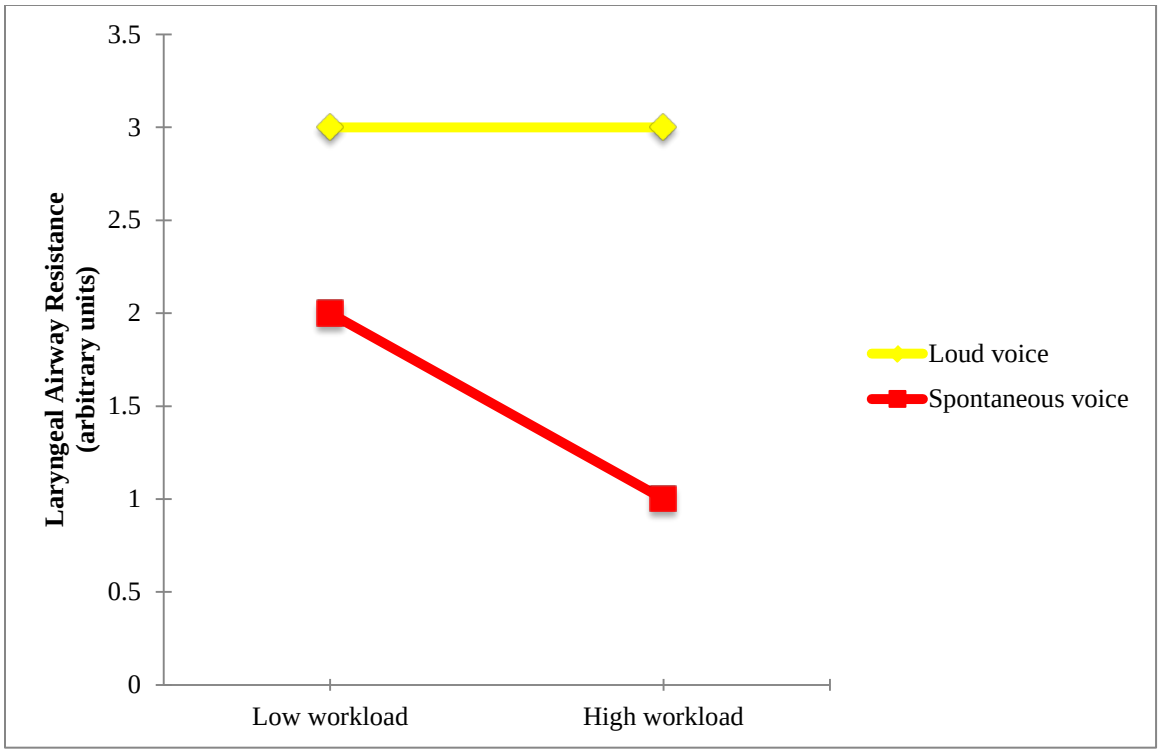

Figure 3. Predicted outcome of percentage change in laryngeal airway resistance as a function of workload and voice goal.

H_A: Loud phonation will increase laryngeal airway resistance (R_{law}) as compared to spontaneous phonation during an aerobic exercise challenge across the levels of respiratory drive. That is, loud phonation will increase R_{law} during an aerobic exercise challenge as compared with spontaneous phonation, regardless of exercise intensity. Findings in this direction would be consistent with a theory of action that states phonatory goals should alter the physiology of phonation in attainment of specified acoustic goals.

H_0: There is no difference in laryngeal airway resistance (R_{law}) between loud phonation and spontaneous phonation during an aerobic exercise challenge across the levels of respiratory

drive. Findings of this type would be inconsistent with action theory that argues phonatory goals matter even under extreme initial conditions.

1.8.2 Specific Aim 2 (SA2)

Investigate the influence of (a) vocal goal and (b) physical activity level on *respiratory* function. The primary outcome variable was minute ventilation (Ve) as an indicator of ventilatory function. Secondary outcomes were oxygen consumption, carbon dioxide production, end-tidal carbon dioxide, and respiratory exchange ratio. The hypothesis for Specific Aim 2 was that adjustments in Ve would be linked to the speaker's acoustic goal (see Figure 4). Specifically, Ve would completely reflect respiratory goals and fully favor metabolic need during silent breathing, thereby increasing as workload increased. In phonation, Ve would reflect "deference" to an intrinsic, biologic respiratory goal in the spontaneous voice condition, thereby showing an increase in Ve as workload increased. Conversely, Ve would reflect "deference" to the phonatory goal in the loud voice condition, even as workload increased.

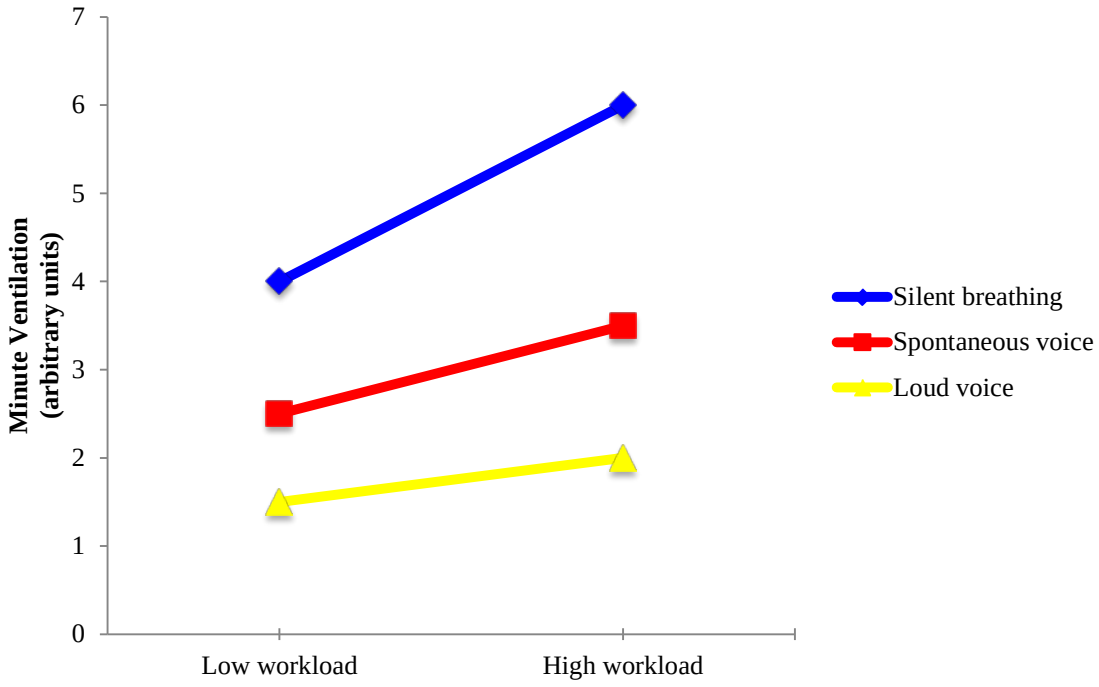

Figure 4. Predicted outcome of percentage change in minute ventilation as a function of workload and voice goal.

H_A: Loud phonation will interfere with respiratory homeostasis and blood gas concentrations as compared with spontaneous phonation or silence during an aerobic exercise challenge among the levels of respiratory drive. That is, loud phonation will decrease minute ventilation during an aerobic exercise challenge as compared with spontaneous phonation or silence, negatively affecting blood-gas concentrations. Findings in this direction would be consistent with a theory of action that states phonation with specified acoustic goals will alter the physiology of phonation in a manner that impacts respiratory goals.

H_0: There is no difference in ventilatory homeostasis and blood gas concentrations between loud phonation, spontaneous phonation, and silence during an aerobic exercise challenge among the

levels of respiratory drive. Findings of this type would be inconsistent with a theory of action that states phonatory goals should produce different physiology of movement, resulting in unique effects of those actions on respiratory goals.

2.0 RESEARCH METHOD

2.1 EXPERIMENTAL DESIGN

Both SA1 and SA2 were addressed in a single experiment. SA1 involved a 2 x 2 within-subjects design (see Table 4). Independent variables were voice condition ("spontaneous" and "loud" vocal goal) and workload (rest, 50% of age-predicted maximal heart rate [low], and 70% of age-predicted maximal heart rate [high]). The primary dependent variable was laryngeal airway resistance (R_{law}, in $cmH_2O/l/s$), which was measured through estimated subglottal pressure (P_s, in cm H_2O) indirectly captured from an intra-oral air pressure signal and mean airflow rate (U, in ml/s) during phonation. SA2 was derived from the same within-subjects experimental design used in SA1 (see Table 4). Independent variables were the same as for SA1, although the voice condition consisted of three levels: "no voice/silence," "spontaneous," and "loud" vocal goal. The primary dependent variable was minute ventilation (Ve, in L/min). Secondary dependent variables were oxygen consumption (VO_2, in mL/kg/min), carbon dioxide production (VCO_2, in mL/kg/min), end-tidal carbon dioxide ($P_{et}CO_2$, in mmHg) and respiratory exchange ratio (RER, unitless).

Table 4. Specific Aims, Dependent Variables, and Independent Variables

Aim	Description	Dependent Variables	Independent Variable 1			Independent Variable 2		
1	*Assess the influence of vocal goal and exercise workload on <u>phonatory function</u>*	P: Laryngeal airway resistance (R_{law}, in cm H_2O/l/s) S: Subglottal pressure (P_s, in cm H_2O) S: Airflow (U, in L/s)	Voice Goal			Workload (% age-predicted max heart rate)		
			Spontaneous	*Loud*		*Rest*	*50%* (Low)	*70%* (High)
2	*Assess the influence of vocal goal and exercise workload on <u>respiratory function</u>*	P: Minute ventilation (Ve, in L/min) S: End-tidal carbon dioxide ($P_{et}CO_2$, in mmHg) S: Oxygen consumption (VO_2, in mL/kg/min) S: Carbon dioxide production (VCO_2, in mL/kg/min) S: Respiratory exchange ratio (RER, unitless)	Voice Goal			Workload (% age-predicted max heart rate)		
			Silence	*Spontaneous*	*Loud*	*Rest*	*50%* (Low)	*70%* (High)

2.2 PARTICIPANTS

2.2.1 Recruitment procedures

Potential participants were recruited using IRB-approved flyers distributed in physical and electronic formats on the University of Pittsburgh's Pittsburgh campus as well as through postings of the flyer electronically on an online community bulletin board (www.craigslist.com). Given the campus' location in the city of Pittsburgh, students and non-student residents alike were able to view flyers. This strategy facilitated recruitment across age and ethnic/racial categories. Individuals who indicated an interest emailed the PI. The PI contacted the individual to provide information about the study and, if the participant was still interested, the PI conducted an online pre-screening. As part of the online pre-screening, potential participants read a brief synopsis of the study's purpose and procedures as well as time commitment and compensation for involvement in the study. By continuing to the questionnaire and submitting answers, individuals provided an initial consent to participate in the study. Screening involved a series of questions using an online survey (see Appendix) created in Google docs. Questions queried the subject about age, gender, general health, vocal status, smoking history, and involvement in physical activity. If the individual passed the online pre-screening according to the study's criteria, described below under inclusion and exclusion criteria, the PI contacted the individual by telephone to make a preliminary decision about the normalcy of the person's vocal quality through an informal auditory-perceptual screening during the conversation. Additionally,

the PI read a script to schedule the individual for an in-clinic screening (see Appendix) and sent the individual a handout specifying proper preparation for it (see Appendix).

2.2.2 Sample size

The primary participants were 32 vocally untrained and recreationally active females, ages 18-35 years. Sample size was derived from published studies on the influence of phonation on ventilatory function (Doust & Patrick, 1981; Meckel et al., 2002; Russell et al., 1998) and past research on changes in phonatory behavior across varying vocal intensities and voice use patterns (E. B. Holmberg et al., 1988; Peterson, Verdolini-Marston, Barkmeier, & Hoffman, 1994; J. Sundberg, Fahlstedt, & Morell, 2005). Using a within-participants design with an alpha of .05, and an anticipated moderate effect size, a sample size of 30 participants was required to achieve 80% statistical power for statistical analyses for **SA1** and **SA2**. However, counterbalancing of experimental conditions described shortly dictated the total participant number needed to be a multiple of four (see Table 5). Therefore, 32 participants completed the protocol. However, a small group of vocally untrained and recreationally active men, ages 18-35 years, were also recruited as a starting point to gather preliminary data for future studies on potential gender effects. Given that counterbalancing dictated multiples of four, the group of men included four individuals. Those gender effects were explored only descriptively in the current series.

Table 5. Counterbalancing Sequence of Conditions

	Voice Goal	
Workload	Spontaneous First	Loud First
Low Workload First	Low: • Spontaneous First • Loud Second High: • Spontaneous First • Loud Second	Low: • Loud First • Spontaneous Second High: • Loud First • Spontaneous Second
High Workload First	High: • Spontaneous First • Loud Second Low: • Spontaneous First • Loud Second	High: • Loud First • Spontaneous Second Low: • Loud First • Spontaneous Second

2.2.3 Inclusion criteria

Inclusion criteria, including the rationale and procedures for each criterion were as follows:

1. Women were targeted for participation because females experience phonotraumatic voice problems more than men (e.g., M. K. Miller & Verdolini, 1995; Roy et al., 2004; E. Smith, Kirchner, et al., 1998) (review in Hunter, Tanner, & Smith, 2011; M. K. Miller & Verdolini, 1995), and thus study of women had more ecological validity than the study of men. Further, sex differences have been reported relative to pulmonary function during exercise. Women are more likely than men to experience expiratory flow limitation and increased muscular work in breathing, which has the potential to negatively affect aerobic capacity and exercise tolerance (Harms, 2006; Sheel & Guenette, 2008). Therefore, to

limit sources of variability in the data, the primary sample was limited to those most likely affected by the results (i.e., 32 women).

2. The study was limited to those individuals between the ages of 18 and 35 years to minimize hormonal influences on voice. Furthermore, this age range represented a period during which time the respiratory system is stable. Participants provided their age and birthdate on the online pre-screening form and confirmed by verbal report during the in-clinic screening.
3. Participants needed to report a recreationally active lifestyle, defined in this study as regular involvement in structured aerobic exercise or sports activity of no less than 90 minutes per week and no more than 150 minutes per week (American College of Sports Medicine, 2005; Marcus et al., 2007). The rationale for requiring regular structured exercise or sports activity was to recruit participants that trained aerobically in order to target a normal cardiorespiratory fitness level and ensure that experimental requirements did not differ from the participants' typical involvement in physical activity. Extent of physical activity was determined by responding to the question "Do you participate in structured exercise and sports activity, and, if so, for how many hours do you participate in structured exercise and sports activity each week?" The participants provided an answer to these questions including the types of activities on the pre-screening form and confirmed verbally during the in-clinic screening.
4. Regular monthly menstruation as of the most recent cycle at the time of data collection (females only) or amenorrhea due to the use of medications that regulate the menstrual cycle. The rationale was that hormonal fluctuations due to the menstrual cycle are reported to deteriorate vocal performance and increase phonotraumatic responses in

approximately 33% of women (Abitbol, Abitbol, & Abitbol, 1999). Female participants reported their menstrual status on the pre-screening form and confirmed during the in-clinic screening.

5. English comprehension sufficient to provide fully informed consent and follow study instructions as judged by the PI during a telephone conversation when scheduling the in-clinic screening.
6. Ability to match pitch (+/- 1 semitone) in order to maintain a constant pitch throughout experimental tasks shown by sustaining a typical conversational pitch (men: C3 [130 Hz] and female: A3 [220 Hz] [+/- 1 semitone]) for 3 seconds when given a reference tone on an electronic keyboard, by the PI's auditory-perceptual judgment.
7. Normal voice as judged by a voice-specialized speech-language pathologist during the in-clinic screening, with scores < 10 on the overall severity subscale of the *Consensus Auditory Perceptual Evaluation of Voice* (CAPE-V) (Kempster, Gerratt, Verdolini Abbott, Barkmeier-Kraemer, & Hillman, 2009). The rationale was to ensure participants did not demonstrate a voice disorder that would potentially add variability into the data of the primary outcome measures related to voice. An informal auditory-perceptual evaluation occurred during a telephone conversation when scheduling the in-clinic screening for the purposes of eliminating individuals with obvious vocal impairment.
8. Normal hearing at 30 dB at 250, 500, 1000, 2000, 4000, and 8000 Hz in the better ear, screened by a speech-language pathologist using an audiometer at the in-clinic screening, to ensure that participants could hear instructions and monitor voice during experimental tasks.

9. Normal larynx, as judged by a fellowship-trained laryngologist during laryngeal endoscopy at the in-clinic screening, to ensure participants did not evidence any pathology that would interfere with normal voice production.
10. Normal body mass index (defined as 18.5–24.9 kg/m^2) (Panel, 1998) +/- 1.0 kg/m2 as assessed by height and weight measurements taken by a speech-language pathologist during the in-clinic screening to ensure normal fat distribution. Participants also reported their height and weight on the pre-screening form.
11. Normal resting non-medicated blood pressure (defined as non-medicated resting systolic blood pressure of <120 mm Hg and non-medicated resting diastolic blood pressure of < 80 mm Hg) (Chobanian et al., 2003) as assessed by a speech-language pathologist using a digital sphygmomanometer to ensure normal cardiovascular function.
12. Normal resting heart rate (60-100 bpm) (American College of Sports Medicine, 2005) as assessed by a speech-language pathologist using a digital heart rate monitor to ensure normal cardiovascular function.
13. Normal pulmonary function (Forced Vital Capacity and Forced Expiratory Volume in 1 second [FVC and FEV_1] values at 80% or above predicted for age, height, and gender) (Crapo, Morris, & Gardner, 1981) as assessed by a speech language pathologist with spirometry to ensure normal ventilation to support exercise-induced hyperpnea.

2.2.4 Exclusion criteria

Participants were excluded from the study if they reported any of the following:

- Lifetime history of voice problems (voice disturbance lasting for greater than 2 weeks, or dysphonia recurring more than 3 times per year) including any voice treatment, as

determined by self-report on the pre-screening form and during the in-clinic screening. In addition, a score of greater than 11 on the Voice Handicap Index-10 (Arffa, Krishna, Gartner-Schmidt, & Rosen, 2012; Rosen, Lee, Osborne, Zullo, & Murry, 2004), which was completed on the pre-screening form.

- History of vocal training with the exception of high school choir (training is defined as greater than 1 year of formal voice lessons), as reported on the pre-screening form and confirmed during the in-clinic screening.
- Any medication use during the month prior to participation in the protocol, with the exception of medications that regulate the menstrual cycle, as reported on the pre-screening form and confirmed during the in-clinic screening.
- Currently pregnant, as confirmed by positive result on a rapid pregnancy test completed at the in-clinic screening through collection of a urine sample, or were pregnant within the previous six months, as determined by self-report during the in-clinic screening.
- History of smoking in the previous 5 years, as reported on the pre-screening form and confirmed during the in-clinic screening.
- History of allergic response to any anesthesia, as reported during the in-clinic screening.
- Contraindications to exercise as outlined by the American College of Sports Medicine (ACSM) (American College of Sports Medicine, 2005) (history of cardiovascular disease [e.g., cardiac, periphery artery, or cerebrovascular disease], pulmonary disease [e.g., chronic obstructive pulmonary disease, asthma, interstitial lung disease, or cystic fibrosis], metabolic disease [e.g., diabetes mellitus (type 1 or type 2), thyroid disorders, and renal or liver disease], or orthopedic complications that would prevent optimal

participation in the exercise component [e.g., heel spurs, severe arthritis]), as reported during the pre-screening form and confirmed during the in-clinic screening.

- Signs or symptoms of active allergies or upper respiratory infection on the day of participation, as confirmed by self-report during the in-clinic screening.

2.3 MEASURES

2.3.1 Independent variables

SA1: Independent variables were voice condition ("spontaneous" and "loud" vocal goals) and workload (rest, 50% of age-predicted maximal heart rate [low], and 70% of age-predicted maximal heart rate [high]).

SA2: Same as SA1, with the addition of the silent voice condition level (i.e., silent, ("spontaneous," and "loud" vocal goals).

2.3.2 Dependent variables

SA1: The primary dependent variable was laryngeal airway resistance (R_{law}, in cm H_2O/l/s), and its component parts of subglottal pressure (P_s, in cm H_2O) and airflow (U, in L/s).

SA2: The primary dependent variable was minute ventilation (Ve, in L/min). Minute ventilation is the volume of air inhaled or exhaled per minute, which is the product of tidal volume (the volume of air inspired or expired during a single respiratory cycle) and breathing frequency (the

rate of breathing cycles [breaths per minute]). Secondary dependent variables were oxygen consumption (VO_2, in mL/kg/min), carbon dioxide production (VCO_2, in mL/kg/min), end-tidal carbon dioxide ($P_{et}CO_2$, in mmHg) and respiratory exchange ratio (RER, unitless).

2.4 EQUIPMENT

2.4.1 Screening phase

Equipment utilized to screen participants for the aforementioned criteria included a Health o meter Professional Digital Scale with Height Rod (Health o meter Professional Scales, McCook, IL), Pregnancy Test Strips (Wondfo, Willowbrook, IL), Omron HEM773AC Blood Pressure Monitor (Omron Healthcare, Inc., Vernon Hills, IL), Audiometer (Maico Diagnostics, Eden Prarie, MN), Beta WBH 54 Head-worn Microphone (Shure, Niles, IL), Computerized Speech Laboratory (KayPENTAX, Montvale, NJ, USA), Koko Spirometer (Grace Medical, Kennesaw, GA), First Act 54-Key Electronic Keyboard (First Act, Inc., Boston, MA), and Flexible Nasoendoscope (Olympus Medical, Center Valley, PA).

2.4.2 Pre-experimental phase

Equipment used in a pre-experimental phase, described shortly, included Polar T31 Transmitter Heart Rate Sensor (Polar Electro, Inc., Lake Success, NY), and Biodex Gait Trainer 2 Treadmill (Biodex Medical Systems, Inc., Shirley, NY).

2.4.3 Experimental Phase

2.4.3.1 Training Phase

Equipment utilized to train participants on the experimental task included Matrix MR500 Metronome, Kratt Master Key MK2S Chromatic Pitch Pipe (Kratt Pitch Pipes, Kenilworth, NJ), and PAS6600 (KayPENTAX, Montvale, NJ, USA).

2.4.4 Experimental Procedures Phase

Equipment utilized to run participants in the experimental phase included iHome iHM60 Rechargeable Mini Speaker (SDI Techonologies Inc., Rahway, NJ), Larson-Davis System 824 Sound Level Meter (Larson-Davis Inc, Depew, NY), Matrix MR500 Metronome, Kratt Master Key MK2S Chromatic Pitch Pipe (Kratt Pitch Pipes, Kenilworth, NJ), PAS6600 (KayPENTAX, Montvale, NJ, USA), CareFusion Encore Metabolic Cart (CareFusion Corp, Yorba Linda, CA), Polar T31 Transmitter Heart Rate Sensor (Polar Electro, Inc., Lake Success, NY), and SensorMedics 2000 Treadmill (SensorMedics Corp, Yorba Linda, CA).

2.4.5 Pre-experimental training and experimental phase equipment set-up and calibration

Instrumental setup utilized components of the Phonatory Aerodynamic System 6600 (PAS 6600), and the CareFusion Encore Metabolic Cart (see Figure 5). The PAS 6600 electronics and software were used for measurement of intra-oral pressure, mean airflow rate, and voice signal using the PAS 6600 disposable facemask, intra-oral pressure tube, pneumotach, and microphone. Details about this system are that hardware consists of a pneumotach coupled to a facemask with

an integral intra-oral pressure tube, and external microphone. Calibration was carried out according to manufacturer's instructions in the manual (KayPENTAX, 2009). The speaker phonates into the facemask with the intra-oral pressure tube placed in the mouth, on top of the tongue. Expired air flows through the pneumotach, which consists of a stainless steel mesh screen with pressure transducers on either side. The system calculates the pressure difference on either side of the screen to determine airflow rate (U) during the vowel portion of a repeated consonant-vowel sequence (e.g., /pæ:pæ:pæ:pæ:pæ:pæ:pæ:/). This system estimates P_s by calculating intra-oral pressures in the pressure tube in the mouth during production of a voiceless stop (e.g., /p/) embedded in that same sequence.

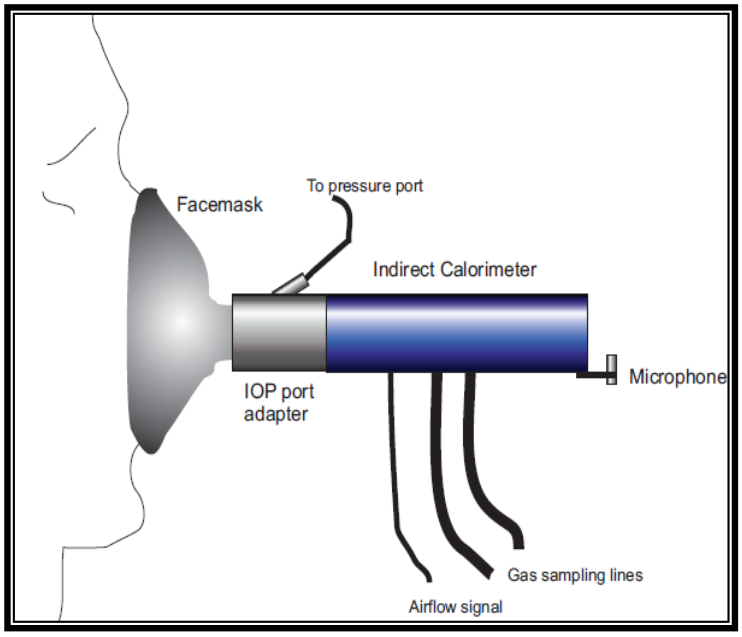

Figure 5. Schematic of equipment set-up.

The facemask and pressure tube used to collect intra-oral pressure signals were changed before every subject. The pneumotach was cleaned regularly to ensure normal calibration values. A microphone located at the terminal end of the pneumotach captured sound. The PAS6600 manufacturer internally calibrates the microphone to represent a distance of approximately 15 cm from the speaker's mouth. However, in this experimental set-up the microphone had to be repositioned from its typical site on the PAS6600 base to accommodate the serial attachment of the CareFusion Encore mass flow sensor behind the PAS6600 pneumotach. Therefore, the PI mounted the microphone onto an extender that was placed onto the PAS6600 base but that maintained the typical microphone arrangement. Accordingly, the microphone in this set up sat behind the terminal end of the CareFusion Encore mass flow sensor rather than the PAS6600 pneumotach to capture the acoustic signal. This new microphone placement required calibration to account for the altered position.

The PAS 6600 components attached to the CareFusion Encore mass flow sensor assembly (which also contains gas sampling ports). CareFusion Encore electronics and software calculated real-time breath-by-breath expired gas volumes and concentrations. Specifically, the mass flow sensor measured real-time breath-by-breath expired gas volumes including minute ventilation (Ve) and gas sampling lines estimated blood-gas concentrations including oxygen consumption (VO_2), carbon dioxide production (VCO_2), end-tidal carbon dioxide ($P_{et}CO_2$), and respiratory exchange ratio (RER). The researcher fixed the mass flow sensor to the end of the pneumotach with a plastic connector. Strong adhesive ensured that the mass flow sensor would not separate from the pneumotach and that the seal between the mass flow sensor and the pneumotach was airtight. In addition, the researcher attached a vertical support piece to the base of the PAS6600 perpendicular to the mass flow sensor to support the weight of the mass flow

sensor and maintain it on a level plane. A new mass flow sensor was placed before every subject. Gas-sensor tubing was replaced as needed to avoid contamination from the build-up of condensation of exhaled air.

The PI carried out calibration of the PAS 6600 according to the device's instruction manual provided by the manufacturer (KayPENTAX, 2009). The PAS 6600 system automatically calibrates the intra-oral pressure tube upon experimenter's selection of *Calibrate Air Pressure Zero Level* from the system's Options menu. Calibration of air pressure was completed before data collection for each experimental condition. To calibrate the pneumotach, a 1.0L syringe was coupled to the airflow head. The syringe plunger was depressed in one continuous motion for 2-4 seconds, emptying the syringe air into the pneumotach. The system provided a calibration value (proper calibration value: 1.0L +/- 1-2%). Calibration of the pneumotach occurred daily and was repeated as prompted by the PAS system (i.e., every four hours following initial daily calibration). Calibration of the microphone occurred before each participant's testing session. The PAS facemask was attached with a neoprene facemask to a styrofoam display head that had a speaker housed within a hole in the display head at the site of the mouth. A 200 Hz pure tone was played for 30 seconds through the speaker and recorded by the PAS microphone. At the same level of the microphone, a sound level meter set to the slow setting and C weighting obtained the tone's actual sound pressure level (SPL) in dB to provide a correction factor for the intensity recorded by the PAS. The correction factor was therefore the pure tone SPL as detected by the sound level meter minus the PAS6600 intensity of the pure tone. The participant's actual SPL was then calculated as the PAS6600 vocal intensity plus the correction factor.

Calibration of the CareFusion Encore Metabolic Cart was carried out prior to each session according to manufacturer specifications. The CareFusion Encore mass flow sensor was calibrated by coupling a 3.0L syringe to it and depressing its plunger in one motion for 2-4 seconds (proper calibration value: 3.0L +/- 1-2%). Gas concentrations were calibrated using two-span gases prior to each study: 1) 26% oxygen, 0% carbon dioxide, and the balance in nitrogen; 2) 16% oxygen, 0% carbon dioxide, and the balance in nitrogen.

2.5 PROCEDURES

2.5.1 Screening procedures

Upon scheduling the in-clinic screening, participants were instructed to wear comfortable, loose fitting clothing and athletic shoes consistent with testing, drink plenty of fluids over the 24-hour period preceding the test, eat a light meal no less than 2 hours before arrival, avoid alcohol and caffeine for at least 3 hours before testing, avoid exercise or strenuous physical activity for 48 hours before the test, and get an adequate amount of sleep, preferably 6 to 8 hours, the night before testing. In addition, the PI emailed participants a handout with these instructions.

The in-clinic screening took place in a quiet, private room at the University of Pittsburgh Voice Center in Pittsburgh, PA. On the day of the screening, first the PI or research assistant presented to prospective participants, both orally and in written form, the potential risks and benefits of the research, ensuring that the individual fully understood their involvement in the research. Upon reviewing, participants had an opportunity to ask questions. After questions, participants provided their informed consent to participate in the study. Upon gaining consent,

satisfaction of self-report and clinician judged inclusion/exclusion was confirmed, as indicated under "Participants." Next, data about participants' body mass as determined from measurements of height and weight was gathered using stadiometry and a digital scale, respectively, to determine normal body mass index +/- 1 kg/m^2 (normal is 18.5–24.9 kg/m^2) (American College of Sports Medicine, 2005). Then, female participants provided a urine sample and submerged a pregnancy test strip to provide confirmation of a negative pregnancy status. Next, vitals including resting non-medicated heart rate and blood pressure were collected for each participant. A digital sphygmomanometer was used to measure heart rate and blood pressure and compared to normative data (normal is defined as non-medicated resting systolic blood pressure of <120 mm Hg and non-medicated resting diastolic blood pressure of < 80 mm Hg) (Chobanian et al., 2003). Normal resting heart rate was considered less than 80 beats per minute (American College of Sports Medicine, 2005). The inflatable cuff attached to a digital sphygmomanometer was placed smoothly and snugly around the upper arm, at roughly the same vertical height as the heart while participants were seated with the arm supported on an armrest. The cuff was automatically inflated and air was released slowly and steadily.

Next, a hearing screening was performed using pure-tone audiometry at 30 dB at 250, 500, 1000, 2000, 4000, and 8000 Hz. Participants were seated comfortably in a quiet environment facing away from the audiometer with over-the-ear headphones placed on the head. Participants indicated that a tone was heard by raising the hand of the same side to which the tone was delivered. Normal hearing was defined as response in the better ear to each of the tested frequencies. Failure to achieve the criteria resulted in the exclusion of participants and individuals with abnormal findings were referred for appropriate clinical evaluation. Following the hearing screening, a voice-specialized speech-language pathologist conducted a clinical voice

evaluation to determine degree of dysphonia. Individuals were seated comfortably in a quiet environment. A condenser microphone was placed at an angle of 45 degrees from the midline of the lips at a 10-cm microphone-to-mouth distance for recording. Participants were asked to read six specific sentences with different phonetic contexts using the *Consensus Auditory-Perceptual Evaluation of Voice* (CAPE-V) (Kempster et al., 2009). The voice-specialized speech-language pathologist made a subjective determination about voice normalcy. A voice was judged as normal with scores < 10 mm on a 100 mm visual analog scale on the overall severity subscale of the CAPE-V.

When the clinical voice evaluation had been completed, participants heard a reference tone (men: C3 and female: A3 [+/- 1 semitone]) on an electronic keyboard and matched the presented typical conversational pitch, as judged by the same specialized SLP, over a period of three seconds. This inclusion criterion was related to the goal of having participants match pitches (roughly) and maintain them during voice production tasks in the experimental procedures.

Then, pulmonary functioning was determined with spirometric testing, per American Thoracic Society (ATS) standards (M. R. Miller et al., 2005). Specifically, all participants assumed a seated position, had a nose-clip attached, placed the mouthpiece of a spirometer in the mouth and closed lips tightly around the mouthpiece. Participants performed a sequence of four tidal breaths into the mouthpiece. Then, participants inhaled completely and rapidly with a pause of < 1 second at total lung capacity. Next, participants exhaled maximally until they failed to expel further air (duration ≥ 6 seconds and a plateau in the volume-time curve) while maintaining an upright posture. These procedures were repeated for a minimum of 3 maneuvers. The test data were checked for repeatability (no extrapolated volume < 5% of Forced Vital

Capacity or 0.150 L, whichever is greater). Results judged as “normal” were free from artifacts such as cough, glottic closure, air leakage, obstruction of mouthpiece, early termination, and perceived sub-maximal effort. In addition, each maneuver had to be completed with only one inhalation. Results confirmed expiration for greater than 6 seconds. All participants had Forced Vital Capacity (FVC) and Forced Expiratory Volume in the first second (FEV_1) within normal limits as predicted by age, height, and gender (greater than 80% of predicted value [Crapo et al., 1981]). Participants had no fewer than three and no more than eight chances to achieve those criteria. Failure to achieve the criteria within those parameters resulted in the exclusion of participants.

Finally, a fellowship-trained laryngologist performed a laryngeal examination using a flexible endoscope. For this procedure, participants were positioned upright, and the nasal passage demonstrating the greatest patency was sprayed with a local anesthetic (i.e., Cetacaine®). When sufficient numbing had been achieved based on participant report and clinician evaluation, a scope was passed through the nose into the laryngopharynx, until the larynx was fully visualized. The initial portion of the examination involved halogen illumination of the larynx at rest during breathing. The larynx was then successively visualized under both halogen and stroboscopic illumination during sustained /i/ at comfortable (spontaneous/modal) and high pitches (about one octave higher than spontaneous comfortable pitch) and quiet and comfortable loudness at each pitch. A fellowship-trained laryngologist made a subjective determination about laryngeal normalcy based on video recordings of the procedure. A normal laryngeal appearance was defined as no visible lesions, normal arytenoid movement on ab/adduction, normal vocal fold shortening and lengthening with pitch changes, and complete closure of the membranous vocal folds during phonation at modal pitch. Individuals with

abnormal findings were excluded from further participation and referred for appropriate clinical evaluation. Individuals satisfying all of the inclusion and exclusion criteria were considered enrolled in the protocol.

2.5.2 Pre-experimental procedures

On the same day as the screening and after eligibility criteria had been satisfied, participants engaged in pre-experimental set-up procedures in another room that contained a treadmill at the University of Pittsburgh Voice Center. The purpose of these procedures was to determine workloads, the speed and grade required for achieving the precise intensity needed to reach age-predicted heart rates for each of the systematically manipulated experimental physical activity levels, described shortly, with the exception of the rest condition, for subsequent experimental sessions. Those procedures required participants to perform exercise on a Biodex Gait Trainer 2 motorized treadmill (Biodex Medical Systems, Inc., Shirley, NY) using a modified ramping protocol that was developed by Jakicic and colleagues for a multi-center Look AHEAD Study (see Table 6) (Ryan et al., 2003). The use of the ramping protocol was preferred over other graded exercise protocols because it initiates at a lower exercise capacity and increases workload in equal and small increments, which allowed for finer control of the target heart rate requirement for the two exercise intensities on the experimental day. For the purposes of this experiment, percent intensity to be maintained during aerobic activity was of two levels: 50% and 70% of age-predicted maximal heart rate (a moderate [low] and moderately vigorous [high] exercise intensity level that is prescribed for recreationally active individuals by personal trainers for improving cardiovascular fitness ([American College of Sports Medicine, 2005]). Furthermore, the low exercise intensity represented a workload during which participants were

expected to be able to speak without difficulty and the high exercise intensity represented a workload during which participants might have difficulty speaking (Persinger, Foster, Gibson, Fater, & Porcari, 2004; Quinn & Coons, 2011). Stated differently, the low condition represented a level at which participants should have had the ability to override metabolic breathing needs with relative ease. The high condition was associated with greater respiratory drive than the low condition as it was selected to induce a larger ventilatory response in participants. Therefore, there would be significant competition between metabolic needs and phonatory demands in the high condition. Under both conditions, participants should have had the ability to achieve the acoustic goal of a loud voice given external motivation but the low condition should have provided greater flexibility in the respiratory control system than the high condition.

ACSM criteria were used to exclude individuals for whom exercise was contraindicated (American College of Sports Medicine, 2005) and, therefore, participants should have had been able to complete the exercise protocol without incident. The use of a treadmill was preferred over a cycle ergometer because of the potential to minimize early leg fatigue. Participants were advised about the potential discomfort associated with the procedure and about the type of information that would be obtained. Participants received a detailed explanation of the exercise protocol, and informed that exercise could be halted at any time. They were instructed to maintain a steady walking pace during exercise and to avoid turning head or trunk, which could compromise balance. Finally, participants were invited to pose any questions before the protocol commenced. The exercise protocol itself involved a systematic increase in treadmill grade and speed following every minute of testing illustrated in Table 6, until participants achieved his or her age-predicted maximal heart rate at the high intensity exercise condition as determined by the Karvonen Formula (target heart rate = [max heart rate, i.e., 220 – age] – resting heart rate * 0.70]

+ resting heart rate). In addition, the ACSM (American College of Sports Medicine, 2009) criteria for exercise termination was followed. Specifically, the exercise protocol would be discontinued if there was an onset of angina or angina-like symptoms, if there were signs of poor perfusion such as light-headedness, confusion, ataxia, pallor, cyanosis, nausea, or cold and clammy skin, physical or verbal manifestations of severe fatigue, or failure of the testing equipment. Participants were monitored during the exercise protocol to ensure safety. Before every change in workload, participants provided indication by gesturing their desire to continue with or terminate the protocol. Heart rate was also continuously monitored throughout the protocol with a heart rate sensor. Upon completion of the exercise protocol, participants were discharged with reminders to maintain normal fluid and dietary intake for the entire period of their participation. To allow for adequate metabolic recovery, initial experimental procedures were scheduled no earlier than 24 hours after the pre-experimental exercise protocol.

Table 6. Submaximal Treadmill Test Protocol

Time (minutes)	Speed (mph)	Grade (%)
0:01 – 1:00	2.0	0.0
1:01 – 2:00	2.5	0.0
2:01 – 3:00	3.0	0.0
3:01 – 4:00	3.0	1.0
4:01 – 5:00	3.0	2.0
5:01 – 6:00	3.0	3.0
6:01 – 7:00	3.0	4.0
7:01 – 8:00	3.0	5.0
8:01 – 9:00	3.0	6.0
9:01 – 10:00	3.0	7.0
10:01 – 11:00	3.0	8.0
11:01 – 12:00	3.0	9.0
12:01 – 13:00	3.0	10.0
13:01 – 14:00	3.0	11.0
14:01 – 15:00	3.0	12.0
15:01 – 16:00	3.0	13.0
16:01 – 17:00	3.0	14.0
17:01 – 18:00	3.0	15.0
18:01 – 19:00	3.2	15.0
19:01 – 20:00	3.4	15.0
20:01 – 21:00	3.6	15.0
21:01 – 22:00	3.8	15.0
22:01 – 23:00	4.0	15.0

2.5.3 Training in laryngeal airway resistance experimental task

R_{law} training occurred on the first experimental visit that followed the visit consisting of the in-clinic screening and pre-experimental procedures in a quiet, private room at the University of Pittsburgh Physical Activity and Weight Management Research Center in Pittsburgh, PA. The first experimental visit occurred no sooner than 24 hours after the in-clinic screening that

included the pre-experimental procedures. Before commencing, participants' spontaneous vocal intensity was determined using well-vetted published procedures (Russell et al., 1998) due to the fact that changes in vocal intensity may influence R_{law} (E. B. Holmberg et al., 1988; J. Sundberg et al., 2005; Verdolini et al., 1998). Notably, the same set-up for experimental procedures proper, consisting of the joined pneumotach and mass flow sensor, was used for collection of spontaneous vocal intensity. The only difference between the two procedures was that, during collection of vocal intensity data, the PI removed the PAS6600 pressure tube, as it was not needed, and placed a cap onto the pressure tube port to close the system and ensure no leakage of air during collection of vocal intensity data. This consistency in set-up allowed the target vocal intensity for the vocal goal in the loud condition to simply be the average spontaneous vocal intensity plus 10 dB (+/-2 dB). The noted SPL level targeted for the "loud" condition was selected to ensure an adequate change of ventilatory stress on the system during speech compared to the spontaneous condition (Russell et al., 1998), and also to provide a distinctly different vocal intensity compared to the "spontaneous" condition (Hoffman-Ruddy et al., 2001). The respective SPL levels were used for "spontaneous" and "loud" conditions in subsequent experimental procedures. Regarding the procedures of the collection of vocal intensity data, standing on the treadmill, participants held the PAS6600 unit by the dual handles with the anesthesia mask firmly placed against the face, surrounding the nose and mouth. A neoprene facemask containing two Velcro straps surrounded the PAS6600 mask and secured it in place to avoid any change in mask position. To determine spontaneous vocal intensity, participants were instructed to read a standard passage, the *Rainbow Passage* (Fairbanks, 1960), for 5 minutes continuously, having been instructed to "speak normally." The reading was recorded and immediately analyzed with the PAS6600 software. The middle three minutes of the passage were

analyzed for vocal intensity. After removing the PAS6600, participants counted aloud from one to five, and held out the /i/ in three to gather a comfortable speaking pitch (Peterson et al., 1994). A pitch pipe confirmed the closest pitch produced during production of /i/. The vocal intensity (SPL) and fundamental frequency (F_0) identified through these procedures were used during the experimental procedures proper to eliminate different SPL and F_0 levels as possible confounding variables.

After determining target SPL and F_0 for experimental procedures, participants learned to properly complete R_{law} data collection procedures prior to the experiment proper. This training first occurred without the PAS6600 in place. R_{law} training required participants to produce a string of seven repeated /pæ:/ syllables at a rate of 1.5 syllables per second in both spontaneous and loud voice until he or she produced them consistently at his or her individually determined spontaneous speaking F_0 (+/- 1 semitone) determined by the counting task during the training procedures. The methods used in the R_{law} task have been well established for their validity and reliability in the phonatory aerodynamic literature (L. B. Helou & N. P. Solomon, 2011; Smitheran & Hixon, 1981). A pitch pipe provided the reference pitch and a Matrix MR500 Metronome provided the rate at which the /pæ:/ syllables were to be repeated by producing a recurring clicking sound at 88 beats per minute (i.e., ~1.5 syllables per second). The PI coached participants in those two aspects of the experimental task until confirming perceptually that participants demonstrated an ability to adequately accomplish them in a spontaneous vocal loudness. Specifically, the PI instructed participants to raise or lower the pitch until it matched the reference tone and speed up or slow down the syllable rate until it equaled that provided by the metronome. With minimal coaching, all participants demonstrated an ability to repeat the syllables at the appropriate rate.

Despite participants' ability to match pitch with relative ease during the screening, some exhibited difficulty with pitch matching during training. The PI provided those participants with an immediate model to assist with matching pitch. When participants continued to demonstrate trouble, the PI and participant produced the pitch in chorus until participants achieved the target as determined perceptually by the PI. Next, participants demonstrated the experimental task in a loud voice. Again, the PI coached participants in accomplishing a loud voice by instructing them to increase or decrease loudness until it was perceptually different from spontaneous. Participants demonstrated a loud voice with minimal coaching. However, as during the spontaneous vocal loudness condition, some participants required more than minimal coaching to achieve the target pitch in loud voice. Research shows that pitch often raises with increased loudness in untrained individuals (Titze & Sundberg, 1992).

After learning the experimental task, participants practiced it while connected to the instrumentation to become comfortable with its presence. At this point, the PAS6600 facemask was placed over nose and mouth, and the PI secured it into place around the participant's head with a neoprene facemask to ensure a good seal between participants and the PAS6600 mask so as to not compromise the validity of air pressure and airflow data. Participants were asked to indicate whether the mask was comfortable, and adjustments were made until an affirmative answer was achieved while at the same time, a firm seal could be verified and placement was adequate for data collection purposes. That is, care was taken by the PI to assure participants fit the facemask snugly over nose and mouth during task production, and that the pressure tube sat lightly on top of the tongue. Mask and pressure tube placement was monitored constantly throughout the study to ensure there was a tight mask-to-face seal and appropriate pressure tube positioning so as to not compromise the validity of data collection. Upon ensuring an adequate

seal had been achieved with pressure tube in the mouth, participants practiced the experimental task at both loudness levels at the pre-determined F_0. Training ceased after both PI and participant expressed confidence in the correct execution of the phonatory R_{law} task.

2.5.4 Experimental procedures proper

2.5.4.1 Research facility

Experimental sessions occurred in a quiet, private room at the University of Pittsburgh Physical Activity and Weight Management Research Center in Pittsburgh, PA. The environmental conditions (i.e., relative humidity and temperature) of the research facility were kept stable and in accordance with recommended standards for fitness facilities (Medicine, 2007).

2.5.4.2 Experimental procedures proper

As noted, participants had been asked to maintain normal fluid and dietary intake throughout the period of their participation of the study. Additionally, experimental sessions were scheduled at roughly the same time of day to control for voice use patterns and hormonal influences on body hydration and voice function. Participants were also instructed to wear similar clothing for pre-experimental and experimental session to minimize the influence of weight differences on exercise performance and measures.

Upon arrival to the first experimental session, with one exception described shortly, the researcher assigned each participant to a randomized, counterbalanced sequence of workload and vocal goal conditions (see Table 5). Online software (www.random.org)(Haahr, 1998) generated the randomized list of counterbalanced conditions. Because the PI withdrew one participant from the study due to an unanticipated adverse event during testing, described shortly, an additional

participant was recruited and assumed the sequence of conditions for the participant who was replaced. The adverse event included a vasovagal response unrelated to the experimental conditions. After the event, the participant reported a history of anxiety disorder requiring medication. However, at the time of the study, she did not take medications and had not taken medication for a period of years. She fully recovered from the adverse event and walked out of the testing facility without any assistance.

An overview of experimental procedures is shown in Table 7. As for the experimental procedures proper, first, participants stood on a SensorMedics 2000 Treadmill (SensorMedics Corp, Yorba Linda, CA) wearing the Polar T31 Transmitter Heart Rate Sensor already connected to the PAS6600, with eyes open, and breathing quietly so respiratory baseline data could be collected. Baseline Ve, VO_2, VCO_2, $P_{et}CO_2$, and RER data were collected using the CareFusion Encore data acquisition system. These data were collected and analyzed on a breath-by-breath basis for 5 minutes of tidal breathing. The last 30 seconds of the initial quiet breathing period were used as baseline respiratory data for the rest condition. Next, facemask still in place, participants produced the syllable sequence at rest while standing, using the pre-determined pitch and "spontaneous" loudness or the pre-determined pitch and loud voice (+10 dB above spontaneous, +/-2 dB) counterbalanced across participants (see Table 5). The same ordering of "spontaneous" versus "loud" speech was used, within participants, for phonatory R_{law} tasks across the entire experimental protocol.

Table 7. Template for Experimental Procedures. Note Order of Vocal Goal (Spontaneous and Loud) and Workload (50 and 70% of Age-Predicted Maximal Heart Rate) were Counterbalanced across Participants

Condition	Activity	Time
First Experimental Session (Visit 2)		
Rest	Breathe quietly with no phonation (rest respiratory baseline)	5 min
	Produce consonant-vowel syllable string (e.g., /pæ:pæ:pæ:pæ:pæ:pæ:pæ/) (spontaneous voice, which served as phonatory baseline)	30 sec
	Breathe quietly with no phonation to return to rest baseline	1 min
	Produce consonant-vowel syllable string (loud voice)	30 sec
Rest-to-work Transition	Walk on treadmill to achieve low exercise intensity	~3 min
50% of age-predicted maximal heart rate (low)	Breathe quietly with no phonation at low exercise intensity (50% baseline)	1 min
	Produce consonant-vowel syllable string (spontaneous voice)	30 sec
	Breathe quietly with no phonation to return to 50% baseline	1 min
	Produce consonant-vowel syllable string (loud voice)	30 sec
Recovery Period	Walk on treadmill until resting heart rate is achieved	~3 min
Second Experimental Session (Visit 3)		
Rest	Breathe quietly with no phonation (rest respiratory baseline)	5 min
	Produce consonant-vowel syllable string (spontaneous voice)	30 sec
	Breathe quietly with no phonation to return to rest baseline	1 min
	Produce consonant-vowel syllable string (loud voice)	30 sec
Rest-to-work Transition	Run on treadmill to achieve high exercise intensity	~3 min
70% of age-predicted maximal heart rate (high)	Breathe quietly with no phonation at high workload (70% baseline)	1 min
	Produce consonant-vowel syllable string (spontaneous voice)	30 sec
	Breathe quietly with no phonation to return to 70% baseline	1 min
	Produce consonant-vowel syllable string (loud voice)	30 sec
Recovery Period	Walk on treadmill until resting heart rate is achieved	~3 min

The instruction for the non-goal-oriented spontaneous phonation condition was, “Speak three sets of seven syllables of /pæ:/ with your typical voice until instructed to stop at the following pitch and speaking rate.” The instruction for loud phonation condition was, “Speak three sets of seven syllables of /pæ:/ continuously, with a loud and clear voice until told to stop at the following pitch and speaking rate." Vocal clarity was included in the instruction as voice quality interacts with loudness (Berry et al., 2001), and clarity is additionally a valid ecological goal for most individuals who engage in phonatory HRD, for intelligibility purposes. The PI presented the reference tone using a pitch pipe and an immediate vocal model of the pitch in the appropriate octave. Some participants demonstrated difficulty with matching pitch during the task and the PI provided a verbal prompt and additional vocal models to guide participants to the correct pitch. The PI provided an up and down hand motion in time with a blinking light from a metronome that was in participants’ view to provide participants with the appropriate speaking rate. The PI only mentioned vocal clarity in the instructions for the loud voice condition but did not intervene during the task. During the spontaneous voice condition, participants produced a string of seven consonant-vowel syllables (e.g., /pæ:pæ:pæ:pæ:pæ:pæ:pæ/) on a single breath, at a rate of 1.5 syllables/second, at the previously established F_0 (+/- 1 semitone). Participants did not target a vocal intensity goal during the spontaneous voice condition. During the loud voice goal condition, the PI monitored vocal intensity by visually inspecting the acoustic signal on the PAS6600 output screen. Two reference lines that denoted the acceptable vocal intensity range were present on the screen to facilitate visual inspection. The PI coached participants as needed to achieve and maintain the target loud vocal intensity within that specified range. Participants repeated the /pæ:/ string a total of three times, although some participants continued to complete more sets if visual inspection determined vocal intensity had not been achieved. Although only

individuals who had the ability to match pitch (+/- 1 semitone) were included in the study, some of the participants' productions did not satisfy F_0 criteria during the experimental procedures for the /pæ:/ task. Samples were not repeated as data from previous studies indicated that F_0 had little influence on R_{law} (E. Holmberg, 1980; E. B. Holmberg et al., 1989). Data for primary and secondary ventilatory measures, including Ve, VO_2, VCO_2, $P_{et}CO_2$, and RER, as well as data for voice measures, R_{law}, P_s, U, F_0, and SPL, were captured during the 7-syllable consonant-vowel syllable task.

Following completion of the first set of phonatory R_{law} tasks, there was a 1-minute rest period to allow the participant's respiratory system to return to that workload's baseline steady state. Given the short duration of the speaking task, this time period was considered sufficient for participants' respiratory system to return to baseline from the speaking-related perturbation. Throughout the protocol, steady state was defined as the point at which oxygen consumption reached baseline values. Rest time was extended until steady state was achieved.

Subsequently, participants completed a rest-to-work transition on the treadmill after completion of the second speaking task. Following rest and during the transition period, the PI set the appropriate workload on the treadmill ergometer per previously established participant-specific speed and grade adjustments to achieve a stable exercise workload of either 50% or 70% of age-predicted maximal heart rate. In other words, the participant went from rest to an exercise workload of 50% or 70% age-predicted maximal heart rate. To ensure that participants did not overshoot the target heart rate, the researcher set the initial workload level on the treadmill ergometer at half of the intended workload. Then, the researcher ramped up the workload in 1-minute increments until participants reached the previously determined target heart rate. At that point, participants maintained that workload for roughly two minutes. This time period was

chosen as the time anticipated to achieve steady state based on the exercise physiology literature that shows 2-3 minutes is sufficient time for steady state to be reached from a change in workload during exercise in healthy individuals (McArdle et al., 2010). New steady state baselines were established for each of the levels of exercise workload (rest, 50% workload, and 70% workload conditions). As noted, order of exercise intensities was counterbalanced across subjects. New respiratory baselines (i.e., steady state) were established for each level of exercise workload (rest, 50% workload, and 70% workload conditions) based on VO_2 values and, once steady state was achieved, defined as no more than 1 MET change in VO_2 from breath to breath, the same speech tasks were repeated as previously, using the same intensities, after workload steady state had been achieved. Again, data for primary and secondary respiratory measures, including Ve, VO_2, VCO_2, $P_{et}CO_2$, and RER, as well as data for voice measures, R_{law}, P_s, and U, were captured during the 7-syllable consonant-vowel syllable task. Finally, participants completed a recovery period on the treadmill that consisted of walking until heart rate responses resolved to ensure safe return of physiologic processes to baseline levels. Total duration for the experimental tasks during this session, including the training in the phonatory R_{law} task, was approximately 45 minutes.

These same procedures were repeated during the second experimental session (Visit 3), which followed the first session by a minimum of 24 hr to allow for dissipation of short-term metabolic effects. One male participant came to the second experimental visit 20 hours after his first experimental visit due to scheduling conflicts. Before initiating the experimental protocol, participants reviewed R_{law} procedures to ensure consistency in execution of the task. The exception was that participants engaged in the exercise intensity condition they had not yet encountered (50% or 70% workload). Within participants, the same order of spontaneous versus

loud phonation conditions was used as for the first experimental session. The second experimental session lasted approximately 30 minutes.

2.5.5 Data storage

The PI assigned each individual a participant number at the time of the in-person/in-clinic screening. For all experimental procedures, the number, not the participant's name, was used to identify the participant. A file linking the participant's number and name was stored in a password protected electronic file (excel spreadsheet). Only the PI had access to the file. Signed informed consent documents were kept in a locked filing cabinet in the Voice Physiology and Motor Learning Laboratory at the University of Pittsburgh. Consent documents were not linked to the participant's unique identification number. All experimental data collected were housed in a separate password protected file. File naming convention for PAS6600 signals consisted of participant number, visit number, workload, and, finally, vocal goal.

2.5.6 Data reduction

The CareFusion Encore data acquisition system software used breath-by-breath analysis to record metabolic gas volume and concentration data. A research assistant recorded the times when participants started and stopped the phonatory R_{law} task to ensure metabolic gas volume and concentration data were time-locked with the voice data. These data were continuously recorded during all periods of silence as well as while participants completed the phonatory R_{law} task in spontaneous and loud vocal goal conditions for each workload, including the rest condition when participants just stood on the treadmill. Mean Ve, VO_2, VCO_2, $P_{et}CO_2$, and RER baseline values were calculated by averaging breath-by-breath data during a 30-second period preceding the start of voicing. For voiced segments, mean Ve data were calculated from the

entire period during which the participant phonated (i.e., from beginning to end of the phonatory R_{law} task). Metabolic gas concentration data including VO_2, VCO_2, $P_{et}CO_2$, and RER were extracted from an approximately 15-second time segment immediately following the completion of the phonatory R_{law} task.

To compute phonatory R_{law}, the PI calculated P_s and U from the PAS6600 aerodynamic signal. The PI was not blinded to condition when analyzing the aerodynamic signal but adhered to strict criteria for selection of data. For calculation of estimated P_s, the middle five pressure peaks generated during /p/ of the respective signals were manually selected and examined for acceptable morphology, intensity, and frequency (L.B. Helou & N.P. Solomon, 2011; E. B. Holmberg et al., 1988; Smitheran & Hixon, 1981). Morphology was considered acceptable if pressure peaks were not pointy or jagged, and flow minima correspond with pressure maxima, based on visual inspection (L.B. Helou & N.P. Solomon, 2011). Therefore, signals were inspected for pressure return to zero baseline during vowel, and airflow return to zero baseline (< 0.10 ml/s) during consonant production (L.B. Helou & N.P. Solomon, 2011; E. B. Holmberg et al., 1988; Smitheran & Hixon, 1981). Fundamental frequency during all vocal goal conditions could vary by ±2 ST. Vocal intensity during the loud condition could fluctuate by ±2 dB. Only samples that did not meet the predefined aerodynamic morphology were discarded. Once deemed acceptable, the average pressure values for peaks two through five from the respective syllables were calculated using the PAS 6600 software (KayPENTAX, 2009) and retained for statistical analysis. The corresponding flow signal during the voiced /æ:/ was also manually selected and inspected. Only airflow values during the vowel portion that corresponded to those pressure peaks meeting morphology criteria were calculated using the PAS6600 software and retained for statistical analysis.

Fundamental frequency (F_0) values were also calculated by the PAS6600 system for the acceptable corresponding pressure peaks. However, some pitch contours displayed on the PAS6600 output screen were incorrect and therefore the corresponding F_0 values were not accurate. Accordingly, the researcher used an online digital tone generator (http://www.seventhstring.com/tuningfork/tuningfork.html)(Limited, 2009) to confirm the F_0 value calculated by the PAS6600 system by listening to the participant's production using the playback function. In the case that the PAS6600 miscalculated the participant's F_0, the PI used the nearest frequency of the tone produced by the online generator that best corresponded to the pitch of the participant's production. Vocal intensity values also corresponding with the acceptable pressure peaks were calculated using the PAS6600 software and retained for statistical analysis. The PI adjusted the vocal intensity values calculated by the PAS6600 system by summing the PAS6600 value and the previously established calibration correction factor.

Percentage change from baseline was computed for R_{law}, P_s, U, Ve, VO_2, VCO_2, $P_{et}CO_2$, and RER. For phonatory outcome measures, values from spontaneous voice at rest, which constituted baseline for phonatory variables, were subtracted from those values in every other condition. The difference was then divided by the baseline value and multiplied by 100 to derive a percentage change value from baseline. For respiratory outcome measures, the same process allowed for the computation of percentage change values. However, for respiratory outcome measures, baseline consisted of silence at rest.

2.5.7 Statistical analysis

The PI conducted all statistical analyses using SPSS 21.0 (IBM, Armonk, NY). The main data of interest were derived from the repeated /pæ:/ task, which provided simultaneous phonatory and

respiratory data. For female participants, for each SA, a two-way within-subjects Analysis of Variance (ANOVA) was conducted to evaluate relations between vocal goal (2 or 3 levels: silence [respiratory DVs only], spontaneous and loud phonation) and workload (2 levels: low and high) on the percentage change from baseline values of the phonatory (R_{law}, P_s, U) and respiratory (Ve, $P_{et}CO_2$, VO_2, VCO_2, RER) variables. If a significant interaction effect of the within-subjects independent variables was found, simple main effects were examined to understand the nature of that interaction, following up on any significant simple main effect with post-hoc analyses consisting of multiple comparisons using a Bonferroni correction. In cases where the interaction effect was not significant, main effects are reported and interpreted. Due to the study's experimental design, conditions that contained no analyzable data of a particular variable require exclusion of all that participant's data of that variable from the final analysis.

Data from male subjects were explored descriptively only. These preliminary data for males were used to obtain an early window on the extent to which results were consistent with those for females, to be pursued in future specifically designed studies on potential gender effects. Due to the small sample size of male participants, an interpretation of sex differences will not be provided beyond providing the descriptive statistics for both female and male participants.

Contrary to study goals, F_0 and SPL were not met for all participants. Consequently, analyses of F_0 and SPL were also conducted to look systematically at how they varied. Analysis of F_0 and SPL utilized the actual values, as opposed to percentage change values, because both are logarithmic scales. Due to multiple baselines of F_0 and SPL at rest, averaging the respective values from each experimental day permitted a collapse of the multiple baseline values completed in the rest condition to yield one value. For those participants who only had one value,

that value was used. For statistical analysis, F_0 and SPL values were submitted to a two-way within subjects ANOVA to evaluate relations between vocal goal (2 levels: spontaneous and loud phonation) and workload (3 levels: rest, low, and high).

3.0 RESULTS

The three assumptions applied to a within-subjects repeated measures design include sphericity, normality, and independence. In regards to the first assumption, Huynh and Feldt (1970) showed that satisfying sphericity (i.e., equal variances of the differences of the dependent variable between all combinations of levels of the repeated measures factor satisfy this assumption) is sufficient and necessary for a within-subject *F* test in repeated measures designs. The assumption of sphericity was met for some, but not all, of the respiratory dependent variables, as assessed by Mauchly's test of sphericity. For a repeated measure factor with only two levels, sphericity is always met and is, therefore, unnecessary to evaluate (Hinton, McMurray, & Brownlow, 2014). Depending on the degree of non-sphericity, as assessed by epsilon, Greenhouse-Geisser ($\varepsilon \leq .750$) or Huynh-Feldt ($\varepsilon > .750$) estimates of sphericity corrected for this violation by adjusting degrees of freedom.

Relative to the second assumption, visual inspection of boxplots and detrended Q-Q plots revealed outliers that were more than 1.5 box-lengths from the edge of the box in the boxplots and had absolute detrended values of greater than or equal to 1.0 in the detrended Q-Q plots, and removal of outliers improved the symmetry of the distribution and most normality violations. That is, only a few of the phonatory and respiratory dependent variables did not demonstrate a normal distribution at each level of the within-subjects factors. Therefore, most, but not all, of the phonatory and respiratory dependent variables met the assumption of normality, as assessed

by the Shapiro-Wilk's test ($p < .05$). Importantly, within-subjects ANOVA is robust against asymmetric distributions as non-normality minimally impacts the level of significance and power of the non-directional *F* test (Glass, Peckham, & Sanders, 1972). Accordingly, the remaining asymmetric distributions that violated the assumption of normality did not undergo transformation. However, non-parametric analysis followed up on any violation of normality. The non-parametric results are reported only when the results of the parametric and non-parametric analyses differed.

The assumption of independent observations is a far more serious issue because non-independence has a significant effect on both the level of significance and power of the *F*-test (Glass et al., 1972). To satisfy the independence assumption, the spontaneous and loud voice goals and the 50% and 70% workloads were counterbalanced and randomly assigned to participants. The significance level was set at an alpha level of .05 for all analyses.

3.1 PARTICIPANTS

One hundred and ninety-nine individuals completed an online pre-screening form to determine initial eligibility. Of those, 32 recreationally active and vocally untrained females met inclusion/exclusion criteria and completed all experimental procedures. To preserve the target sample size, one female participant replaced an individual who experienced an unanticipated adverse event. Therefore, the results reflect analyses completed on the data of 32 female individuals. Additionally, four males who were recreationally active and vocally untrained also participated in and completed the experiment as volunteers. Violations in the pressure and airflow morphology criteria in at least one condition excluded the use of data from 15 female

participants in inferential statistical analysis of R_{law}. For females, fourteen percent (37 out of 256) of the total number of cells containing R_{law} data were excluded from analysis for not meeting morphology criteria. In considering missing data from specific conditions, 9% of cells were from spontaneous voice at rest, 14% of cells were from loud voice at rest, 19% cells were from spontaneous voice during the low workload, 19% of cells were from loud voice during the low workload, 19% of cells were from spontaneous voice during the high workload, and 13% of cells were from loud voice during the high workload. Figure 6 provides a flow chart showing the course of the study from initial screening through data analysis. Participant characteristics are shown in Table 8 and Table 9.

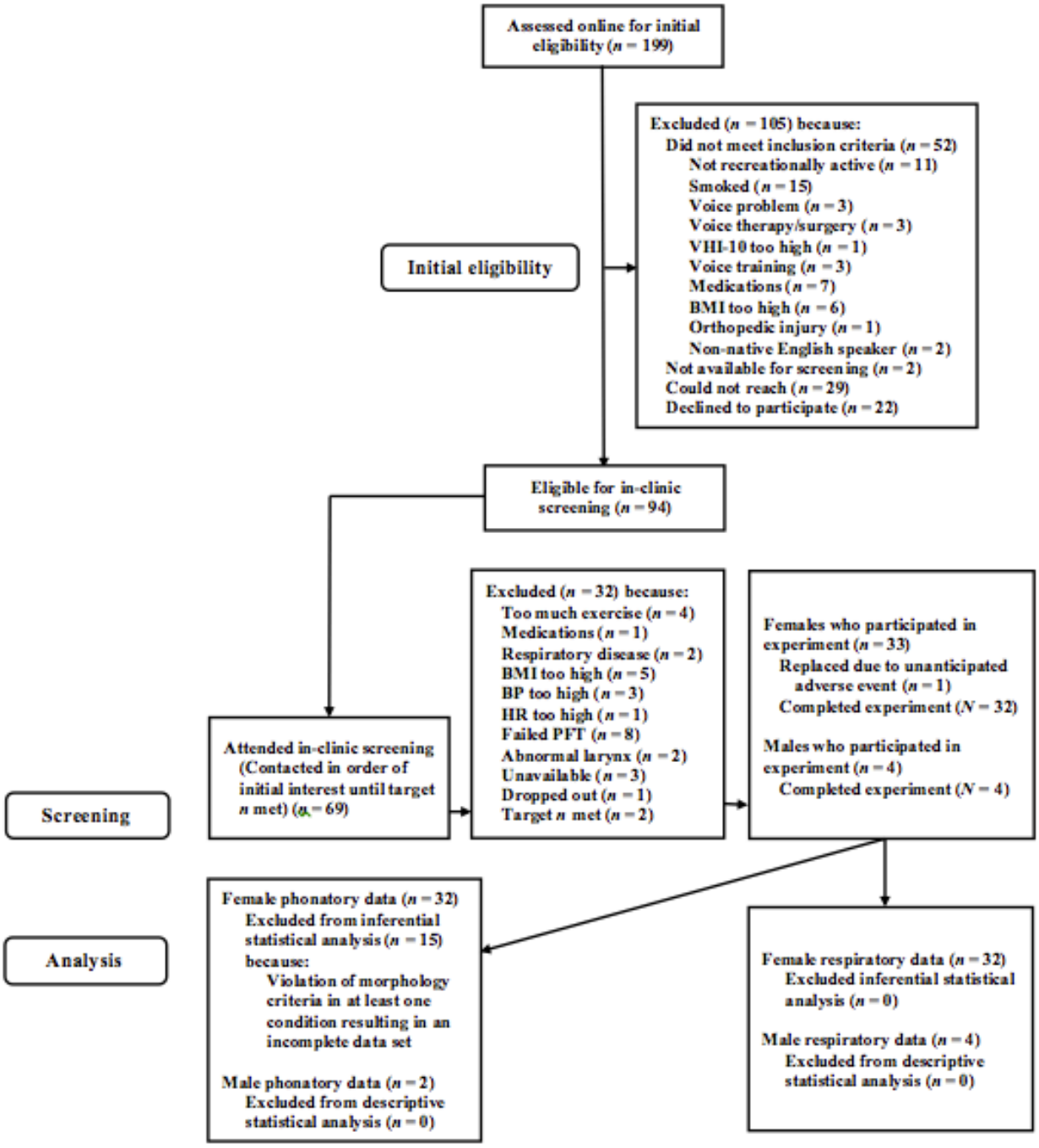

Figure 6. Flowchart of study procedures.

Table 8. Distribution of Racial and Ethnic Identity by Sex

Characteristic	Female (*N* = 32)	Male (*N* = 4)
Race		
White/Caucasian	29	3
Black/African-American	1	0
Asian	1	1
American Indian/Alaska Native	1	0
Ethnicity		
Hispanic	5	1
Non-Hispanic	27	3

Table 9. Participant Physical and Voice Characteristics

Characteristic	Female *M* (*SD*)	Male *M* (*SD*)
Age (years)	23.0 (3.4)	24.8 (3.4)
Body weight (kg)	59.8 (7.4)	69.5 (13.0)
Body height (cm)	165.5 (6.5)	178.4 (11.0)
BMI	21.8 (2.2)	21.8 (1.7)
Heart rate (bpm)	69.4 (9.2)	65.3 (8.6)
Systolic blood pressure (mm Hg)	99.6 (7.4)	110.8 (5.1)
Diastolic blood pressure (mm Hg)	64.9 (5.4)	66.5 (6.4)
FVC[a] (L; % predicted)	4.0 (0.5) (98%)	5.3 (1.2) (95%)
FEV1[a] (L; % predicted)	3.4 (0.3) (97%)	4.4 (0.8) (95%)
CAPE-V rating (mm)	3.4 (2.3)	2.5 (1.9)
VHI-10 score	1.9 (2.0)	3.5 (3.3)

Note. BMI = body mass index; FVC = forced vital capacity; FEV1 = forced expiratory volume in 1st s; CAPE-V = Consensus Auditory-Perceptual Evaluation of Voice; VHI-10 Voice Handicap Index-10.

[a]% predicted based on normative data from Crapo et al., 1981.

3.2 SATISFACTION OF TASK REQUIREMENTS

The study set out to control for fundamental frequency (F_0) in all voice conditions and vocal intensity (SPL) in the loud voice condition. Although participants received target pitches from a pitch pipe prior to each utterance and also "live" from the PI in the appropriate octave, and participants received verbal prompts from the PI about raising or lowering F_0 as appropriate, most participants failed to meet pitch criteria during the actual experiment, even though they demonstrated pitch-matching capabilities during screening. However, all participants maintained a F_0 within the modal register for all conditions throughout the study as evaluated perceptually during the experiment and verified with acoustic analysis. Given that the majority of participants failed to meet pitch criteria in at least one condition and since fundamental frequency lacks a clear, consistent influence over R_{law}, statistical analyses did not exclude any participant data due to a violation of the fundamental frequency task constraint.

Additionally, some participants overshot the individualized SPL target by more than two decibels, especially during low and high workloads. However, all participants performed spontaneous and high effort types of phonation that were categorically different from one another and represented two distinct manners of voicing. Accordingly, analysis of phonatory and respiratory primary and secondary variables included the data from all participants irrespective of whether or not they violated the loudness task constraint, as well as follow-up analyses of those data from participants who met their loudness goal. Taking into consideration the failure to meet task constraints, both F_0 and SPL were analyzed in order to understand the nature of F_0 and SPL violations.

3.2.1 Fundamental Frequency (F_0)

The means and standard deviations of F_0 for females and males are reported in Table 10 and 11. A 2 × 3 repeated measures ANOVA was performed on F_0 values as a function of voice goal (spontaneous, loud) and workload (rest, low, high). One participant had missing data due to a technical issue. There were four outliers detected and removed. Mauchly's test of sphericity revealed the assumption of sphericity had been violated only for the workload effect [Mauchly's $W = .745$, $\chi^2(2) = 7.371$, $p = .025$]. The data were normally distributed in each condition except for F_0 values for spontaneous voice at the low [$W = .902$, $p = .015$] and high workloads [$W = .913$, $p = .027$] (see Table 12). All other assumptions of ANOVA were met. This ANOVA revealed significant main effects of voice goal [$F(1, 26) = 53.233$, $p < .001$, partial $\eta^2 = .672$] and workload [$F(1.680, 43.693) = 15.246$, $p < .001$, partial $\eta^2 = .370$]. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had significantly higher F_0 during loud voice ($M = 253.7$, $SE = 5.226$) than spontaneous ($M = 228.1$, $SE = 5.038$). In addition, participants had significantly higher F_0 in low ($M = 242.3$, $SE = 5.179$) and high workloads ($M = 251.3$, $SE = 6.510$) compared to rest ($M = 229.3$, $SE = 4.083$), $p = .001$ and $p < .001$, respectively, but there was no statistically significant difference in F_0 between low and high workloads, $p = .232$. The interaction of voice goal and workload was not significant [$F(2, 52) = 1.205$, $p = .308$, partial $\eta^2 = .044$].

Table 10. Group Means and Standard Deviations of Fundamental Frequency and Vocal Intensity During Three Workloads and Two Vocal Goals in All Women

	F0 (Hz)		SPL	
	M	*SD*	*M*	*SD*
Rest				
Spontaneous	217.9	21.0	86.5	3.3
Loud	240.7	23.9	95.1	2.7
Low Workload				
Spontaneous	229.4	29.2	91.3	4.4
Loud	255.2	30.1	97.3	3.6
High Workload				
Spontaneous	237.2	35.2	91.6	3.9
Loud	265.4	36.0	97.3	2.7

Note. F_0 = fundamental frequency; SPL = sound pressure level.

Table 11. Group Means and Standard Deviation of Fundamental Frequency and Vocal Intensity During Three Workloads and Two Vocal Goals in All Men

	F_0 (Hz)		SPL (dB)	
	M	*SD*	*M*	*SD*
Rest				
Spontaneous	122.9	14.5	94.0	3.0
Loud	162.5	17.2	100.8	1.4
Low Workload				
Spontaneous	136.3	26.5	97.6	1.8
Loud	172.3	22.3	102.5	2.1
High Workload				
Spontaneous	129.7	13.6	97.4	3.5
Loud	193.6	39.3	106.2	0.6

Note. F_0 = fundamental frequency; SPL = sound pressure level.

Table 12. Test of Normality on Fundamental Frequency as a Function of Workload and Voice Goal

Condition	Shapiro-Wilk *W*	*df*	*p*
Spontaneous/Rest	.960	27	.376
Spontaneous/Low	.902	27	.015
Spontaneous/High	.913	27	.027
Loud/Rest	.982	27	.903
Loud/Low	.967	27	.527
Loud/High	.958	27	.340

3.2.2 Vocal Intensity (SPL)

The means and standard deviations of SPL for females and males are reported in Tables 10 and 11. A 2 × 3 repeated measures ANOVA was performed on SPL as a function of voice goal (spontaneous, loud) and workload (rest, low, high). Six participants had incomplete data sets. The data were normally distributed except for SPL values for loud voice at the low workload [W = .903, p = .018] (see Table 13). All other assumptions of ANOVA were met. This ANOVA revealed a significant interaction effect of voice goal and workload [$F(2, 50) = 11.347$, $p < .001$, partial $\eta^2 = .312$], meaning that the effect of workload on SPL depends on the level of voice goal. Additionally, the ANOVA revealed significant main effects of voice goal [$F(1, 25) = 141.764$, $p < .001$, partial $\eta^2 = .850$] and workload [$F(2, 50) = 45.385$, $p < .001$, partial $\eta^2 = .645$].

Table 13. Test of Normality on Sound Pressure Level as a Function of Workload and Voice Goal

Condition	Shapiro-Wilk *W*	*df*	*p*
Spontaneous/Rest	.953	26	.268
Spontaneous/Low	.949	26	.220
Spontaneous/High	.949	26	.219
Loud/Rest	.945	26	.179
Loud/Low	.903	26	.018
Loud/High	.986	26	.970

In order to find the pattern of differences on SPL depending on workload between spontaneous and loud voice, the simple main effect of workload was examined for each level of voice goal. The significant differences on SPL among workload for each voice goal were followed by simple comparisons. In addition, simple main effect of voice goal was examined for each level of workload by a paired samples *t*-test since it has only two levels. The simple main effect of workload for spontaneous [$F(2, 50) = 44.811$, $p < .001$, partial $\eta^2 = .642$] and loud voice [$F(2, 52) = 14.026$, $p < .001$, partial $\eta^2 = .350$] was significant. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had significantly higher SPL during spontaneous voice production in low as well as high workloads compared to rest, $p < .001$ and $p < .001$, respectively, but there was no difference in SPL during spontaneous voice between low and high workloads, $p = 1.000$. In addition, the participants had significantly higher SPL during loud voice in low as well as high workloads compared to rest, $p < .001$ and $p < .001$, respectively, but there was no difference in SPL during loud voice between low and high workloads, $p = 1.000$.

The simple main effect of voice goal for rest, low, and high workloads was significant. At each workload, participants had higher SPL in loud voice compared to spontaneous: rest [$t(31) = 15.684$, $p < .001$], low [$t(27) = 7.138$, $p < .001$], and high [$t(29) = 9.089$, $p < .001$].

Due to a violation of normality during loud voice at the low workload, the non-parametric Related-samples Friedman's Test was completed. The Friedman Test revealed there was a statistically significant difference in SPL in the different conditions, ($X^2(5) = 96.022$, $p < .001$). Like the findings of the 2 × 3 repeated measures ANOVA, median SPL was significantly higher during spontaneous voice in low (91.0) as well as high (91.4) workloads compared to rest (86.6), $p = .004$ and $p = .010$, respectively, but there was no difference in SPL during spontaneous voice between low and high workloads, $p = 1.000$. Contrary to the findings repeated measures ANOVA, there was no significant difference in median SPL during loud voice between rest (94.5) and low (97.3), rest and high (97.0), nor low and high workloads, $p = .392$, $p = .569$, and $p = 1.000$. Again, similar to findings of the repeated measures ANOVA, median SPL was significantly higher during loud voice compared to spontaneous during each workload (rest: $p < .001$, low: $p < .001$, high: $p < .001$).

3.3 CONFIRMATION OF TARGET WORKLOADS

A paired-samples *t*-test was performed to determine if a difference existed between target heart rate and observed heart rate at the low and high workloads in females. One outlier was detected at the high workload. The difference scores at the low workload violated the assumption of normality [$W(32) = .921$, $p = .022$] (Table 14). All other assumptions were met. There were no significant differences between target and observed heart rates at the low [133.1 (5.0) vs. 134.1

(5.8)] and high [158.6 (3.7) vs. 159.1 (5.5)] workloads, $t(31) = 1.599$, $p = .120$, $d = 0.28$ and $t(31) = 0.758$, $p = .454$, $d = 0.13$, respectively. These findings confirmed that participants exercised at the target workload.

Table 14. Test of Normality on Difference Scores Between Target and Observed Heart Rates in Females as Function of Workload

Workload	Shapiro-Wilk *W*	*df*	*p*
Low	0.921	32	.022
High	0.938	32	.067

To determine if females completed three different workloads, a one-way repeated measures analysis of variance (ANOVA) was performed on observed heart rates during silence as a function of workload. Heart rates at the rest workload violated the assumption of normality [$W(32) = .911$, $p = .012$] (Table 15). Mauchly's test of sphericity indicated that the assumption of sphericity had also been violated [Mauchly's $W = .661$, $X^2(2) = 12.426$, $p = .002$]. The ANOVA for observed heart rates revealed a significant main effect of workload [$F(1.494, 46.299) = 2866.643$, $p < .001$, $\eta^2 = .989$]. *Post hoc* analysis performed with a Bonferroni adjustment revealed mean heart rate was significantly different for all pairwise comparisons, in the predicted directions (rest [69.4 (9.6)] vs. low [134.1 (5.8)], $p < .001$; rest vs. high [159.1 (5.5)], $p < .001$; and low vs. high, $p < .001$). These findings confirmed that participants completed three different workloads. For males, descriptively, the mean observed heart rates at rest, low workload, and high workload were 65.3 (8.6), 128.8 (2.6), and 154.0 (8.4), respectively.

Table 15. Test of Normality on Heart Rates in Females as a Function of Workload

Workload	Shapiro-Wilk *W*	*df*	*p*
Rest	0.911	32	.012
Low	0.989	32	.985
High	0.981	32	.817

3.4 PHONATORY PRIMARY OUTCOMES

Going forward, descriptive and inferential statistics of phonatory variables for all participants are presented first. Subsequent to the overall group analysis, follow up analysis of the phonatory variables using inferential statistics examine the data from a subset of female participants who produced valid pressure peaks in conjunction with utterances satisfying SPL goals in the loud voice condition at all workload levels (See Figure 7).

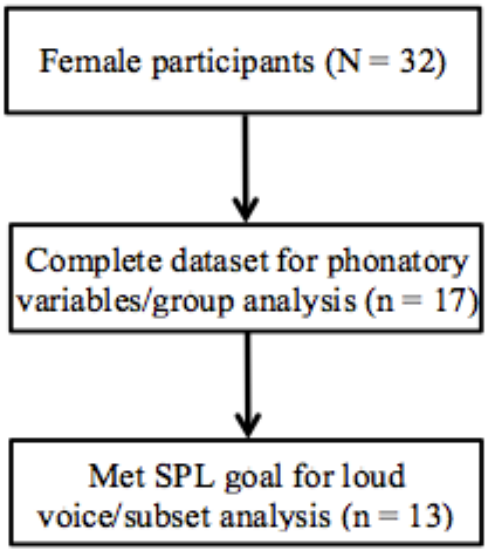

Figure 7. Flowchart of statistical analysis of phonatory measures.

3.4.1 Laryngeal airway resistance (R_{law})

Descriptive results of the actual R_{law} values for both females and males are displayed in Tables 16 and 17. For females, in the spontaneous voice condition, mean R_{law} increased from 62.6 (20.3) cm H_2O/L/s at rest to 69.8 (33.3) cm H_2O/L/s and 69.3 (28.6) cm H_2O/L/s at low and high workloads, respectively. In the loud voice condition, mean R_{law} decreased from 120.5 (45.8) cm H_2O/L/s at rest to 104.1 (40.5) cm H_2O/L/s and 92.7 (42.4) cm H_2O/L/s at low and high workloads, respectively. For males, in the spontaneous voice condition, mean R_{law} decreased from 40.6 (15.6) cm H_2O/L/s at rest to 28.0 (9.3) cm H_2O/L/s and 24.6 (0.5) cm H_2O/L/s at low and high workloads, respectively. In the loud voice condition, mean R_{law} changed from 53.8 (25.7) cm H_2O/L/s at rest to 53.4 (25.0) cm H_2O/L/s at the low workload, and decreased to 45.3 (32.2) cm H_2O/L/s at the high workload.

Table 16. Group Means and Standard Deviations of Phonatory Measures for All Females During Three Workloads and Two Voice Goals

	R_{law} (cm H_2O/L/s)		P_s (cm H_2O)		U (L/s)	
	M	*SD*	*M*	*SD*	*M*	*SD*
Rest						
Spontaneous	62.6	20.3	9.5	2.3	0.16	0.05
Loud	120.5	45.8	16.5	4.2	0.15	0.05
Low Workload						
Spontaneous	69.8	33.3	12.0	3.4	0.20	0.07
Loud	104.1	40.5	17.0	4.1	0.18	0.06
High Workload						
Spontaneous	69.3	28.6	12.7	3.4	0.21	0.07
Loud	92.7	42.4	16.8	5.6	0.20	0.07

Note. R_{law} = phonatory laryngeal airway resistance; P_s = estimated subglottal pressure; U = translaryngeal airflow.

Table 17. Group Means and Standard Deviations of Phonatory Measures for All Males During Three Workloads and Two Voice Goals

	R_{law} (cm H_2O/L/s)		P_s (cm H_2O)		U (L/s)	
	M	*SD*	*M*		*M*	*SD*
Rest						
Spontaneous	40.6	15.6	7.2	0.8	0.19	0.05
Loud	53.8	25.7	14.5	5.9	0.28	0.02
Low Workload						
Spontaneous	28.0	9.3	8.6	1.0	0.33	0.10
Loud	53.4	25.0	18.4	4.8	0.37	0.09
High Workload						
Spontaneous	24.6	0.5	9.8	3.2	0.33	0.01
Loud	45.3	32.2	15.8	6.3	0.41	0.14

Note. R_{law} = phonatory laryngeal airway resistance; P_s = estimated subglottal pressure; U = translaryngeal airflow.

Table 18 and Figure 8 displays results for percentage change in R_{law}, for female subjects. A 2 × 2 repeated measures ANOVA was performed on the R_{law} percentage change values as a function of voice goal (spontaneous, loud) and workload (low, high). One outlier in the R_{law} percentage change values was detected and removed. All assumptions for ANOVA were met. This ANOVA revealed significant main effects of voice goal [$F(1, 15) = 18.298$, $p = .001$, $\eta^2 = .550$] and workload [$F(1, 15) = 7.064$, $p = .018$, $\eta^2 = .320$]. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a larger mean percentage change in R_{law} from baseline in loud voice ($M = 0.599$, $SE = 0.119$) compared to spontaneous voice ($M = 0.244$, $SE = 0.094$). In addition, participants had a larger mean percentage change in R_{law} in low workload ($M = 0.566$, $SE = 0.130$) compared to high ($M = 0.277$, $SE = 0.092$). The interaction of voice goal and workload was not significant [$F(1, 15) = 2.414$, $p = .141$, $\eta^2 = .139$].

Table 18. Group Means and Standard Deviations of Percentage Change in Phonatory Laryngeal Airway Resistance as a Function of Workload and Voice Goal for Females who Met Vocal Intensity Goal Versus All Females

	Low Workload		High Workload	
	Met Goal	All	Met Goal	All
Vocal Goal	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Spontaneous	15.0 (28.2)	33.3 (46.4)	7.6 (30.9)	15.5 (35.5)
Loud	51.1 (41.0)	79.9 (65.3)	36.4 (52.8)	40.0 (48.5)

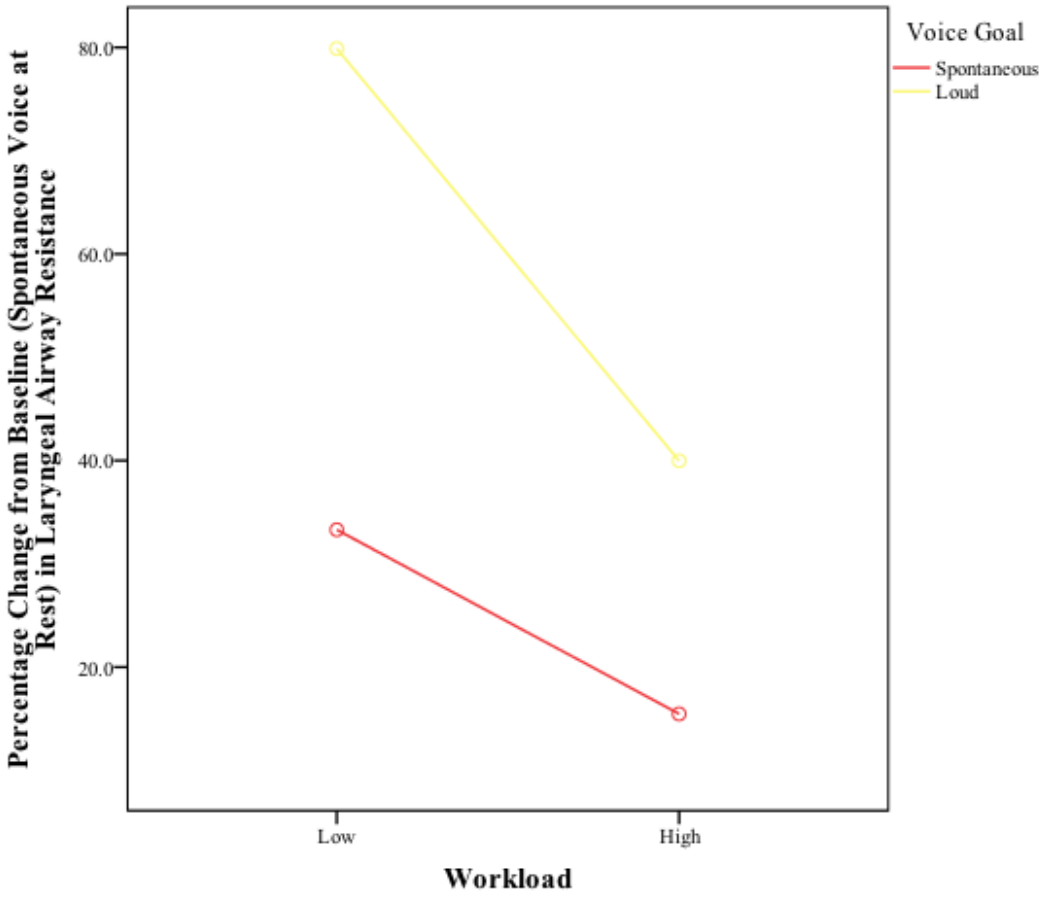

Figure 8. Comparisons of marginal means for percentage change from a baseline of spontaneous voice at rest in laryngeal airway resistance as a function of workload and voice goal.

3.4.1.1 Follow-up analysis for participants who met loud goal

For participants who satisfied SPL criteria in the loud condition for all workloads, a 2 × 2 repeated measures ANOVA was performed on the R_{law} percentage change values as a function of voice goal (spontaneous, loud) and workload (low, high). One outlier was detected and removed. All assumptions of ANOVA were met. This ANOVA revealed only a significant main effect of voice goal [$F(1, 11) = 12.623$, $p = .005$, $\eta^2 = .534$]. *Post hoc* pairwise comparisons using a

Bonferroni adjustment revealed participants had a larger mean percentage change in R_{law} in loud voice (M = 0.438, SE = 0.120) compared to spontaneous (M = 0.113, SE = 0.082). The main effect of workload was not significant [$F(1, 11) = 2.533$, $p = .140$, $\eta^2 = .187$], nor was the interaction of voice goal and workload [$F(1, 11) = 0.276$, $p = .610$, $\eta^2 = .025$].

3.4.2 Components of laryngeal airway resistance

3.4.2.1 Subglottal pressure (P_s)

Descriptive results of the actual P_s values for both females and males are displayed in Tables 16 and 17. For female participants, in the spontaneous voice condition, mean P_s increased from 9.5 (2.3) cm H_2O at rest to 12.0 (3.4) cm H_2O and 12.7 (3.4) cm H_2O at the low and high workloads, respectively. In the loud voice condition, mean P_s stayed fairly constant at 16.5 (4.2) cm H_2O, 17.0 (4.1) cm H_2O, and 16.8 (5.6) cm H_2O at rest and the low and high workloads, respectively. For males, in the spontaneous voice condition, mean P_s increased from 7.2 (0.8) cm H_2O at rest to 8.6 (1.0) cm H_2O and 9.8 (3.2) cm H_2O at the low and high workloads, respectively. In the loud voice condition, mean P_s increased from 14.5 (5.9) cm H_2O at rest to 18.4 (4.8) cm H_2O at the low workload and then decreased to 15.8 (6.3) cm H_2O at the high workload.

Table 19 and Figure 9 displays results for percentage change in P_s, for female subjects. A 2 × 2 repeated measures ANOVA was performed on the percentage change in P_s values as a function of voice goal (spontaneous, loud) and workload (low, high). Two outliers in the percentage change in P_s values were detected and removed. All assumptions of ANOVA were met. This ANOVA revealed a significant main effect of voice goal [$F(1, 14) = 12.319$, $p = .003$, $\eta^2 = .468$]. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a larger mean percentage change in P_s in loud voice (M = 0.842, SE = 0.116) compared to

spontaneous (M = 0.414, SE = 0.043). The main effect of workload was not significant [F(1, 14) = 1.972, p = .182, η^2 = .123], nor was the interaction of voice goal and workload [F(1, 14) = 3.849, p = .070, η^2 = .216].

Table 19. Group Means and Standard Deviations of Percentage Change in Subglottal Pressure as a Function of Workload and Voice Goal for Females who Met Vocal Intensity Goal Versus All Females

	Low Workload		High Workload	
	Met Goal	All	Met Goal	All
Vocal Goal	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Spontaneous	34.3 (14.9)	41.6 (26.7)	36.4 (15.9)	41.2 (16.2)
Loud	84.8 (54.0)	94.8 (52.8)	74.8 (54.3)	73.6 (46.4)

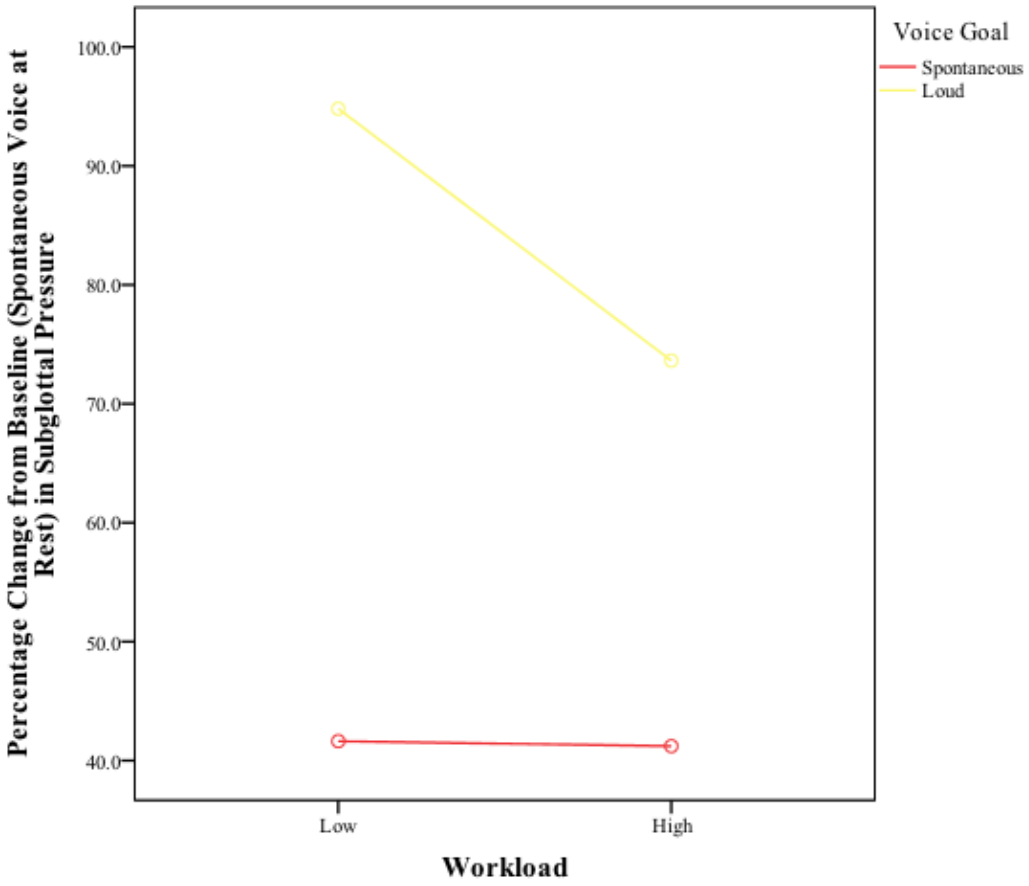

Figure 9. Comparisons of marginal means for percentage change from a baseline of spontaneous voice at rest in subglottal pressure as a function of workload and voice goal.

(a) Follow-up analysis for participants who met loud goal

For participants who satisfied SPL criteria in the loud condition for all workloads, a 2 × 2 repeated measures ANOVA was performed on the percentage change in P_s values as a function of voice goal (spontaneous, loud) and workload (low, high). Two outliers in the percentage change in P_s values were detected and removed. All assumptions of ANOVA were met. This ANOVA revealed only a significant main effect of voice goal [$F(1, 10) = 7.236$, $p = .023$, $\eta^2 = .420$]. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a

larger mean percentage change in P_s in loud voice (M = 0.798, SE = 0.154) compared to spontaneous (M = 0.353, SE = 0.034). The main effect of workload was not significant [$F(1, 10) = 0.309$, $p = .590$, $\eta^2 = .030$], nor was the interaction of voice goal and workload [$F(1, 10) = 1.188$, $p = .301$, $\eta^2 = .106$].

3.4.2.2 Translaryngeal airflow (U)

Descriptive results of the actual U values for both females and males are displayed in Tables 16 and 17. For females, in the spontaneous voice condition, mean U increased from 0.16 (0.05) L/s at rest to 0.20 (0.07) L/s and 0.21 (0.07) L/s at the low and high workloads, respectively. In the loud voice condition, mean U increased from 0.15 (0.05) L/s at rest to 0.18 (0.06) L/s and 0.20 (0.07) L/s at low and high workloads, respectively. For males, in the spontaneous voice condition, mean U increased from 0.19 (0.05) L/s at rest to 0.33 (0.10) L/s and 0.33 (0.01) L/s at low and high workloads, respectively. In the loud voice condition, mean U increased from 0.28 (0.02) L/s at rest to 0.37 (0.09) L/s and 0.41 (0.14) L/s at low and high workloads, respectively.

Table 20 and Figure 10 displays results for percentage change in U, for female subjects. A 2 × 2 repeated measures ANOVA was performed on the percentage change in U values as a function of voice goal (spontaneous, loud) and workload (low, high). One outlier in the percentage change in U values was detected and removed. All assumptions of ANOVA were met. Neither the main effect of voice goal [$F(1, 15) = 0.019$, $p = .891$, $\eta^2 = .001$] nor workload [$F(1, 15) = 2.726$, $p = .120$, $\eta^2 = .154$] were significant, nor was the interaction of voice goal and workload [$F(1, 15) = 0.013$, $p = .911$, $\eta^2 = .001$].

Table 20. Group Means and Standard Deviations of Percentage Change in Airflow as a Function of Workload and Voice Goal for Females who Met Vocal Intensity Goal Versus All Females

	Low Workload		High Workload	
Vocal Goal	Met Goal *M* (*SD*)	All *M* (*SD*)	Met Goal *M* (*SD*)	All *M* (*SD*)
Spontaneous	18.2 (21.8)	11.7 (24.3)	19.5 (25.0)	17.6 (23.9)
Loud	17.7 (28.1)	10.6 (28.3)	19.4 (34.1)	17.2 (31.4)

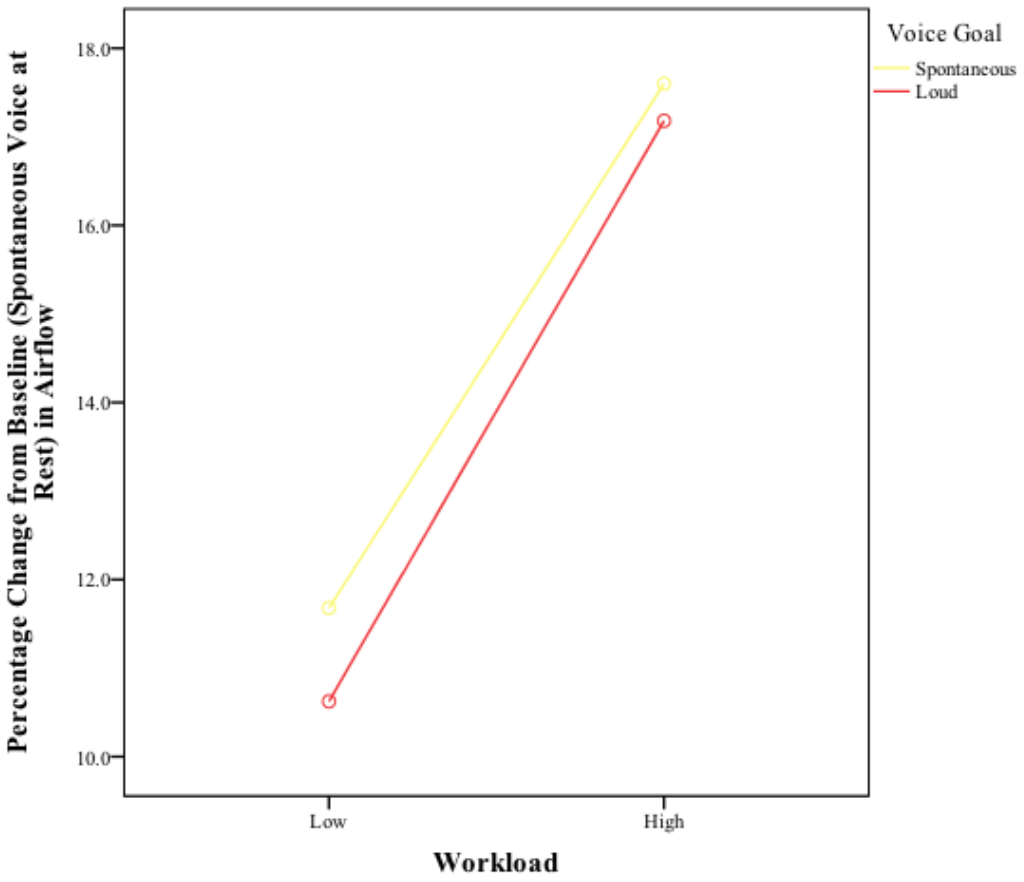

Figure 10. Comparisons of marginal means for percentage change from a baseline of spontaneous voice at rest in airflow as a function of workload and voice goal.

(a) Follow-up analysis for participants who met loud goal

For participants who satisfied SPL criteria in the loud condition for all workloads, a 2 × 2 repeated measures ANOVA was performed on the percentage change in U values as a function of voice goal (spontaneous, loud) and workload (low, high). One outlier in the percentage change in U values was detected and removed. All assumptions of ANOVA were met. The main effects of voice goal [$F(1, 11) = 0.002$, $p = .966$, $\eta^2 < .001$] and workload [$F(1, 11) = 0.129$, $p = .726$, $\eta^2 = .012$] were not significant, nor was the interaction of voice goal and workload [$F(1, 11) = 0.003$, $p = .956$, $\eta^2 < .001$].

3.5 RESPIRATORY PRIMARY OUTCOME

Going forward, descriptive and inferential statistics of respiratory variables for all participants are presented first. Subsequent to the overall group analysis, follow up analysis of the respiratory variables using inferential statistics examine the data from a subset of female participants whose average vocal intensity over the sets of the repeated /pæ:/ task met the loud SPL goal at all workload levels (see Figure 11).

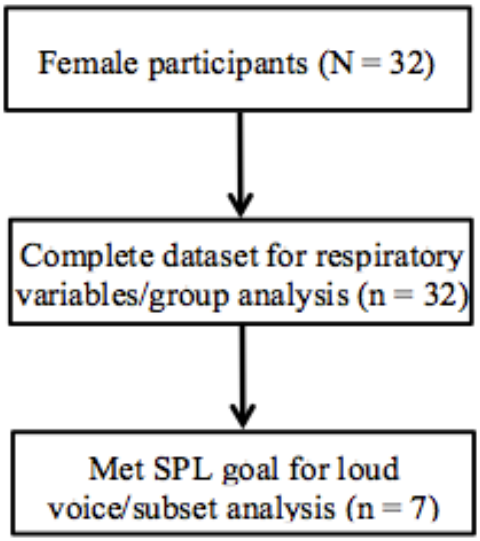

Figure 11. Flowchart of statistical analysis of respiratory measures.

3.5.1 Minute ventilation (Ve)

Descriptive results of the actual Ve values for both females and males are displayed in Tables 21 and 22. For females, in silence, mean Ve increased from 11.9 (1.7) L/min at rest to 36.1 (6.8) L/min and 46.6 (8.9) L/min at low and high workloads, respectively. In spontaneous voice, mean Ve increased from 9.7 (2.9) L/min at rest to 15.3 (4.5) L/min and 18.0 (4.0) L/min at low and high workloads, respectively. In loud voice, mean Ve decreased from 10.6 (2.7) L/min at rest to 16.4 (8.7) L/min and 19.3 (13.7) L/min at low and high workloads, respectively. For males, in silence, mean Ve increased from 12.7 (1.4) L/min at rest to 43.0 (11.1) L/min and 57.8 (12.4) L/min at low and high workloads, respectively. In spontaneous voice, mean Ve increased from 8.9 (3.1) L/min at rest to 12.2 (3.2) L/min and 16.8 (3.1) L/min at low and high workloads,

respectively. In loud voice, mean Ve increased from 8.5 (0.5) L/min at rest to 13.4 (4.5) L/min and 22.4 (7.7) L/min at low and high workloads, respectively.

Table 21. Group Means and Standard Deviations of Respiratory Measures for All Females During Three Workloads and Three Voice Goals

	Ve (L/min) *M* (*SD*)	$P_{et}CO_2$ (mm Hg) *M* (*SD*)	VO_2 (mL/kg/min) *M* (*SD*)	VCO_2 (mL/kg/min) *M* (*SD*)	RER (unitless) *M* (*SD*)
Rest					
Silent	11.9 (1.7)	32.1 (2.5)	4.5 (0.5)	3.4 (0.4)	0.75 (0.06)
Spontaneous	9.7 (2.9)	34.0 (2.2)	4.5 (0.7)	3.6 (0.7)	0.81 (0.14)
Loud	10.6 (2.7)	33.1 (2.0)	4.6 (0.8)	3.9 (0.8)	0.86 (0.15)
Low Workload					
Silent	36.1 (6.8)	43.7 (3.0)	23.5 (3.4)	20.4 (3.6)	0.86 (0.06)
Spontaneous	15.3 (4.5)	54.5 (4.5)	27.3 (4.1)	23.7 (4.5)	0.87 (0.11)
Loud	16.4 (8.7)	53.9 (4.1)	27.3 (4.3)	23.7 (4.7)	0.87 (0.12)
High Workload					
Silent	46.6 (8.9)	45.2 (3.7)	30.1 (4.0)	28.1 (4.1)	0.93 (0.05)
Spontaneous	18.0 (4.0)	59.5 (5.3)	35.2 (5.0)	32.6 (5.6)	0.93 (0.11)
Loud	19.3 (13.7)	59.1 (4.5)	35.0 (5.2)	32.8 (5.6)	0.94 (0.11)

Note. Ve = minute ventilation; $P_{et}CO_2$ = end-tidal carbon dioxide; VO_2 = oxygen consumption; VCO_2 = carbon dioxide production; RER = respiratory exchange ratio.

Table 22. Group Means and Standard Deviations of Respiratory Measures for All Males During Three Workloads and Three Voice Goals

	Ve (L/min) *M* (*SD*)	$P_{et}CO_2$ (mm Hg) *M* (*SD*)	VO_2 (mL/kg/min) *M* (*SD*)	VCO_2 (mL/kg/min) *M* (*SD*)	RER (unitless) *M* (*SD*)
Rest					
Silence	12.7 (1.4)	34.9 (1.5)	4.6 (0.6)	3.7 (0.4)	0.81 (0.04)
Spontaneous	8.9 (3.1)	36.7 (2.2)	4.5 (1.3)	3.6 (0.7)	0.83 (0.09)
Loud	8.5 (0.5)	34.9 (2.4)	4.3 (0.5)	3.8 (0.7)	0.90 (0.10)
Low Workload					
Silence	43.0 (11.1)	47.1 (4.3)	25.8 (4.5)	23.9 (5.3)	0.92 (0.06)
Spontaneous	12.2 (3.2)	58.3 (6.3)	29.8 (7.5)	28.2 (5.4)	0.96 (0.12)
Loud	13.4 (4.5)	56.7 (5.0)	28.6 (7.8)	27.9 (7.5)	0.98 (0.06)
High Workload					
Silence	57.8 (12.4)	52.2 (6.8)	33.7 (2.0)	33.3 (2.5)	0.99 (0.04)
Spontaneous	16.8 (3.1)	62.9 (4.1)	38.0 (6.4)	40.6 (3.6)	1.08 (0.10)
Loud	22.4 (7.7)	60.6 (4.6)	37.8 (6.4)	38.6 (4.0)	1.04 (0.15)

Note. Ve = minute ventilation; $P_{et}CO_2$ = end-tidal carbon dioxide; VO_2 = oxygen consumption; VCO_2 = carbon dioxide production; RER = respiratory exchange ratio.

Table 23 and Figure 12 displays results for percentage change in Ve, for female subjects. A 3 × 2 repeated measures ANOVA was performed on mean percentage change in Ve as a function of voice goal (silent, spontaneous, loud) and workload (low, high). Four outliers were detected and removed. Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for the main effect of voice [Mauchly's $W = .496$, $X^2(2) = 18.224$, $p < .001$] as well as for the interaction of voice and workload [Mauchly's $W = .429$, $X^2(2) = 22.034$, $p < .001$]. All other assumptions of ANOVA were met. This ANOVA revealed a significant interaction effect of voice goal and workload [$F(1.273, 34.362) = 44.702$, $p < .001$, partial $\eta^2 = .623$], meaning that the effect of workload on Ve depends on the level of voice goal, as well as significant main effects of voice goal [$F(1.330, 35.907) = 532.629$, $p < .001$, partial $\eta^2 = .952$] and workload [$F(1, 27) = 47.010$, $p < .001$, partial $\eta^2 = .635$].

Table 23. Group Means and Standard Deviations of Percentage Change in Minute Ventilation as a Function of Workload and Voice Goal for Females who Met Vocal Intensity Goal Versus All Females

	Low Workload		High Workload	
	Met Goal	All	Met Goal	All
Vocal Goal	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Silence	220.8 (59.2)	201.2 (47.2)	301.1 (31.6)	288.2 (66.0)
Spontaneous	36.4 (54.1)	23.6 (32.9)	60.3 (34.1)	50.3 (33.0)
Loud	42.1 (65.8)	27.8 (34.5)	47.1 (49.7)	47.3 (41.9)

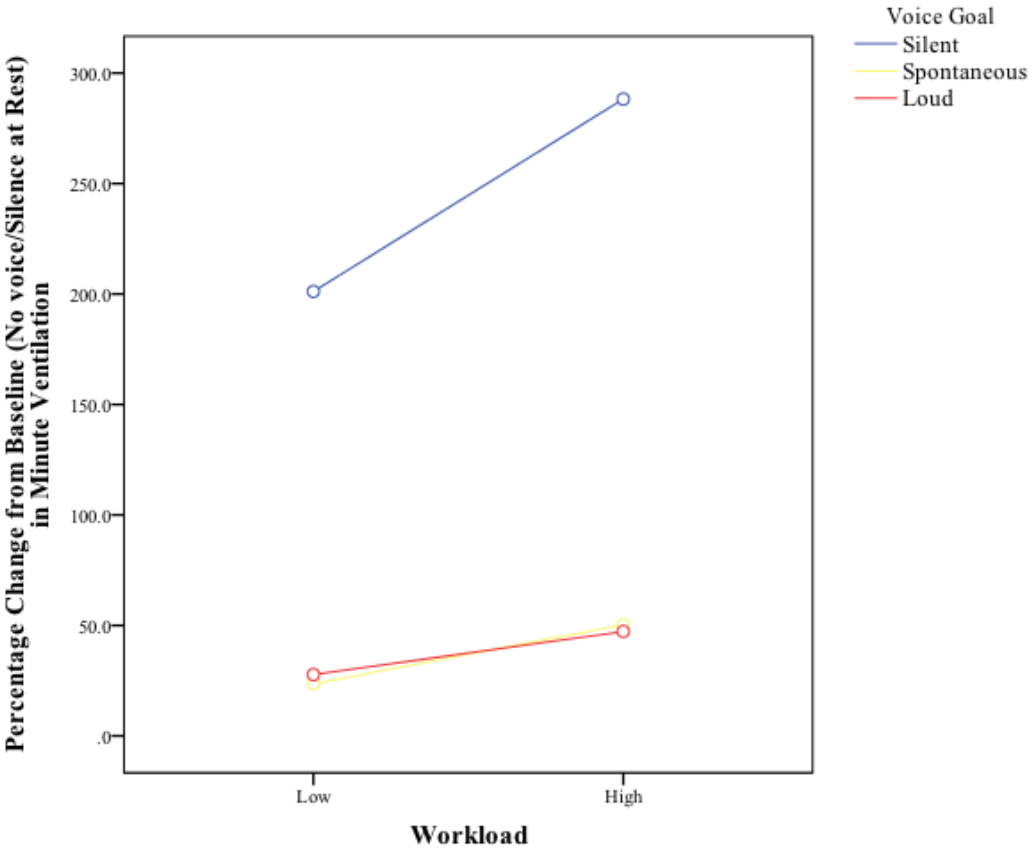

Figure 12. Comparisons of marginal means for percentage change from a baseline of no voice/silence at rest in minute ventilation as a function of workload and voice goal.

In order to find the pattern of differences on mean percentage change in Ve depending on voice goal between low and high workloads, simple main effect of voice goal was examined for each level of workload. The significant differences on mean percentage change in Ve among voice goal for each workload were followed by simple comparisons. In addition, simple main effect of workload was examined for each level of voice goal by a paired samples *t*-test since it had only two levels. The simple main effect of voice goal for low [$F(1.310, 35.378) = 364.771$, $p < .001$, partial $\eta^2 = .931$] and high workloads [$F(1.289, 34.803) = 464.766$, $p < .001$, partial $\eta^2 = .945$] was significant. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly greater mean percentage change in Ve during silence in low and high workloads compared to spontaneous and loud voice at both workloads, *p*s < .001, but there was no difference in mean percentage change in Ve between spontaneous and loud voice at both low and high workloads, *p*s = 1.000.

The simple main effect of workload for all three levels of voice goal was significant. Participants had a significantly greater mean percentage change in Ve in the high workload compared to low at all levels of voice: silence [$t(27) = 8.053$, $p < .001$], spontaneous [$t(27) = 4.570$, $p < .001$], and loud [$t(27) = 3.226$, $p = .003$].

3.5.1.1 Follow-up analysis for participants who met loud goal

For participants who satisfied SPL criteria in the loud condition for all workloads, a 3 × 2 repeated measures ANOVA was performed on mean percentage change in Ve as a function of voice goal (silent, spontaneous, loud) and workload (low, high). One outlier was detected and removed. All assumptions of ANOVA were met. This ANOVA revealed a significant interaction effect of voice goal and workload [$F(2, 10) = 10.893$, $p = .003$, partial $\eta^2 = .685$], meaning that the effect of workload on Ve depends on the level of voice goal. Additionally, the ANOVA

revealed only a significant main effect of voice goal [$F(2, 10) = 142.253$, $p < .001$, partial $\eta^2 = .966$]. The main effect of workload was not significant [$F(1, 5) = 5.759$, $p = .062$, partial $\eta^2 = .535$].

In order to find the pattern of differences on mean percentage change in Ve depending on voice goal between low and high workloads, simple main effect of voice goal was examined for each level of workload. The significant differences on mean percentage change in Ve among voice goal for each workload were followed by simple comparisons. In addition, simple main effect of workload was examined for each level of voice goal by a paired samples *t*-test since it has only two levels. The simple main effect of voice goal for low [$F(2, 10) = 87.836$, $p < .001$, partial $\eta^2 = .946$] and high workloads [$F(2, 10) = 126.965$, $p < .001$, partial $\eta^2 = .962$] was significant. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly greater mean percentage change in Ve during silence in low and high workloads compared to spontaneous and loud voice at both workloads, $p = .001$ (silence at low compared to spontaneous at low) and ps $< .001$ (all other comparisons), but there was no difference in mean percentage change in Ve between spontaneous and loud voice at both low and high workloads, $p = 1.000$ and $p = .926$, respectively.

There was only a significant simple main effect of workload in silence. Participants had a significantly greater mean percentage change in Ve in the high workload compared to low during silence [$t(5) = 3.944$, $p = .011$]. The simple main effect of workload during spontaneous [$t(5) = 1.737$, $p = .143$] and loud voice [$t(5) = 0.257$, $p = .807$] was not significant.

3.6 SECONDARY RESPIRATORY OUTCOMES

3.6.1 End-tidal carbon dioxide ($P_{et}CO_2$)

Descriptive results of the actual $P_{et}CO_2$ values for both females and males are displayed in Tables 21 and 22. For females, in silence, mean $P_{et}CO_2$ increased from 32.1 (2.5) mm Hg at rest to 43.7 (3.0) mm Hg and 45.2 (3.7) mm Hg at low and high workloads, respectively. In spontaneous voice, mean $P_{et}CO_2$ increased from 34.0 (2.2) mm Hg at rest to 54.5 (4.5) mm Hg and 59.5 (5.3) mm Hg at low and high workloads, respectively. In loud voice, mean $P_{et}CO_2$ increased from 33.1 (2.0) mm Hg at rest to 53.9 (4.1) mm Hg and 59.1 (4.5) mm Hg at low and high workloads, respectively. For males, in silence, mean $P_{et}CO_2$ increased from 34.9 (1.5) mm Hg at rest to 47.1 (4.3) mm Hg and 52.2 (6.8) mm Hg at low and high workloads, respectively. In spontaneous voice, mean $P_{et}CO_2$ increased from 36.7 (2.2) mm Hg at rest to 58.3 (6.3) mm Hg and 62.9 (4.1) mm Hg at low and high workloads, respectively. In loud voice, mean $P_{et}CO_2$ increased from 34.9 (2.4) mm Hg at rest to 56.7 (5.0) mm Hg and 60.6 (4.6) mm Hg at low and high workloads, respectively.

Table 24 and Figure 13 displays results for percentage change in $P_{et}CO_2$, for female subjects. A 3 × 2 repeated measures ANOVA was performed on mean percentage change in $P_{et}CO_2$ as a function of voice goal (silent, spontaneous, loud) and workload (low, high). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated only for the main effect of voice [Mauchly's W = .800, $X^2(2)$ = 6.702, p = .035]. All other assumptions of ANOVA were met. This ANOVA revealed a significant interaction effect of voice goal and workload [$F(2, 62)$ = 34.017, $p < .001$, partial η^2 = .523], meaning that the effect of workload on $P_{et}CO_2$ depends on the level of voice goal. Additionally, this ANOVA revealed

significant main effects of voice goal [$F(1.750, 54.240) = 583.699$, $p < .001$, partial $\eta^2 = .950$] and workload [$F(1, 31) = 48.468$, $p < .001$, partial $\eta^2 = .610$].

Table 24. Group Means and Standard Deviations of Percentage Change in End-tidal Carbon Dioxide as a Function of Workload and Voice Goal for Females who Met Vocal Intensity Goal Versus All Females

	Low Workload		High Workload	
	Met Goal	All	Met Goal	All
Vocal Goal	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Silence	31.8 (13.0)	35.8 (12.3)	35.3 (16.2)	42.7 (14.6)
Spontaneous	68.9 (17.4)	69.3 (14.3)	81.2 (23.7)	87.8 (17.8)
Loud	67.8 (19.6)	67.4 (15.7)	81.2 (22.6)	86.5 (16.5)

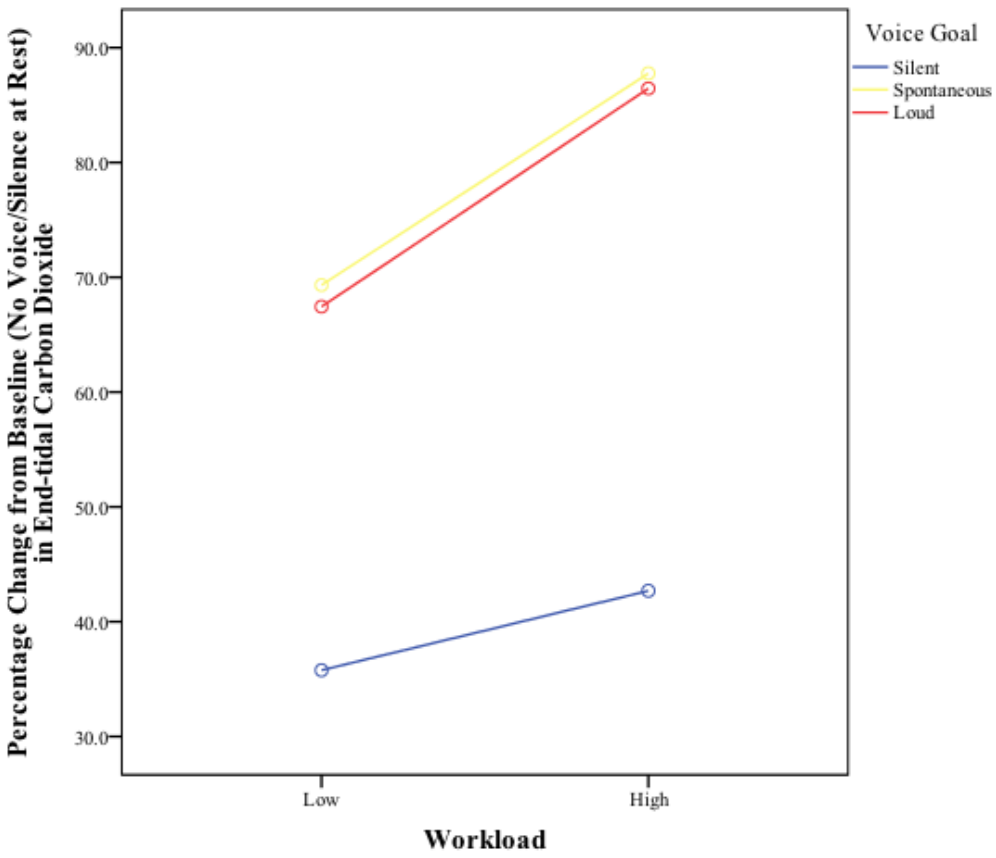

Figure 13. Comparisons of marginal means for percentage change from a baseline of no voice/silence at rest in end-tidal carbon dioxide as a function of workload and voice goal.

In order to find the pattern of differences on mean percentage change in $P_{et}CO_2$ depending on voice goal between low and high workloads, simple main effect of voice goal was examined for each level of workload. The significant differences on mean percentage change in $P_{et}CO_2$ among voice goal for each workload were followed by simple comparisons. In addition, simple main effect of workload was examined for each level of voice goal by a paired samples *t*-test since it has only two levels. The simple main effect of voice goal for low [$F(1.758, 54.506) = 359.054$, $p < .001$, partial $\eta^2 = .921$] and high workloads [$F(2, 62) = 473.017$, $p < .001$, partial η^2

= .938] was significant. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly smaller mean percentage change in $P_{et}CO_2$ during silence in low and high workloads compared to spontaneous and loud voice at both workloads, *p*s < .001, but there was no difference in mean percentage change in $P_{et}CO_2$ between spontaneous and loud voice at both low and high workloads, $p = .252$ and $p = 1.000$, respectively.

The simple main effect of workload for all three levels of voice goal was significant. Participants had a significantly greater mean percentage change in $P_{et}CO_2$ in the high workload compared to low at all levels of voice: silent [$t(31) = 3.784$, $p = .001$], spontaneous [$t(31) = 7.180$, $p < .001$], and loud [$t(31) = 7.550$, $p < .001$].

3.6.1.1 Follow-up analysis for participants who met loud goal

For participants who satisfied SPL criteria in the loud condition for all workloads, a 3 × 2 repeated measures ANOVA was performed on mean percentage change in $P_{et}CO_2$ as a function of voice goal (silent, spontaneous, loud) and workload (low, high). All assumptions of ANOVA were met. This ANOVA revealed a significant interaction effect of voice goal and workload [$F(2, 12) = 6.410$, $p = .013$, partial $\eta^2 = .517$], meaning that the effect of workload on $P_{et}CO_2$ depends on the level of voice goal. Additionally, this ANOVA revealed significant main effects of voice goal [$F(2, 12) = 130.400$, $p < .001$, partial $\eta^2 = .956$] and workload [$F(1, 6) = 22.013$, $p = .003$, partial $\eta^2 = .786$].

In order to find the pattern of differences on mean percentage change in $P_{et}CO_2$ depending on voice goal between low and high workloads, simple main effect of voice goal was examined for each level of workload. The significant differences on mean percentage change in $P_{et}CO_2$ among voice goal for each workload were followed by simple comparisons. In addition, simple main effect of workload was examined for each level of voice goal by a paired samples *t*-

test since it has only two levels. The simple main effect of voice goal for low [$F(2, 12) = 107.680$, $p < .001$, partial $\eta^2 = .947$] and high workloads [$F(2, 12) = 102.459$, $p < .001$, partial $\eta^2 = .945$] was significant. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly smaller mean percentage change in $P_{et}CO_2$ during silence in low and high workloads compared to spontaneous and loud voice at both workloads, *p*s < .001, but there was no difference in mean percentage change in $P_{et}CO_2$ between spontaneous and loud voice at both low and high workloads, *p*s = 1.000.

There was a significant simple main effect of workload for spontaneous and loud voice. Participants had a significantly greater mean percentage change in $P_{et}CO_2$ in the high workload compared to low in spontaneous [$t(6) = 4.456$, $p = .004$] and loud voice [$t(6) = 5.151$, $p = .002$]. The simple main effect of workload for silence was not significant [$t(6) = 1.258$, $p = .255$].

3.6.2 Oxygen consumption (VO_2)

Descriptive results of the actual VO_2 values for both females and males are displayed in Tables 21 and 22. Results are displayed in Table X and Figure Y. For females, in silence, mean VO_2 increased from 4.5 (0.5) mL/kg/min at rest to 23.5 (3.4) mL/kg/min and 30.1 (4.0) mL/kg/min at low and high workload, respectively. In spontaneous voice, mean VO_2 increased from 4.5 (0.7) mL/kg/min at rest to 27.3 (4.1) mL/kg/min and 35.2 (5.0) mL/kg/min at low and high workloads, respectively. In loud voice, mean VO_2 increased from 4.6 (0.8) mL/kg/min at rest to 27.3 (4.3) mL/kg/min and 35.0 (5.2) mL/kg/min at low and high workloads, respectively. For males, in silence, mean VO_2 increased from 4.6 (0.6) mL/kg/min at rest to 25.8 (4.5) L/min and 33.7 (2.0) mL/kg/min at low and high workloads, respectively. In spontaneous voice, mean VO_2 increased from 4.5 (1.3) mL/kg/min at rest to 29.8 (7.5) mL/kg/min and 38.0 (6.4) mL/kg/min at low and

high workloads, respectively. In loud voice, mean VO_2 increased from 4.3 (0.5) mL/kg/min at rest to 28.6 (7.8) mL/kg/min and 37.8 (6.4) mL/kg/min at low and high workloads, respectively.

Table 25 and Figure 14 displays results for percentage change in VO_2, for female subjects. A 3 × 2 repeated measures ANOVA was performed on mean percentage change in VO_2 as a function of voice goal (silent, spontaneous, loud) and workload (low, high). The assumption of normality was not met for only one condition (see Table 26). All other assumptions of ANOVA were met. This ANOVA revealed a significant interaction effect of voice goal and workload [$F(2, 62) = 5.850$, $p = .005$, partial $\eta^2 = .159$], meaning that the effect of workload on VO_2 depends on the level of voice goal. Additionally, the ANOVA revealed significant main effects of voice goal [$F(2, 62) = 66.009$, $p < .001$, partial $\eta^2 = .680$] and workload [$F(1, 31) = 73.382$, $p < .001$, partial $\eta^2 = .703$].

Table 25. Group Means and Standard Deviations of Percentage Change in Oxygen Consumption as a Function of Workload and Voice Goal in Females who Met Vocal Intensity Goal Versus All Females

	Low Workload		High Workload	
	Met Goal	All	Met Goal	All
Vocal Goal	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Silence	434.6 (75.9)	426.6 (67.7)	535.9 (113.8)	572.4 (113.2)
Spontaneous	471.7 (90.7)	505.6 (91.2)	624.5 (132.0)	689.5 (154.6)
Loud	526.4 (103.4)	510.6 (90.8)	616.2 (147.7)	685.5 (163.8)

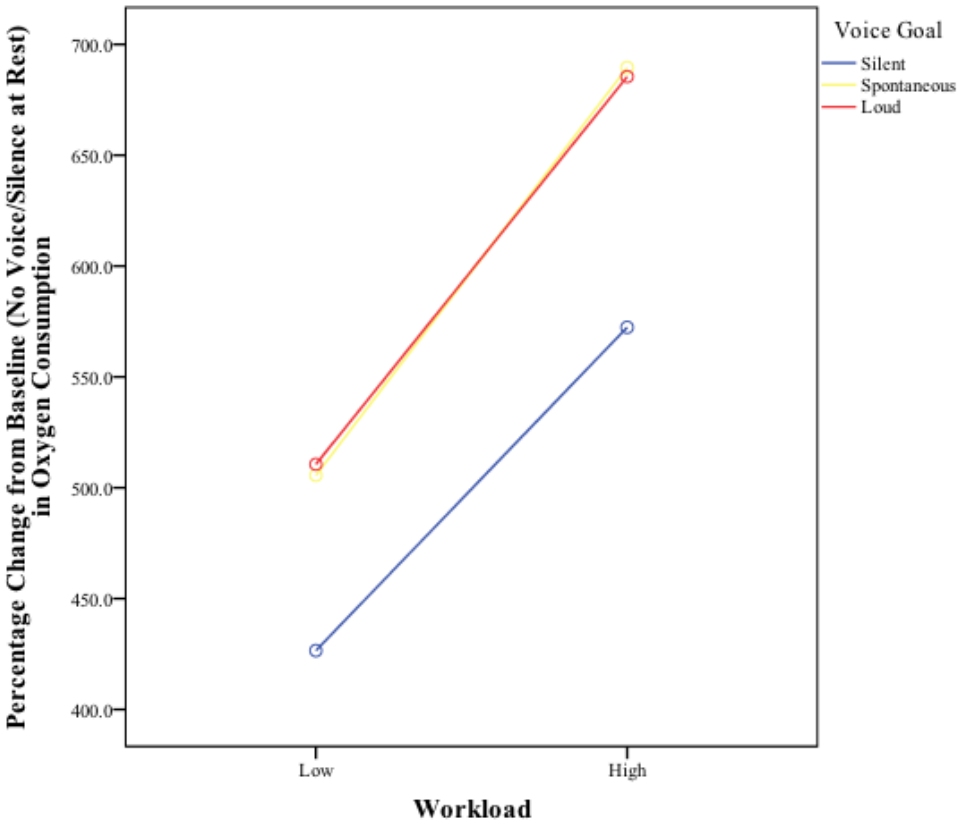

Figure 14. Comparisons of marginal means for percentage change from a baseline of no voice/silence at rest in oxygen consumption as a function of workload and voice goal.

Table 26. Test of Normality on Percentage Change in Oxygen Consumption as Function of Workload and Voice Goal

Condition	Shapiro-Wilk *W*	*df*	*p*
Silence/50%	0.975	32	.648
Silence/70%	0.974	32	.618
Spontaneous/50%	0.970	32	.488
Spontaneous/70%	0.975	32	.661
Loud/50%	0.930	32	.040
Loud/70%	0.965	32	.375

In order to find the pattern of differences on mean percentage change in VO_2 depending on voice goal between low and high workloads, simple main effect of voice goal was examined for each level of workload. The significant differences on mean percentage change in VO_2 among voice goal for each workload were followed by simple comparisons. In addition, simple main effect of workload was examined for each level of voice goal by a paired samples *t*-test since it has only two levels. The simple main effect of voice goal for low [$F(2, 62) = 36.034$, $p < .001$, partial $\eta^2 = .538$] and high workloads [$F(1.339, 41.513) = 63.269$, $p < .001$, partial $\eta^2 = .671$] was significant. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly smaller mean percentage change in VO_2 during silence in low and high workloads compared to spontaneous and loud voice at both workloads, $p < .001$, but there was no difference in mean percentage change in VO_2 between spontaneous and loud voice at both low and high workloads, *p*s = 1.000.

The simple main effect of workload for all three levels of voice goal was significant. Participants had a significantly greater mean percentage change in VO_2 in the high workload compared to low: silence [$t(31) = 8.297$, $p < .001$], spontaneous [$t(31) = 8.625$, $p < .001$], and loud [$t(31) = 7.606$, $p < .001$]. Due to a violation of normality during loud voice at the low workload, a non-parametric Related-samples Friedman's Test was completed. The Friedman's Test revealed the same findings as the repeated measures ANOVA.

3.6.2.1 Follow-up analysis for participants who met loud goal

For participants who satisfied SPL criteria in the loud condition for all workloads, a 3 × 2 repeated measures ANOVA was performed on mean percentage change in VO_2 as a function of voice goal (silent, spontaneous, loud) and workload (low, high). All assumptions of ANOVA were met. This ANOVA revealed a significant interaction effect of voice goal and workload

[$F(2, 12) = 6.412$, $p = .013$, partial $\eta^2 = .517$], meaning that the effect of workload on VO_2 depends on the level of voice goal. Additionally, this ANOVA revealed significant main effects of voice goal [$F(2, 12) = 16.019$, $p < .001$, partial $\eta^2 = .728$] and workload [$F(1, 6) = 7.828$, $p = .031$, partial $\eta^2 = .566$].

In order to find the pattern of differences on mean percentage change in VO_2 depending on voice goal between low and high workloads, simple main effect of voice goal was examined for each level of workload. The significant differences on mean percentage change in VO_2 among voice goal for each workload were followed by simple comparisons. In addition, simple main effect of workload was examined for each level of voice goal by a paired samples *t*-test since it has only two levels. The simple main effect of voice goal for low [$F(2, 12) = 8.543$, $p = .005$, partial $\eta^2 = .587$] and high workloads [$F(2, 12) = 27.946$, $p < .001$, partial $\eta^2 = .823$] was significant. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly smaller mean percentage change in VO_2 during silence in the high workload compared to spontaneous voice in the high workload, $p < .001$, but not during the low workload, $p = .279$. In addition, participants had a significantly smaller mean percentage change in VO_2 during silence in low and high workloads compared to loud voice at both workloads, $p = .018$ and $p = .009$. There was no difference in mean percentage change in VO_2 between spontaneous and loud voice at both low and high workloads, $p = .236$ and $p = 1.000$.

The simple main effect of workload for spontaneous and loud voice was significant. Participants had a significantly greater mean percentage change in VO_2 in the high workload compared to low at spontaneous [$t(6) = 3.231$, $p = .018$] and loud voice [$t(6) = 2.466$, $p = .049$]. The simple main effect of workload for silence was not significant [$t(6) = 2.372$, $p = .055$].

3.6.3 Carbon dioxide production (VCO_2)

Descriptive results of the actual VCO_2 values for both females and males are displayed in Tables 21 and 22. Results are displayed in Table X and Figure Y. For females, in silence, mean VCO_2 increased from 3.4 (0.4) mL/kg/min at rest to 20.4 (3.6) mL/kg/min and 28.1 (4.1) mL/kg/min at low and high workloads, respectively. In spontaneous voice, mean VCO_2 increased from 3.6 (0.7) mL/kg/min at rest to 23.7 (4.5) mL/kg/min and 32.6 (5.6) mL/kg/min at low and high workloads, respectively. In loud voice, mean VCO_2 increased from 3.9 (0.8) mL/kg/min at rest to 23.7 (4.7) mL/kg/min and 32.8 (5.6) mL/kg/min at low and high workloads, respectively. For males, in silence, mean VCO_2 increased from 3.7 (0.4) mL/kg/min at rest to 23.9 (5.3) mL/kg/min and 33.3 (2.5) mL/kg/min at low and high workloads, respectively. In spontaneous voice, mean VCO_2 increased from 3.6 (0.7) mL/kg/min at rest to 28.2 (5.4) mL/kg/min and 40.6 (3.6) mL/kg/min at low and high workloads, respectively. In loud voice, mean VCO_2 increased from 3.8 (0.7) mL/kg/min at rest to 27.9 (7.5) mL/kg/min and 38.6 (4.0) mL/kg/min at low and high workloads, respectively.

Table 27 and Figure 15 displays results for percentage change in VCO_2, for female subjects. A 3 × 2 repeated measures ANOVA was performed on mean percentage change in VCO_2 as a function of voice goal (silent, spontaneous, loud) and workload (low, high). All assumptions of ANOVA were met. This ANOVA revealed a significant interaction of voice goal and workload [$F(2, 62) = 3.598$, $p = .033$, partial $\eta^2 = .104$], meaning that the effect of workload on VCO_2 depends on the level of voice goal. Additionally, the ANOVA revealed significant main effects of voice goal [$F(2, 62) = 89.716$, $p < .001$, partial $\eta^2 = .743$] and workload [$F(1, 31) = 78.063$, $p < .001$, partial $\eta^2 = .716$].

Table 27. Group Means and Standard Deviations in Percentage Change in Carbon Dioxide Production as a Function of Workload and Voice Goal in Females who Met Vocal Intensity Goal Versus All Females

	Low Workload		High Workload	
	Met Goal	All	Met Goal	All
Vocal Goal	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Silence	530.1 (112.8)	515.3 (123.7)	683.4 (171.5)	740.3 (167.9)
Spontaneous	653.9 (162.9)	622.7 (142.3)	801.2 (206.5)	876.2 (209.4)
Loud	645.4 (123.4)	613.7 (143.5)	781.0 (191.4)	881.0 (208.0)

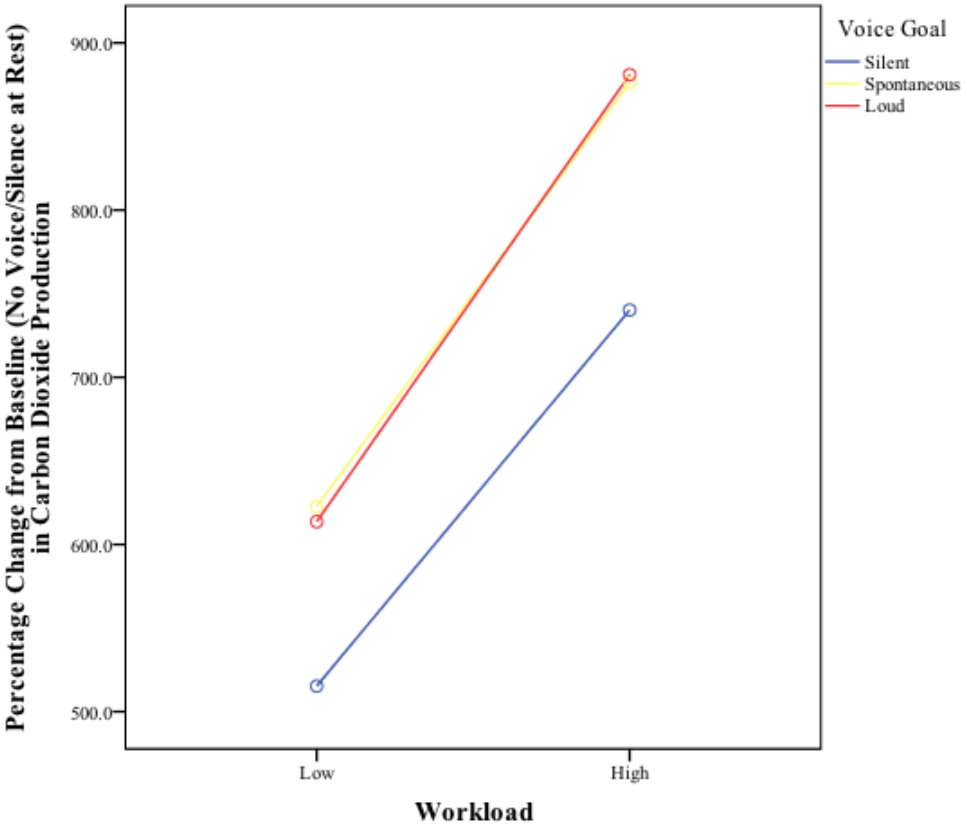

Figure 15. Comparisons of marginal means for percentage change from a baseline of no voice/silence at rest in carbon dioxide production as a function of workload and voice goal.

In order to find the pattern of differences on mean percentage change in VCO_2 depending on voice goal between low and high workloads, simple main effect of voice goal was examined for each level of workload. The significant differences on mean percentage change in VCO_2 among voice goal for each workload were followed by simple comparisons. In addition, simple main effect of workload was examined for each level of voice goal by a paired samples *t*-test since it has only two levels. The simple main effect of voice goal for low [$F(1.602, 49.648) = 33.870$, $p < .001$, partial $\eta^2 = .522$] and high workloads [$F(1.626, 50.414) = 93.208$, $p < .001$, partial $\eta^2 = .750$] was significant. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly smaller mean percentage change in VCO_2 during silence in low and high workloads compared to spontaneous and loud voice at both workloads, *p*s < .001, but there was no difference in mean percentage change in VCO_2 between spontaneous and loud voice at both low and high workloads, *p*s = 1.000.

The simple main effect for workload was significant at all levels of voice. Participants had a significantly greater mean percentage change in VCO_2 in the high workload compared to low: silence [$t(31) = 8.554$, $p < .001$], spontaneous [$t(31) = 8.792$, $p < .001$], and loud [$t(31) = 8.016$, $p < .001$].

3.6.3.1 Follow-up analysis for participants who met loud goal

For participants who satisfied SPL criteria in the loud condition for all workloads, a 3 × 2 repeated measures ANOVA was performed on mean percentage change in VCO_2 as a function of voice goal (silent, spontaneous, loud) and workload (low, high). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for only the voice goal by workload interaction effect [Mauchly's $W = .214$, $X^2(2) = 7.712$, $p = .021$]. All other assumptions of ANOVA were met. This ANOVA revealed significant main effects of voice goal [$F(2, 12) =$

11.319, p = .002, partial η^2 = .654] and workload [F(1, 6) = 8.767, p = .025, partial η^2 = .594]. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly smaller mean percentage change in VCO_2 during silence (M = 6.068, SE = 0.442) compared to spontaneous (M = 7.275, SE = 0.675) and loud voice (M = 7.132, SE = 0.547), p = .040 and p = .001, respectively, but there was no difference in mean percentage change in VCO_2 between spontaneous and loud voice, p = 1.000. In addition, participants had a significantly greater mean percentage change in VCO_2 in the high workload (M = 7.552, SE = 0.708) compared to low (M = 6.098, SE = 0.449), p = .025. The interaction of voice goal and workload was not significant [F(1.120, 6.718) = 0.120, p = .767, η^2 = .020].

3.6.4 Respiratory exchange ratio (RER)

Descriptive results of the actual RER values for both females and males are displayed in Tables 21 and 22. For females, in silence, mean RER increased from 0.75 (0.06) at rest to 0.86 (0.06) and 0.93 (0.05) at low and high workloads, respectively. In spontaneous voice, mean RER increased from 0.81 (0.14) at rest to 0.87 (0.11) and 0.93 (0.11) at low and high workloads, respectively. In loud voice, mean RER increased from 0.86 (0.15) at rest to 0.87 (0.12) and 0.94 (0.11) at low and high workloads, respectively. For males, in silence, mean RER increased from 0.81 (0.04) at rest to 0.92 (0.06) and 0.99 (0.04) at low and high workloads, respectively. In spontaneous voice, mean RER increased from 0.83 (0.09) at rest to 0.96 (0.12) and 1.08 (0.10) at low and high workloads, respectively. In loud voice, mean RER increased from 0.90 (0.10) at rest to 0.98 (0.06) and 1.04 (0.15) at low and high workloads, respectively.

Table 28 and Figure 16 displays results for percentage change in RER, for female subjects. A 3 × 2 repeated measures ANOVA was performed on mean percentage change in RER

as a function of voice goal (silent, spontaneous, loud) and workload (low, high). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for only main effect of voice [Mauchly's $W = .528$, $X^2(2) = 19.132$, $p < .001$]. All other assumptions of ANOVA were met. This ANOVA revealed only a significant main effect of workload [$F(1, 31) = 12.756$, $p = .001$, partial $\eta^2 = .292$]. *Post hoc* pairwise comparisons using a Bonferroni adjustment revealed participants had a significantly greater mean percentage change in RER in the high workload ($M = 0.247$, $SE = .024$) compared to low ($M = 0.165$, $SE = .023$). The main effect of voice goal was not significant [$F(1.359, 42.134) = 0.173$, $p = .756$, $\eta^2 = .006$], nor was the interaction of voice goal and workload [$F(2, 62) = 0.380$, $p = .685$, $\eta^2 = .012$].

Table 28. Group Means and Standard Deviations in Percentage Change in Respiratory Exchange Ratio as a Function of Workload and Voice Goal in Females who Met Vocal Intensity Goal Versus All Females

	Low Workload		High Workload	
	Met Goal	All	Met Goal	All
Vocal Goal	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Silence	17.5 (8.6)	16.2 (12.8)	22.6 (5.9)	24.5 (11.4)
Spontaneous	21.1 (6.4)	16.6 (13.9)	23.9 (12.9)	23.9 (16.4)
Loud	19.1 (9.1)	16.8 (16.6)	23.3 (10.8)	25.5 (16.2)

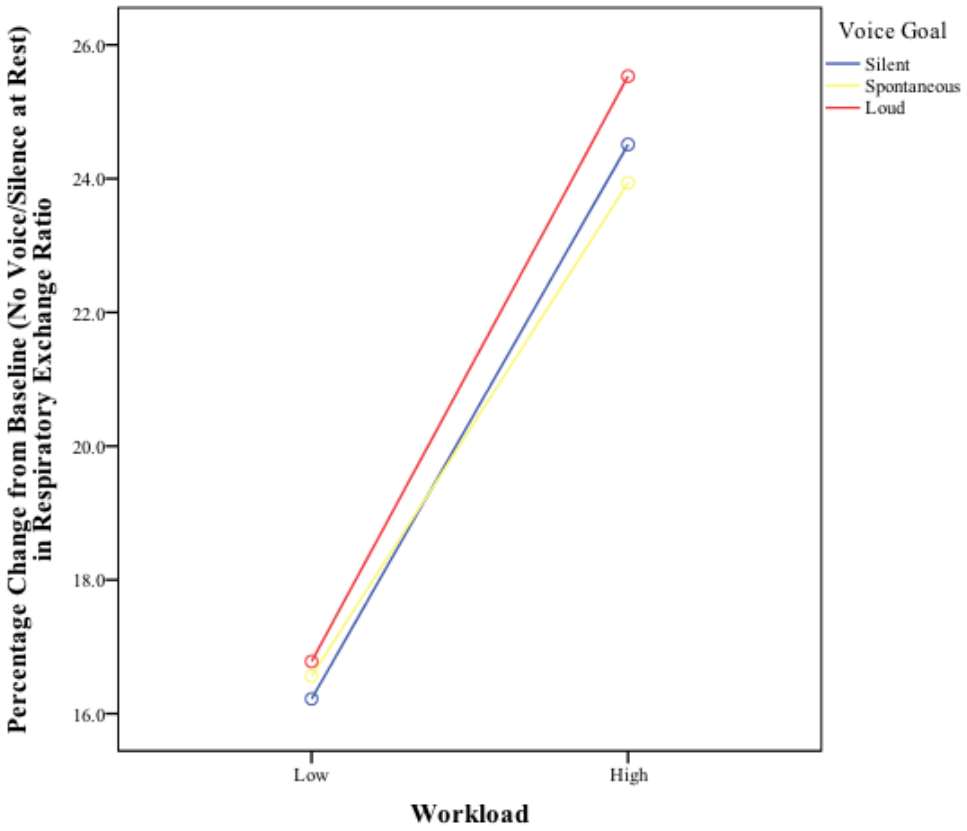

Figure 16. Comparisons of marginal means for percentage change from a baseline of no voice/silence at rest in respiratory exchange ratio as a function of workload and voice goal.

3.6.4.1 Follow-up analysis for participants who met loud goal

For participants who satisfied SPL criteria in the loud condition for all workloads, a 3 × 2 repeated measures ANOVA was performed on mean percentage change in RER as a function of voice goal (silent, spontaneous, loud) and workload (low, high). All assumptions of ANOVA were met. The main effects of voice goal [$F(2, 12) = 0.437$, $p = .656$, $\eta^2 = .068$] and workload [$F(1, 6) = 1.429$, $p = .277$, $\eta^2 = .192$] were not significant, nor was the interaction of voice goal and workload [$F(2, 12) = 0.266$, $p = .771$, $\eta^2 = .042$].

4.0 DISCUSSION

The purpose of this study was to investigate the dual, possibly competing functions of the larynx as airflow regulator and oscillating sound source. That is, the experiment set out to understand the effect of metabolic versus acoustic goals of the larynx to understand respiratory and phonatory functions in accomplishing simultaneous gas exchange and voice production. Hypotheses were, even under extreme respiratory perturbations induced by elevated levels of physical activity, when acoustic goals were specified, phonatory function would compromise respiratory function, and the respiratory-laryngeal system would do this in a way that implied potential disturbance to respiratory homeostasis. Specifically, the hypothesis was that phonatory functions, which in this study were measured by R_{law}, would stay constant across workload conditions when the goal was loud phonation, as predicted by action theory, ultimately limiting respiratory function and reducing gas exchange. Conversely, in extreme respiratory conditions, when acoustic goals were unconstrained, respiratory function would prevail over normal phonatory functions as needed to meet gas exchange goals, and the respiratory-laryngeal system would accomplish this in such a manner that sacrificed phonatory output. That is, the specific hypothesis was that respiratory function, as measured by Ve, would maintain relatively effective and efficient gas exchange across workload conditions when the goal was comfortable phonation, as predicted by action theory, eventually leading to deterioration in phonatory function and impairing communication effectiveness.

Discussion of results will be limited to those for the primary cohort, females. Overall, a majority of the results were congruent with study hypotheses, although some results existed that were inconsistent with study hypotheses. In general, and consistent with study hypotheses, the acoustic goal of loud voice produced R_{law} that was greater than spontaneous voice, regardless of workload, suggesting that loud voice can "win" even in the face of extreme respiratory perturbations. A finding for R_{law} that was partially inconsistent with study hypotheses was that accomplishing respiratory goals resulted in decreased R_{law} from low to high workloads. However, consistent with study hypotheses, loud voice maintained a deference toward phonatory goals even in the high workload. Like phonatory variables, results of respiratory variables were somewhat inconsistent with study hypotheses. Both spontaneous and loud voice interfered with Ve and other metabolic variables similarly. In spite of this somewhat inconsistent finding, spontaneous and loud voice altered respiratory variables differently than during silence, suggesting that phonation imposes a change in respiratory function that favors voice production. A more detailed discussion of results follows.

4.1 PHONATORY OUTCOMES

In line with study hypotheses, and as predicted by action theory, for the target participant group, percentage change in R_{law} increased for both loud and spontaneous voice from baseline, which was spontaneous voice at rest, and that increase was a significantly greater change from baseline for loud compared to spontaneous voice, regardless of workload. Furthermore, regardless of voice goal, percentage change in R_{law} resulted in a significantly larger increase from baseline in the low workload compared to high. Inconsistent with hypotheses, the study failed to

demonstrate a dependence of percentage change in R_{law} on the interaction of workload and voice goal. In other words, loud voice and spontaneous voice exhibited similar decreases in percentage change in R_{law} from low to high workload, although consistent with study hypotheses and a theory of action, loud voice maintained a higher percentage change from baseline compared to spontaneous voice at both workloads.

Only a fraction of participants (n = 4) failed to achieve their target SPL in the loud voice condition among workloads. For the subset of participants who did satisfy that task requirement, follow-up analysis revealed similar findings to those for the overall target group. As with the overall target group, the subset of participants also exhibited a larger increase in percentage change in R_{law} from baseline in loud voice compared to spontaneous voice, regardless of workload. In contrast to the overall target group, the subset of females did not exhibit a different percentage change in R_{law} from baseline between the low and high workload, regardless of voice goal. Interestingly, even despite achieving a target SPL, like the overall target group, analyses failed to find a dependence of percentage change in R_{law} from baseline on the interaction of workload and voice goal for the subset that consistently produced a loud voice. Despite those last two findings, the subset of females displayed similar patterns of percentage change in R_{law} from baseline to the overall target group, albeit the percentage change values were consistently lower in all conditions for the subset of females than for the overall group.

In terms of the component parts, for the overall target group, percentage change in P_s showed a significantly larger increase from baseline in the loud voice compared to spontaneous voice condition, irrespective of workload. No other finding was significant for P_s. The subset of female participants who consistently met their target SPL in the loud voice condition among workloads displayed the same pattern of results as the overall target group. Regarding U

(airflow), for the overall target group as well as the subset of female participants, no significant findings were detected, which is perplexing given different R_{law}. Although both spontaneous and loud voice conditions exhibited a numerical increase in percentage change in U from baseline as workload increased from low to high, further inspection of U percentage change values does suggest subtle differences between the two voice conditions. Spontaneous voice always yielded larger percentage change values than loud voice. Accordingly, under HRD conditions, loud voice appears to occur under lower U compared to spontaneous voice. Regarding workload, increasing respiratory drive led to a rather similar pattern of increases in U between spontaneous and loud voice from a low workload to high. This change in U impacted R_{law} somewhat similarly by causing it to decrease, although the high workload consistently resulted in greater R_{law} percentage change values than the low workload. Taken together, increases in percentage change in R_{law} from baseline appear to be driven by disproportionate increases in P_s relative to U. However, smaller increases in percentage change in U from baseline in loud voice did seem to result in larger increases in R_{law} compared to spontaneous voice. Unlike voice condition, percentage change values in R_{law} seem to decrease from low to high workload as a result of a disproportionate increase in U relative to P_s.

4.2 RESPIRATORY OUTCOMES

Consistent with study hypotheses and as predicted by action theory, for the overall target group, percentage change in Ve from baseline exhibited a significantly greater increase during breathing compared to the addition of voice in both the low and high workloads. However, contrary to study hypotheses and inconsistent with action theory, a change in voice goal from spontaneous to

loud did not appear to significantly impact Ve at either workload. In line with study expectations, an increase in workload from low to high significantly increased percentage change in Ve from baseline during silence, as well as in spontaneous and loud voice. A similar pattern of results emerged for the subset of female participants who consistently met their SPL target in the loud voice condition, except the increase in percentage change in Ve from baseline for spontaneous and loud voice goals did not reach significance. Only breathing silently demonstrated a significantly larger increase in the high workload compared to low. In spite of that finding, numerically, the high workload resulted in a larger increase in percentage change in Ve compared to the low workload in both spontaneous and loud voice.

Elevated levels of workload typically increase ventilation while increases in airway resistance from the addition of phonation generally decrease ventilation, and theses changes in ventilation are known to potentially impact gas exchange. Accordingly, consistent with study hypotheses and in agreement with action theory, for the overall target group, percentage change in $P_{et}CO_2$ from baseline increased significantly more with the addition of voice compared to breathing silently in both low and high workloads, although spontaneous and loud voice did not differ significantly. That is, decreased ventilation as a result of the airflow limitation from voice led to greater accumulation of $P_{et}CO_2$ compared to breathing silently with an unimpeded airstream. However, despite greater increases in percentage change in R_{law} from baseline in loud voice compared to spontaneous voice, $P_{et}CO_2$ remained unaffected by those differences, which is inconsistent with action theory. Consistent with study hypotheses, changes in workload impacted $P_{et}CO_2$ differently such that a high workload resulted in a significantly greater increase in percentage change in $P_{et}CO_2$ from baseline compared to low during silent breathing as well as in spontaneous and loud voice. The subset of female participants who consistently met the SPL

target in loud voice exhibited a similar pattern of results as the overall target group for percentage change in $P_{et}CO_2$ from baseline, with one exception being that during silent breathing $P_{et}CO_2$ did not differ significantly between low and high workloads. Although the change in $P_{et}CO_2$ during silent breathing from low to high workload was not significant, it did increase as workload intensified.

Elevated workloads require chemical reactions as a result of processes that support muscular contraction. More specifically, the body consumes more oxygen and produces more carbon dioxide to support an increase in workload (McArdle, Katch, & Katch, 2010). However, additional muscular involvement to phonate may also impact both VO_2 and VCO_2. The overall target group exhibited a significantly greater increase in percentage change in VO_2 from baseline with the addition of voice compared to breathing silently at both the low and high workloads, suggesting that the body consumed increased amounts of oxygen when phonating as opposed to breathing silently. However, loud voice did not differ significantly from spontaneous voice in percentage change in VO_2 at either workload. As expected, an increase in workload from low to high resulted in a significantly larger increase in percentage change in VO_2 during silent breathing, and in spontaneous and loud voice. Stated differently, the body consumed more oxygen at high compared to low workload, and this increase was consistent among voice conditions.

Regarding VCO_2, the overall target group demonstrated a significantly larger increase in percentage change in VCO_2 from baseline during voice compared to breathing at both the low and high workload, suggesting that additional muscular involvement from phonation resulted in increased VCO_2 compared to breathing silently. Contrary to expectations, spontaneous and loud voice did not affect percentage change in VCO_2 differently. As expected, increasing workload

from low to high led to significantly greater increases in VCO_2 during silent breathing as well as in spontaneous and loud voice. In other words, elevated workloads resulted in more carbon dioxide production as a result of an increase in aerobic metabolism to support more intense physical activity.

Regarding RER, the overall target group exhibited significantly greater increases in percentage change in RER from baseline in the high workload compared to the low workload. This finding suggests that at the high workload, as opposed to low, participants utilized a different proportion of energy substrates to support an increased intensity of physical activity. Accordingly, high workloads rely more on carbohydrates than low workloads, which access fats to supply energy (McArdle, Katch, & Katch, 2010). Unexpectedly, voice goal did not significantly impact RER, nor did the effect of workload on RER depend on the level of voice goal.

4.3 INTERPRETATION OF OUTCOMES AS PREDICTED BY ACTION THEORY

4.3.1 Phonatory outcomes

A theory of action motivated the overarching study hypotheses, which essentially said that motor behavior is goal-oriented and functionally specific. That is, the physiology of the respiratory-laryngeal coordinative structure depends on functional goals, even in spite of changing initial conditions. Furthermore, motor equivalence of a system allows for variability in movement patterns that may occur as a result of different initial conditions. Accordingly, the expectation that motivated this study was that phonatory function, as reflected by R_{law}, would favor acoustic

goals over metabolic needs when a voice target was specified, but would sacrifice phonatory output in voice conditions that were left unconstrained. In other words, the coupled respiratory-laryngeal systems would sacrifice phonatory output by decreasing R_{law} when the action to accomplish was respiration. Conversely, a loud acoustic goal that enhances the voice signal's discriminative power would negatively impact ventilation and gas exchange by increasing R_{law} under all workload conditions.

In fact, goal-oriented loud voice did result in a greater change in R_{law} from baseline compared to spontaneous voice that afforded participants to meet a loud acoustic goal, at least over a short duration. That is, the pulmonary-laryngeal coordinative system acted to achieve R_{law} that produced an elevated vocal intensity. This finding is consistent with research on R_{law} in conditions of normal drive, in which increases in vocal intensity cause R_{law} to increase (E. B. Holmberg et al., 1988; Leeper & Graves, 1984; Wilson & Leeper, 1992). Other volitional acts of breathing such as breath holding demonstrate similar goal-oriented control. The point at which individuals terminate a breath hold can be prolonged when distracted or motivated by a cognitive task, even when partial pressure of CO_2 rises to supposedly dangerous levels (Parkes, 2006). In this study, participants demonstrated a similar ability to override metabolic need in attainment of loud voice, at least for a short duration, when motivated to accomplish an explicitly stated acoustic goal. Interestingly, even in spontaneous voice, which was supposed to place speakers in a condition that allowed deference toward respiratory goals, participants somewhat maintained R_{law}. Upon further inspection, the increases from baseline in R_{law} in spontaneous voice seemed to occur due to changes in P_s that arose from increases in workload intensity. As individuals exercise, active expiration results in recruitment of abdominal muscles in addition to lung-thorax

elastic recoil forces to maximize ventilation. A consequence of that active expiration is an increase in expiratory pressure (Farrell et al., 2011).

Returning to loud voice, such increases in R_{law} can be explained in terms of respiratory and laryngeal kinematics. Relative to respiratory behavior, individuals initiate speech at increased lung volumes with increases in vocal intensity. Large lung volumes cause increases in alveolar pressures due to increased recoil forces and result in elevated subglottal pressures (Thomas J Hixon et al., 1973; T. J. Hixon et al., 1976). Speakers also increase TA muscle activation in loud voice (Finnegan et al., 2000). This contraction of the TA muscle can lead to a shortening and thickening of the TA muscle, thus acting as a vocal fold adductor as it bulges and potentially causing a decrease in airflow. However, producing voice at high lung volumes when maintaining has been shown to decrease vocal fold contact area and increase subglottal pressure and airflow rates (Iwarsson, Thomasson, & Sundberg, 1998). Given that R_{law} is the ratio of subglottal pressure to airflow, in the current study and to be discussed shortly, subglottal pressure increased disproportionately compared to airflow from spontaneous to loud voice, yielding higher R_{law} in loud voice compared to spontaneous.

This study also demonstrated that the increase in percentage change in R_{law} from baseline decreased as workload increased. In other words, when the goal was respiration, the pulmonary-laryngeal coordinative structure served gas exchange to meet somatic goals as evidenced by larger increases in percentage change in R_{law} from baseline at the high compared to low workload. Although this study is the first to investigate R_{law} changes as a function of whole body physical activity, Gillespie (2013) found that participant's R_{law} values in spontaneous voice did not differ significantly between eupnea and a hypercapnic condition induced chemically through inspiration of CO_2, which is known to increase respiratory drive, compared to normal drive. That

being said, Gillespie (2013) found that R_{law} in spontaneous voice did decrease for some participants. The author suggested R_{law} may be a control parameter for voice that individuals maintain at a relatively constant level, despite respiratory gas exchanges. Unlike Gillespie (2013), in the current study R_{law} decreased from low to high workload. This finding suggests that even in the face of increased subglottal pressures from baseline, the increase in airflow that occurred was enough to result in a decrease in R_{law} as a result of increased workload intensity. Differences in findings between the two studies may have to do with the ventilation requirements imposed on participants in the current study compared to those in the study by Gillespie (2013). On average, participants in the current study increased their minute ventilation by approximately 24 L/min in the spontaneous voice at the low workload over the baseline condition of silent breathing at rest and 35 L/min in the spontaneous voice at the high workload over the baseline condition of silent breathing at rest. In contrast, participants in Gillespie's study only increased their minute ventilation by 12 L/min from eupnea to hyperpnea. Airway dilation is a known consequence of increased respiratory drive during exercise, and certainly the larynx is a part of the body's physiologic response to accommodate an increase in ventilation (Farrell et al., 2011). The current study suggests that R_{law} is susceptible to changes in ventilation, although not to the point at which phonation is no longer possible nor do ventilatory requirements prevent individuals from varying their vocal intensity. In the current study, participants continued to maintain a greater increase in percentage change in R_{law} in loud voice compared to spontaneous speech, in relation to baseline, across workloads. In light of this difference in R_{law} between spontaneous and loud voice, phonatory goals seem important when specified, but left unconstrained, phonation favors metabolic need as evidenced by lower R_{law}.

Relative to respiratory and laryngeal kinematics, increases in respiratory drive alter laryngeal movements during breathing to accommodate increases in ventilation. Studies show that during breathing the vocal folds abduct more in high respiratory drive compared to normal drive, which leads to greater expiratory flow rates (England & Bartlett, 1982; England, Bartlett, & Knuth, 1982). However, as previously mentioned, initiation of speech occurs at higher lung volumes as respiratory drive increases from normal to high (Bailey & Hoit, 2002; Hoit et al., 2007), and large lung volumes decrease vocal fold contact area and increase subglottal pressure and airflow rates (Iwarsson et al., 1998). Again, considering that R_{law} is a ratio, the finding that it increased less during the high workload compared to low suggests that airflow rates increased relative to baseline, and presumably as vocal fold contact area decreased, to cause the percentage change in R_{law} to decrease from low to high. That difference occurred in spite of presumably increasing expiratory pressures as workload changed from low to high.

Taken together with the differences observed in R_{law} between spontaneous and loud voice, the respiratory-laryngeal coordinative structure tunes to accommodate ventilation but also strives to accomplish vocal goals. Research on neural mechanisms of breathing support a motor control organization in which two pathways exist for breathing, that is, a cortical pathway that serves volitional acts of breathing and exerts influence on the medullary respiratory center and a bulbar pathway for central pattern generation of breathing for life (Jürgens, 2009; Ludlow, 2005). The finding in this study on R_{law} provides new evidence that participants make attempts to meet phonation goals, at least over a short duration, even when metabolic need is high. This evidence is in line with the ability of human beings to overcome increasing metabolic pressures of other volitional acts of breathing such as breathholding, despite abnormally high partial carbon dioxide pressures that have the potential to induce syncope (Parkes, 2006), suggesting

that cortical processes exert influence on lower breathing centers. The degree of influence may depend on factors affecting decision-making such as rewards, costs, and risks (Cisek & Kalaska, 2010).

The contributions of the pulmonary and laryngeal systems to the coordinative structure regulating R_{law} underwent analysis to understand the actions of those subsystems in the context of acoustic and workload goals. The fact that R_{law} is a ratio may help explain the finding as individual components of subglottic pressure (P_s) and flow (U) may have changed in a way that maintained a relatively constant R_{law} in each voice condition. The only change observed in percentage change in Ps from baseline was a larger increase in loud voice compared to spontaneous, suggesting greater driving pressures in loud than spontaneous voice. Such changes in P_s are consistent with research on mechanisms of vocal intensity control in which the main contributor to increased vocal intensity is P_s (Finnegan et al., 2000; Stathopoulos & Sapienza, 1993); Titze & Sundberg, 1992). However, presumably the difference in percentage change in P_s between the due to voice condition is due to respiratory and laryngeal kinematics because both were produced in the theater of elevated expiratory pressures that would have increased P_s uniformly. Perhaps in the spontaneous voice, pulmonary elastic recoil forces alone mediated the increase in P_s by generating large expiratory pressures. However, to achieve an additional increase in P_s during loud voice, presumably the abdominal excursion increased to exert a force on the lungs or glottal adduction increased to allow for a greater build up of air molecules below the vocal folds, or a combination of both.

The expectation in this study for phonatory function was that an acoustic goal would impose a restriction on ventilation by exhibiting a tighter valve and result in a decrease in percentage change in U from baseline during loud compared to spontaneous voice production.

However, contrary to study hypotheses, U remained did not change significantly between voice conditions or workloads. Despite a failure to find any significant differences, percentage change in U from baseline was numerically consistently smaller in loud voice than spontaneous. Accordingly, participants appeared to create a greater constriction at the level of the glottis thereby limiting airflow in loud voice as compared to spontaneous voice. Furthermore, percentage change in U from baseline was consistently larger in high workload than low. Although not significant, this finding suggests that women responded to increased respiratory drive by loosening laryngeal valving to accommodate large minute ventilations. In light of modest accommodations in U as workload increased, these U results clearly suggest that phonation occurs within a narrow range of U values and corroborate research that finds voice production occurs within a narrow range of laryngeal valving to be able to maintain a relatively normal voice (Scherer, 2006). In other words, system constraints seem to dictate the extent of valving required for phonation. This finding suggests that phonation occurs under within a narrow range of R_{law}.

Accordingly, different pressure-flow profiles seem to exist for spontaneous voice compared to loud voice, even as the vocal intensity in spontaneous voice approached that of loud voice. R_{law} is higher in loud voice compared to spontaneous voice, and this difference was accomplished by reducing flow. Whereas pressure and flow both increased in spontaneous voice in a somewhat parallel fashion, flow increased less in loud voice compared to spontaneous voice. Gillespie and colleagues (2010) also observed different aerodynamic profiles in a cohort of individuals with muscle tension dysphonia. Taken together, P_s, rather than U, seems to be the principal driver of R_{law} in the context of extreme respiratory perturbations induced by exercise. That being said, U does exhibit a modicum of flexibility that results in smaller percentage change

from baseline in loud voice compared to spontaneous. Accordingly, U appears to contribute to the increased R_{law} in loud compared to spontaneous voice. In sum, the respiratory-laryngeal coordinative structure exhibits motor equivalence in the accomplishment of acoustic goals, and aerodynamic profiles of different types of phonation exist. In light of these findings, models of voice therapy might address systems involved in phonation rather than just component parts.

4.3.2 Respiratory outcomes

The second aim of the study was to investigate Ve as a measure of adequate and efficient gas exchange, to understand how the body achieves somatic goals in the face of voice production. During elevated intensities of whole body physical activity working muscles demand greater amounts of oxygen to support aerobic metabolism and the body strives to eliminate carbon dioxide from biochemical processes that create energy to support muscle contraction (McArdle, Katch & Katch, 2010). This study found increases in workload induced a systematic increase in percentage change in Ve from baseline during breathing as well as in spontaneous and loud voice, providing evidence that participants experienced a hyperpneic response due to increases in respiratory drive that resulted from a change in workload. The literature provides robust evidence for an exercise-induced response in Ve in the same direction as found in this study (McArdle, Katch, & Katch, 2010). Gillespie (2013) found that participants significantly increased Ve in response to an increase in respiratory drive from inspired CO_2. This finding reflects the response of peripheral chemoreceptors that react to changes in partial pressures of carbon dioxide to maintain respiratory homeostasis. However, unlike Gillespie (2013), the hyperpneic response observed in this study in which Ve increased most likely reflects several mechanisms, including

locomotor-linked stimuli, namely central command (feedforward) and muscle receptors (feedback) and to closely match the body's metabolic needs (feedback) (Farrell et al., 2011).

As expected, the addition of voice blunted this hyperpneic response at both the low and high workloads. Accordingly, participants exhibited smaller percentage change in Ve from baseline in spontaneous and loud voice conditions compared to quiet breathing at both workloads. In consideration of respiratory physiology, this finding corroborates the suggestion that upper airway resistance contributes substantially to limitations in ventilation (England, Bartlett, & Knuth, 1982). Numerous studies have also demonstrated this flow limitation provided by speech (Bailey & Hoit, 2002; Baker et al., 2008; Bunn & Mead, 1971; Doust & Patrick, 1981; Hale & Patrick, 1987; Hoit et al., 2007; Meckel et al., 2002; Otis & Clark, 1968). Those studies found that comfortable speech during exercise results in hypoventilation, which contrasts with the finding that comfortable speech at rest provides a slight hyperventilatory effect and loud speech at rest magnifies this hyperventilation (Bunn & Mead, 1971; Hoit & Lohmeier, 2000; Meanock & Nicholls, 1981); (Russell et al., 1998; Warner et al., 1983). Therefore, whereas comfortable and loud speech at rest causes individuals to exhale slightly more than necessary, the opposite is true of speech during exercise. That is, comfortable speech, and now loud speech, during exercise produces a substantial reduction in exhalation as required by the body to support aerobic metabolism.

However, the obstructive nature of the larynx during phonation did not appear to manifest differently when considering different vocal intensities. In contrast to a study of loud voice in normal drive (Russel et al., 1998), loud voice in HRD conditions did not result in a greater increase in percentage change in Ve than spontaneous voice. The lack of a significant difference in percentage change in Ve between voice conditions at the low and high workloads may result

from already elevated expiratory pressures due to the hyperpneic response as a consequence of increased respiratory drive. Perhaps, the hyperpneic response to exercise as ventilatory drive increased from rest produced large expiratory pressures to improve gas exchange and the system saturated in terms of how much greater expiratory pressures could rise. Most likely, loud voice relied on respiratory kinematics to generate an adequate subglottal pressure (Thomas J Hixon et al., 1973; T. J. Hixon et al., 1976), as expiratory pressure is the main control mechanism of vocal intensity (I.R. Titze, 2000). Furthermore, inspection of vocal intensity and subglottal pressure data corroborates this finding in which participants increased vocal intensity and subglottal pressure in spontaneous voice with increases in workload, suggesting that expiratory pressures between spontaneous and loud voice converge at higher workloads due to increased respiratory drive and the hyperpneic response to whole body physical activity.

Taken together, participants strive to meet acoustic goals for a short duration despite sacrificing somatic goals to some extent. Moreover, implicit somatic goals only impact acoustic goals, whether spontaneous or loud, when they substantially increase in strength (Baker et al, 2008; Meckel et al., 2002). That finding reflects a motor control of respiration that favors the ability of the individual to override ventilatory needs when prioritizing acoustic goals, even when somatic goals are strong. That is, when motivated to communicate, at least for a short duration, individuals can sacrifice metabolic needs to meet communication goals. The goals of the speaker ultimately seem to determine which function prevails.

Physical activity requires production of ATP, which occurs due to chemical reactions that produce carbon dioxide. Intense exercise results in greater amounts of carbon dioxide production than moderate exercise (McArdle et al., 2010). For that reason, the finding that participants exhibited a significantly larger increase in percentage change in $P_{et}CO_2$ from baseline in the high

workload compared to low is not surprising. Prior research shows that exercise results in an increase in $P_{et}CO_2$, and reflects the importance of lung perfusion and alveolar ventilation in an effort to eliminate CO_2 from the body to restore partial pressures of CO_2 and acid-base balance (McArdle et al., 2010). As $P_{et}CO_2$ reflects the carbon dioxide that diffused out of the blood stream and into the lungs for release into the atmosphere, larger amounts of carbon dioxide in exhaled air is expected and reflects normal respiratory function.

However, given that the function of the respiratory system is to eliminate carbon dioxide to restore gas partial pressures and maintain respiratory homeostasis, the flow limitation imposed by the larynx during speech could be problematic. In this study, participants exhibited a significantly greater increase in percentage change in $P_{et}CO_2$ from baseline during spontaneous and loud voice compared to breathing at low and high workloads. The increase in upper airway resistance with phonation, which restricts airflow and impedes efficient carbon dioxide elimination, resulted in a greater change in $P_{et}CO_2$ in both spontaneous and loud voice compared to quiet breathing at the low and high workloads. Research on the effects of speech on $P_{et}CO_2$ during exercise supports the finding that speech is obstructive and, consequently, participants demonstrate hypoventilation (Bailey & Hoit, 2002; Baker et al., 2008; Bunn & Mead, 1971; Doust & Patrick, 1981; Hale & Patrick, 1987; Hoit et al., 2007; Meckel et al., 2002; Otis & Clark, 1968).

Regarding the impact of different vocal intensities, percentage change in $P_{et}CO_2$ did not differ significantly between spontaneous and loud voice at the low and high workloads, suggesting that differences in vocal intensity does not have a differential effect on $P_{et}CO_2$. This finding suggests that neither vocal intensities provide a greater ventilatory limitation and hypoventilatory response that would allow for a difference in the buildup of $P_{et}CO_2$. That being

said, one study found that loud voice in normal drive results in less $P_{et}CO_2$ than comfortable speech, presumably due to the increased ventilation observed in loud speech (Russell et al., 1998). Perhaps, the difference between that study and the current one lies in the nature of the ventilatory response from speech. At rest, speech results in hyperventilation but during exercise speech results in hypoventilation. Another difference between the two studies may be the duration of the speech task. Perhaps, a longer speech task such as the 7- minute one in Russell and colleagues (1998) is required to see a difference between vocal intensities in $P_{et}CO_2$.

A change in workload from low to high caused a significant increase in percentage change values in VO_2 during breathing and spontaneous and loud voice. This finding replicates prior studies that demonstrate a robust cardiorespiratory response as workload intensity increases to facilitate delivery to and consumption of greater amounts of O_2 and support aerobic metabolism (McArdle et al., 2010). The addition of voice, as opposed to breathing, also caused a significant increase in percentage change in VO_2 at both low and high workloads. At least for this short voice task, the additional use of respiratory and laryngeal muscles for voice production, whether spontaneous or loud, appeared to substantially impact VO_2. The use of muscles for voice production, regardless of the vocal intensity, may have required additional VO_2 to support increased muscle contraction. This finding is somewhat in agreement with research that found increases in VO_2 during loud voice compared to quiet breathing at rest, but not for spontaneous voice (Russell et al., 1998). Perhaps, during elevated workloads in which VO_2 is already high, the addition of respiratory and laryngeal musculature to produce voice increases the work of breathing, but at rest comfortable speech is no more costly than breathing. Although intrinsic laryngeal muscles vary in the distribution of different muscle fiber types, those muscles recruited for voice production, the thyroarytenoid and interarytenoid, do contain some type I muscle fibers

that rely on aerobic metabolism (J. F. Hoh, 2005; Tellis, Rosen, Thekdi, & Sciote, 2004). At least in one study, increased oxygen consumption from a baseline of quiet breathing at rest occurred during a 1-minute loud voice task in which participants phonated on the vowel "ah" between 90 and 100 dB as measured by visible light spectroscopy (Tellis, Rosen, Carroll, Fierro, & Sciote, 2011). Therefore, the vocal folds seem to increase oxygen consumption even in a short voice task such as the one in the current study.

Despite differences in percentage change in VO_2 between quiet breathing and the addition of voice production, changes in vocal intensity do not result in differences in percentage change in VO_2. The lack of differences between spontaneous and loud voice at both low and high workloads is unexpected. Apparently, for a short duration, an increase in vocal intensity does not shift VO_2 in any systematic way. Again, this finding reveals that additional use of muscles to produce loud voice does not impact VO_2, at least in a short voice task. Rather, the use of voice and not the intensity of voice production drive an increase in VO_2, at least for a task of a short duration. As the work of breathing is already sufficiently high in elevated workloads, this finding suggests that an increase in vocal intensity does not further increase the need for oxygen consumption beyond what is needed for spontaneous voice. In addition, respiratory and laryngeal muscle recruitment benefits from the increased cardiorespiratory response as a result of an increase in respiratory drive from elevated workloads as blood flow increases in working respiratory muscles to support the additional cost of breathing for elevated workloads. Importantly, the vocal folds also contain a predominance of type II muscle fibers that do not rely on oxygen (J. F. Hoh, 2005; Tellis et al., 2004). Perhaps, an additional increase in vocal intensity from spontaneous to loud caused recruitment of type II muscle fibers rather than type I muscle fibers.

A change in workload from low to high resulted in consistently higher levels of VCO_2 and a statistically significant increase in percentage change in VCO_2 during quiet breathing as well as spontaneous and loud voice production. Carbon dioxide production, a consequence of anaerobic glycolysis, is known to increase with workload intensity. Research supports this finding that chemical reactions occurring as a result of anaerobic metabolism for the synthesis of ATP cause increased VCO_2 (McArdle et al., 2010). Participants also exhibited significantly larger percentage change in VCO_2 with phonation compared to quiet breathing at low and high workloads, but different vocal intensities did not induce changes in percentage change in VCO_2. This finding is somewhat consistent with results from prior research in which VCO_2 increased from quiet breathing in normal drive with loud voice (Russell et al., 1998). These findings suggest that the use of additional respiratory and laryngeal muscles associated with phonation increases chemical reactions to provide energy for muscle contraction and produces carbon dioxide. Muscle fibers within the intrinsic laryngeal muscles largely consist of type II fibers (J. F. Hoh, 2005; Tellis et al., 2004). The increase in VCO_2 with the use of voice is consistent with the large distribution of type II muscle fibers found in the larynx, which would depend on anaerobic glycolysis for ATP production. However, failure to find a difference between spontaneous and loud voice suggests that, at elevated workloads, potential recruitment of different muscles due to short durational changes in vocal intensity does not impact the body as it is already producing large amounts of carbon dioxide to support exercise.

Contrary to expectations, voice condition did not significantly affect RER at low and high workloads. The lack of a difference between voice conditions is perplexing. At least in this short speaking task, the use of additional muscles during voice production, whether spontaneous or loud, did not appear to impact biochemical processes in a way that disproportionately affected

the energy pathway that was utilized. Accordingly, limited use of additional muscles for voice production does not appear to negatively interfere with metabolic processes for physical activity. Furthermore, presumably participants use greater muscle activation for loud voice production compared to spontaneous voice production. Perhaps participants increased inspiratory checking forces during spontaneous voice production given the large alveolar pressures from increased respiratory drive and exploited elastic recoil forces during loud voice production. Such respiratory kinematics would explain the paradoxical finding. Despite the lack of significant findings in RER, numerically, values increased systematically from low to high workloads. This finding met expectations that participants would increase VCO_2 relative to VO_2 during elevated workloads, suggesting that participants increasingly utilized anaerobic glycolysis as workload intensity increased. That whole body physical activity in the high workload led to a change in RER compared to low workloads is not surprising. Given the short duration of exercise and phonation, changes in RER were probably only related to VCO_2 (McArdle et al., 2010). At least for a short task, the addition of phonation, even at the high workload, did not lead to greater VCO_2 than VO_2, which provides preliminary evidence that short periods of phonation at a high workload do not lead to peripheral fatigue.

5.0 CONCLUSIONS

This study set out to investigate test an action of theory in the investigation of the function of the respiratory-laryngeal coordinative structure under different respiratory and phonatory goals. Results revealed that experimentally manipulating acoustic and workload conditions leads to functional specificity in the physiology of activity of the respiratory-laryngeal coordinative structure. Specifically, individuals who focus on an acoustic goal such as loud voice demonstrate muscular coordination patterns that result in R_{law} differing from spontaneous voice, even despite increasing metabolic need. Accordingly, participants exhibited the ability to accomplish goal-directed behavior for loud and, surprisingly to some extent, spontaneous voice production, even in the face of extreme respiratory perturbations.

However, variability in R_{law} within loud voice production among workloads suggests the respiratory-laryngeal coordinative structure operates within an equivalent class of movement. Therefore, motor control of loud phonation appears to demonstrate tuning at the peripheral level by exhibiting different muscular coordination patterns in response to changing initial conditions. Furthermore, even though spontaneous voice resulted in numerically greater U than loud voice, it also exhibited tuning of the respiratory-laryngeal coordinative structure that maintained R_{law} at a level that permitted phonation. These results demonstrate greater flexibility in respiratory control to achieve volitional acts of breathing such as loud voice production and, to some extent spontaneous voice, than previously thought, even in spite of increasing somatic goals.

In relation to respiratory goals, the increased flow restriction that phonation places on airflow during loud voice production does appear to impact ventilation requirements and metabolic needs, but, for a short duration, acoustic goals may supersede respiratory goals. That being said, individuals tune the respiratory-laryngeal coordinative structure to meet acoustic goals yet, at the same time, that adaptation continues to accommodate somatic needs, especially when voice is left unconstrained such as occurs in spontaneous voice. Stated differently, spontaneous voice allows for less interference with somatic needs than loud voice production. Consequently, in spontaneous voice, ventilatory responses better serve metabolic needs when respiratory goals take precedence.

In sum, as predicted by action theory, goals matter. The physiology of phonation changes depending on the functional goal targeted. Even in spite of extreme initial respiratory conditions, motor equivalence permits attainment of functional goals by tuning of the respiratory-laryngeal coordinative structure. Accordingly, participants demonstrated an ability to accomplish phonatory goals during loud voice, at least for a short duration. Conversely, spontaneous voice, but not loud voice, demonstrated deference to metabolic goals as a result of an acute hyperpneic respiratory response during exercise. Taken together, as predicted by action theory, results indicated phonatory function favors metabolic needs during exercise when acoustic goals remain unspecified. However, purposeful loud phonation, a condition in which acoustic goals are specified, can override respiratory function, at least for a short duration. The implication of this study is that different acoustic goals change phonatory function in unique ways under conditions of HRD conditions. Accordingly, perturbation studies may reveal different findings in terms of voice physiology, and by extension, speech production, when individuals are tasked with different goals.

5.1 LIMITATIONS

Several limitations with this study deserve mention. First, heart rates, as opposed to oxygen consumption, determined target workloads. Although heart rates increase linearly in response to changes in workload as measured by oxygen consumption in healthy, recreationally active individuals, other factors influence heart rate and may have affected heart rate levels on the day of testing. Factors include stimulants such as caffeine or anxiety associated with wearing a restrictive facemask for a prolonged period of time, although the study required participants to refrain from consuming caffeine as determined by self-report and participants reported no limitations to participating in the study. However, diagnosis of an anxiety disorder was not part of the exclusion criteria. Percent VO_{2max} to establish workloads may have decreased variability in the degree to which individuals responded to the physiologic stressor.

Along the same line, participants used self-report to quantify extent of weekly physical activity as a proxy for determining physical fitness level. Self-report can be unreliable in determining the amount and intensity of exercise performed each week. Potentially, inaccurate reporting of weekly recreational activity could decrease the extent to which participants possessed similar physical fitness levels. Variability in physical fitness level may have lead to differences in the response to a physiologic stressor experienced by participants. Again, future studies should control for physical fitness level by having participants complete a submaximal exercise test to determine VO_{2max}.

Another limitation was that participants presented with difficulty matching and maintaining pitch in the loud condition despite demonstrating an ability to match pitch at the screening. Although research shows that vocal intensity, not fundamental frequency, exerts the greatest influence on laryngeal airway resistance, studies should control for fundamental

frequency by testing for an ability to match pitch in spontaneous as well as loud voice. A consistent fundamental frequency may have resulted in less variability in laryngeal airway resistance and its component parts. Also relevant to task requirements, and even though participants did produce two distinct loudness levels, some participants overshot their goal in the loud voice condition. These two factors, increased fundamental frequencies as well as excessive vocal intensity in the loud conditions, presumably involve additional recruitment of muscles and, as a result, may influence metabolic measures such as oxygen consumption and carbon dioxide production. These two variables reflect biochemical processes of muscular contraction and may have altered due to additional muscle activation. Related, the iolation of

In this study, the speaking task used was short in duration and did not allow for attainment of steady state of metabolic variables. Individuals generally reach steady state from changes in exercise intensity in 2-3 minutes. Accordingly, results of the metabolic variables should be interpreted cautiously. Future studies should require participants to sustain speaking long enough for metabolic variables to reach a new steady state.

Yet another limitation of this study was the lack of control for lung volume. Research shows that respiratory kinematics influence phonatory function, including laryngeal adduction (Iwarsson & Sundberg, 1998; Iwarsson et al., 1998). Future studies should measure respiratory kinematics simultaneously with phonatory measures. Similarly, individuals demonstrated different ventilatory responses to similar workloads. Minute ventilation, the product of tidal volume and breathing frequency, changes in part with the degree of vocal fold movement by increasing or decreasing upper airway resistance. Although this study set out to investigate the ventilatory response to a physiologic stressor, control of minute ventilation may have allowed for decreased variability in the laryngeal airway resistance data.

One final limitation deserves mention. Many participants exhibited pressure peaks that did not meet morphology requirements. Those unacceptable peaks were discarded. The loss of a considerable number of pressure peaks limited the number of participants who had complete data sets. A power analysis revealed that the study needed a sample size of 30 to achieve 80% statistical power for statistical analyses. The reduced number of analyzable data sets may have impacted the study's power. Possibly, the jarring that occurred while participants walked on the treadmill at an incline may have contributed to this loss of data. Despite this data loss, results were more or less as predicted, and it would be unlikely that the "non-predicted" data were preferentially lost. That being said, future studies may consider alternate types of physical activity with less pounding movements such as cycling or treading water. Additionally, aerodynamic equipment that allows for better observation of morphology should be considered.

5.2 FUTURE DIRECTIONS

Lacking from the literature are studies investigating respiratory-phonatory interaction in a way that specifies the interaction of the two subsystems as they navigate a changing environment that leads to concurrent neural processes representing possible motor actions. This current study provided a window into phonatory and respiratory function during simultaneous voice production and physical activity. For improved understanding, future studies should consist of a series of experiments that aim to understand how respiratory perturbations such as physical activity impact phonatory function, and in particular, how speakers accomplish targeted vocal production under those perturbed conditions. To contribute toward achieving this aim, researchers should complete experiments that incorporate acoustic, aerodynamic, kinematic,

electromyographic, perceptual, and metabolic measurements to understand the interactions between metabolic breathing and voice production. Specifically, experiments should require participants to engage in various levels and types of physical activity while performing a variety of speaking tasks. Parameters such as fundamental frequency, vocal intensity, vocal quality, among others, should be systematically varied to simulate a range of communication goals. Measurement of respiratory kinematics should occur to assess how voice goal and workload alter lung volumes, indirectly measured from chest wall movements. Along those lines, collecting lung volume data in addition to manipulating voice goal and workload will also provide an understanding of how they interact and affect phonatory and respiratory function. In addition, the effect of training should be investigated to gain an understanding of how vocal ability and physical fitness, possible risk or protective factors, impact the manner in which individuals phonate. Beyond those early research goals, future studies should investigate the effect of aging on the body and one's ability to accomplish targeted voice production, with particular interest in the response of the respiratory and laryngeal systems to aging.

In terms of experimental design, researchers should give special consideration to the type of physical activity performed or the kind of ergometer used to alter workload to avoid a potential loss of phonatory data from the speculated pounding of participants' feet on the treadmill as they walked on an incline. Along the lines of equipment considerations, researchers might use alternative aerodynamic equipment to the automatized phonatory aerodynamic system for capturing pressure and airflow in order to have increased control over data collection and inspecting signal morphology. Furthermore, studies should consider activities that represent the natural situations in which PAVUs must function such as teaching an aerobics class or while singing and dancing on a stage. In those natural contexts, an appreciation of the unconstrained

physiology required to accomplish action should emerge.

Studies should explore the effects of dehydration from increased ventilation and fluid loss or the impact of exercise-induced reflux on decreased laryngeal function and possible causes of dysphonia in PAVUs. Also, researchers should study the effects of peripheral/neuromuscular and central mechanisms of fatigue from elevated physical activity levels. Attentional factors from the split focus on dance and singing technique may also contribute to poor coordination and result in injury to the body or the voice. Studies should systematically manipulate the allocation of attentional resources to understand how two tasks performed simultaneously may impact motor control. Continuing with motor control, this study tested a theory of action for voice motor control. Future studies should continue to understand competing action systems and the impact of that competition on motor control processes. Specifically, putting PAVUs who engage in these particular situations and testing the physiology of activity would allow for investigation of functional specificity of the act. To that end, future studies should investigate tuning mechanisms of the respiratory-laryngeal coordinative structure by looking at immediate responses to the perturbation and adaptability of the coordinative structure to the perturbation over time. Collecting neurophysiological data during those activities will provide an understanding of the neural structures and pathways as well as the timing of neural processes involved in accomplishing action.

Regarding mechanical stress, in addition to continuing to investigate impact stress, other types of mechanical stress such as aerodynamic or shear stress should undergo study. Furthermore, Titze and colleagues (2003) suggested that viscoelastic properties of the tissue change from an accumulation of heat that dissipates over repetitive vibratory cycles and contribute to fatigue fracture. Not only the frequency but also the amplitude of vibration.

Accordingly, large amplitude vibration occurring with increases in respiratory drive and loud voice production alongside increases in body temperature from exercise may contribute to weakened molecular bonds of vocal fold tissue. Studies should seek to understand tissue fatigue tolerance and fatigue life. Along those lines, changes in blood flow from the redistribution to large muscle groups during elevated levels of physical activity may interfere with important biochemical processes that support muscular and wound healing events in the vocal folds and warrant investigation.

The knowledge gained from this line of research will guide the development of relevant evaluation and treatment approaches for voice users who experience pathologic and nonpathologic respiratory perturbations. The development and testing of models of voice therapy specific to individuals who experience pathologic and nonpathologic respiratory perturbation should occur. Future clinical management of these unique voice users with combined cardiorespiratory (aerobic) training, respiratory muscle strength training, and voice training.

APPENDIX A

STUDY FLYER

Get PAID to *Walk and Talk*!!!

Subjects Needed for University of Pittsburgh Study

Roughly 2.5 million individuals in the U.S. work in jobs that require simultaneous speech and physical activity. Examples include military drill instructors, aerobics instructors, and physical education teachers. Such individuals are at risk for voice problems. *This research study will characterize the effects of aerobic activity on voice and breathing.*

YOU SHOULD PARTICIPATE IF:

- You are a healthy individual between the ages of 18 and 35 years
- You do 90 to150 min of aerobic activity per week (cycle, run, swim, etc.)
- You are a non-singer with less than 1 year of singing training (high school choir is okay!)
- Do not have a history of voice problems or respiratory disorders
- Have not smoked within the last 5 years
- Do not use medications except for medications that regulate menstrual cycle

WHAT DOES THE STUDY INVOLVE:

- A screening visit
- 2 study visits over a 1-week period (total time ~1 hr 15 min)
- Walking on treadmill and talking
- Earn up to $40

If interested, contact pittvoicelab@gmail.com

Write "Exercise and Voice Study" in Subject Line

APPENDIX B

EMAIL SCRIPT FOR ONLINE PRE-SCREENING

Thank you for inquiring about the Exercise and Voice Study! We truly appreciate your interest. In this study, you will be asked to speak while walking on a treadmill. To determine if you qualify for the study you will need to fill out a short online screening survey. The screening survey gives you additional information about the study, and by answering the questions, allows us to determine if you are eligible for a face-to-face in-clinic screening. The screening survey should take no more than five minutes to complete. The following link will give you online access to the screening survey:

https://docs.google.com/spreadsheet/viewform?fromEmail=true&formkey=dEJaX1NvR0pNRDlsbnlCLUpyXzVwV2c6MQ

If you have any questions, please do not hesitate to contact us. Again, thank you for contacting us and we look forward to receiving your answers.

APPENDIX C

ONLINE PRE-SCREENING QUESTIONNAIRE

Thank you for your interest in the Exercise and Voice Study. Before progressing any further, please review some important details about the study.

Individuals who need to speak loudly during physical activity such as law enforcement officers and firefighters may be at risk for a voice problem. Therefore, we would like to learn more about the voice during exercise.

Study participation requires an in-clinic screening and 2 testing visits to occur within a week at the University of Pittsburgh. As part of the study will first undergo a screening that includes answering some questions about your health, having your voice box examined, taking a pregnancy test (females only), and having your hearing screened. During the same visit, you will also be asked to walk on a treadmill. Then, you will be asked to return on two separate occasions to perform aerobic exercise on a treadmill for roughly 20 minutes at two intensities. While walking on the treadmill, you will also be asked to speak at a comfortable and loud level.

You will be compensated $40 at the completion of your participation. A partial payment of $10 will be given if you complete the in-clinic screening but are deemed ineligible to participate in the remainder of the study.

We require some information to determine if you are eligible to participate in the study. Upon completion of this questionnaire, we will send you a response on whether or not you qualify for an in-clinic screening.

******Please press SUBMIT at the end of the survey to send us your answers.******

1. Name (Last, First)
2. Email address
3. Phone number (e.g., xxx-xxx-xxxx)
4. As of today, are you between the ages of 18 and 35?
5. Date of Birth (e.g., xx/xx/xxxx)
6. Age
7. Gender
8. Ethnic Category
9. Racial Category
10. On average, do you do between 3 and 5 days per week of aerobic activity (e.g., cycling, running, swimming, aerobic classes, etc.)?
11. On average, do you do between 90 and 150 minutes per week of aerobic activity (e.g., cycling, running, swimming, aerobic classes, etc.)?
12. Height (in feet and inches)
13. Weight (in pounds)
14. Have you smoked within the last five years?
15. Do you currently have a voice problem?

16. Have you ever had hoarseness lasting for greater than 2 weeks or hoarseness recurring more than 3 times per year?
17. Have you ever had voice training with the exception of high school choir (training is defined as greater than 1 year of formal voice lessons)?
18. Do you currently take any medications (with the exception of medications that regulate menstrual cycle)?
19. Do you have any known health conditions that would prohibit you from completing aerobic exercise?
20. VHI-10 Questionnaire (Rosen et al., 2004)

VHI-10 Instructions: These are statements that many people have used to describe their singing and the effects of their singing on their lives. Please circle the response that indicates how frequently you have had the same experience in the last 4 weeks.	0=Never 1=Almost Never 2=Sometimes 3=Almost Always 4=Always				
1. My voice makes it difficult for people to hear me.	0	1	2	3	4
2. People have difficulty understanding me in a noisy room.	0	1	2	3	4
3. My voice difficulties restrict personal and social life.	0	1	2	3	4
4. I feel left out of conversations because of my voice.	0	1	2	3	4
5. My voice problem causes me to lose income.	0	1	2	3	4
6. I feel as though I have to strain to produce voice.	0	1	2	3	4
7. The clarity of my voice is unpredictable.	0	1	2	3	4
8. My voice problem upsets me.	0	1	2	3	4
9. My voice makes me feel handicapped.	0	1	2	3	4
10. People ask, “What’s wrong with your voice?”	0	1	2	3	4

21. Have you ever received voice therapy or had vocal cord surgery?
22. Are you willing to persist in the entire 3-visis protocol, which will include baseline voice and exercise testing and two other sessions, over the course of approximately one week?

23. If eligible, would you be able to attend an in-clinic screening at the University of Pittsburgh Voice Center located at UPMC Mercy Hospital in downtown Pittsburgh?

    THANK YOU! Please remember to press SUBMIT.

    We will be in contact with you shortly to let you know if you have qualified for an in-clinic screening.

APPENDIX D

EMAIL SCRIPT FOR IN-CLINIC SCREENING

Thank you for participating in the *Exercise and Voice Study*. You are scheduled to complete your in-clinic screening on **DATE (xx/xx/xx) and TIME (xx:xx)** at the University of Pittsburgh Voice Center. The UPMC Voice Center is located at Mercy Hospital, 1400 Locust St. Building D, 2nd floor, room 2100 (take purple elevators up to the 2nd floor and Voice Center is in front of you as you exit those elevators). Parking will be validated (up to two hours), but is NOT SPONTANEOUS. The parking garage is across the street from the hospital. The hospital is also easily accessed by multiple Port Authority Bus lines. We have attached a participant information handout to help you prepare for the screening visit. If you are unable to make your appointment, or would like to re-schedule, please email pittvoicelab@gmail.com or call XXX-XXX-XXXX.

Please remember, you will be **compensated** for all of your time participating in the study, including this session. Thank you very much for your time. We are counting on you, and are very grateful for your efforts.

APPENDIX E

PARTICIPANT INFORMATION HANDOUT

Thank you for your interest in the *Exercise and Voice Study*. This handout is provided to prepare you for your participation in the study. You are scheduled to complete the first day of the *Exercise and Voice Study* on **DATE (xx/xx/xx) and TIME (xx:xx).** Please arrive 10 minutes before your scheduled time. If you are unable to make your appointment, or would like to re-schedule, please email pittvoicelab@gmail.com or call XXX-XXX-XXXX. Please remember, you will be **compensated** for all of your time participating in the study.

You will need to maintain *normal fluid and diet intake* throughout the period of participation in the study. You will also need to wear *comfortable clothing*.

For the study:

1. Wear comfortable, loose fitting clothing and athletic shoes consistent with exercise testing. Please do not wear any jewelry (including watches, necklaces, bracelets and earrings).
2. *Males*, make sure you are clean shaven.
3. *Females*, make sure you put your hair up in a ponytail.
4. Drink plenty of fluids over the 24-hour period preceding the submaximal treadmill test.
5. Eat a light meal, but don't eat within 2 hours of exercise testing.
6. Try not to consume energy supplements or use any medications (including over-the counter medications) within 48 hours of exercise testing.
7. Try to avoid caffeine for at least 3 hours before exercise testing.
8. Try to avoid alcohol for at least 24 hours before exercise testing.
9. Try to avoid strenuous exercise or physical activity other than your *regular* routine for 48 hours before exercise test.
10. Get an adequate amount of sleep, preferably 6 to 8 hours, the night before exercise testing.

Thank you very much for your time. We are counting on you, and are very grateful for your effort.

APPENDIX F

CONSENT FORM

CONSENT TO ACT AS A PARTICIPANT IN A RESEARCH STUDY

TITLE: A Study of Ventilatory Homeostasis and Phonatory Behavior During Aerobic Exercise

PRINCIPAL INVESTIGATOR: Aaron Ziegler, M.A.
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SOURCE OF SUPPORT: SHRS Research Development Funds, Dr. Katherine Verdolini Abbott, Department of Communication Science and Disorders.

Why is this research being done?

Estimates indicate roughly 2.5 million individuals in the U.S. are employed in jobs requiring simultaneous loud communication and physical activity. Examples include military drill instructors, police officers, aerobics instructors, physical education teachers, and musical theater performers. An impression emerges that such individuals are at risk for voice problems. The purpose of this study is to characterize the effects of aerobic activity on respiratory and laryngeal function.

Who is being asked to take part in this research study?
People invited into this study can be male or female, have to be between 18-35 years old, in good health, recreationally active, nonsmoker for the last five years, and have normal hearing. Because this study examines normal vocal function, people cannot have long-term voice problems (greater than 2 weeks, or recurring greater than 3 times per year). Furthermore, you must not be on any medications other than medications that regulate menstrual cycle. We will ask you questions regarding the medications you are taking. The research involves high intensity exercise so you must not have any diseases that preclude participation in exercise (cardiovascular, respiratory, metabolic, neurologic or orthopedic). You must not have a history of an eating disorder. In addition, if you are pregnant or were pregnant within the last six months you cannot take part in this study. Furthermore, you must be able to tolerate exercise, so only individuals who participate in structured exercise or sports activity of no less than 30 minutes per day on at least 3 days per week during the previous six months will be allowed to take part in the study. Also, people who may have an allergy to anesthetics, gag easily, have a small nasal passage or a deviated septum may not participate in this study. In addition to the in-clinic screening visit, you will need to be available for 2 study visits to occur within a 1-week time span. The total duration of all study visits and procedures is estimated to be 3 hours. A total of 36 individuals will be enrolled for the study.

What procedures will be performed for research purposes?
If you decide to take part in this research study, you will undergo the following procedures.

Screening Procedures
Procedures to determine if you are eligible to take part in a research study are called "screening procedures." For this research study, screening procedures include completing a demographic, medical, and speech-language survey. You will have a urine pregnancy test. You will have your height and weight measured. You will have your vitals taken including blood pressure, heart rate, and oxygen saturation. You will also have a hearing test from a speech-language pathologist. You will listen to different frequencies and indicate if they were audible. A voice-specialized speech-language pathologist will listen to your voice and determine if the sound of your voice is normal and you will fill out a questionnaire about your voice. You will have your respiratory function tested by a speech-language pathologist. A spirometer, a device that measures airflow, will be placed in the front of your mouth and you will breathe in and out forcefully. A speech-language pathologist will examine your nasal passage and throat with a rigid scope. This is a lighted optical instrument passed through the mouth and used to get a deep look inside the body and examine areas such as the throat. You can proceed to participate in the fuller study if you can tolerate the scope without an excessive gag response, without anesthesia. These screening procedures will take about 20 minutes of your time and will take place at the University of Pittsburgh Voice Center.

If you qualify to take part in this research study, you will undergo the following pre-experimental and experimental procedures at the University of Pittsburgh Voice Center and the University of Pittsburgh Physical Activity and Weight Management Research Center. The pre-experimental procedures will take about 40 minutes of your time.
Pre-experimental Procedures
1. Spontaneous pitch and loudness in speech will be obtained as you read while

wearing a head-worn microphone.

2. You will be trained in the experimental tasks. First, you will put a facemask up to your face with a plastic tube placed in your mouth, resting on top of the tongue. You will produce a string of seven consonant-vowel syllables (/pæ:pæ:pæ:pæ:pæ:pæ:pæ/) at your spontaneous pitch and loudness. Then, you will repeat the task with a loud voice. Training of this task will cease when you produce the syllable string at the targeted loudness and pitch levels on three consecutive trials. Next, you will read aloud with a loud voice. Training of this task will cease when you consistently achieve the loud voice over the reading of an entire passage.

3. You will complete an assessment of cardiorespiratory fitness. The test performed to assess cardiorespiratory fitness will be a submaximal treadmill test. Before the submaximal exercise test begins, shoeless height and weight will be obtained and resting heart rate and blood pressure will be gathered. The speed and grade of the treadmill will be changed in small increments at one-minute intervals until test termination. The test will be terminated at the point where you achieve 70-85% of your age-predicted maximal heart rate. The exercise can be stopped at any time. There may be some discomfort associated with the procedure. Real-time breath-by-breath expired gas volumes and concentrations will be assessed. Heart rate and blood pressure will be continuously monitored throughout the testing. After this procedure and before discharging home and scheduling of the next visit, you will complete a cool down/recovery period that consists of 2 to 3 minutes of walking at a speed of 2.0 to 3.0 mph at 0% grade followed by seated rest for 2 to 3 minutes to ensure safe return of physiologic processes to normal levels.

Experimental Procedures

Tasks: There are several tasks that will be completed during the two visits that will be separated by at least a 24-hour period within a 1-week period. The duration of the experimental procedures is about 2 hours total across the two visits. The experimental procedures will take place at the University of Pittsburgh Physical Activity and Weight Management Research Center. These tasks include:

a) Measuring Vocal Cord Vibration

We will place a pair of electrodes on the surface of your throat. While the electrodes are in place, you will be asked to produce a string of seven consonant-vowel syllables (/pæ:pæ:pæ:pæ:pæ:pæ:pæ/) at conversational pitch and at a comfortable and loud loudness at rest and while you are exercising at two intensities (50 and 70% of age-predicted maximal heart rate).

b) Measuring Air Pressure

We will measure airflow rate and air pressure by putting a small mask on your face and placing a small plastic tube in your mouth. While the mask and the tube is in place, you will be asked to produce a string of seven consonant-vowel syllables (/pæ:pæ:pæ:pæ:pæ:pæ:pæ/) at conversational pitch and at a comfortable and loud loudness at rest and while you are exercising at two intensities (50 and 70% of age-predicted maximal heart rate). Your voice will also be recorded simultaneously with a microphone attached to the equipment that measures airflow and air pressure.

c) Measuring Expired Gas Volumes and Concentrations

We will measure the expired gas volumes and concentrations as you exercise and speak. We will measure these parameters using a sensor that is connected to the small mask on your face. Expired gas volumes and concentrations will be measured while you read aloud at conversational pitch and at a comfortable and loud loudness at rest and while you are exercising at two intensities (50 and 70% of age-predicted maximal heart rate).

After these procedures and before discharging home, you will complete a cool down/recovery period that consists of 2 to 3 minutes of walking at a speed of 2.0 to 3.0 mph at 0% grade followed by seated rest for 2 to 3 minutes to ensure safe return of physiologic processes to normal levels.

What are the possible risks, side effects, and discomforts of this research study?

Rare (occurs in less than 1% of people – less than 1 out of 100 people): In this study, there is a rare chance you may have an adverse reaction to use of local anesthetics or numbing spray such as Cetacaine® (difficulty breathing, shock, and even death). There is also a rare chance you may have an adverse reaction to the aerobic exercise (myocardial infarction [heart attack], arrhythmia [problem with the rate or rhythm of the heartbeat], hemodynamic instability [abnormal blood pressure or inadequate amount of blood pumped out by the heart], orthopedic injury [injury to the bones or the associated muscles, joints, or ligaments], and even death). This is unlikely in a young, healthy, recreationally active individual. Your risk of an adverse reaction to the aerobic exercise will be minimized by obtaining clearance to participate in exercise from your primary care physician, monitoring heart rate and blood pressure during the procedures, and having a physician trained in exercise ECG interpretation evaluate the results of the exercise test. There is also a chance you will have a negative response to the pulmonary function test (shortness of breath and lightheadedness). A physician and emergency drugs and equipment will be readily available should you experience any adverse reactions from study procedures.

Common side effects (occurs in 10-25% of people – 10 to 25 out of 100 people): You may experience gagging from the use of the scope to view your throat. This side effect is usually mild in severity and will be reduced by giving a topical anesthetic (numbing spray) to your throat area and vocal folds. You will probably cough briefly when the spray is put into your throat. The spray's effect lasts for about 10-20 minutes, during which time you will be advised not to eat or drink. As with any clinical procedure, experimental or otherwise, there may be adverse events or side effects that are currently unknown and some of these unknown risks could be permanent, severe, or life threatening.

What are the possible benefits from taking part in this study?

There is no direct benefit to you for participation in the study. The scientific knowledge to be gained from this study may help investigators understand the interaction of the respiratory and phonatory systems and benefit people with vocal fold injury in the future.

If I agree to take part in this research study, will I be told of any new risks that may be found in the course of the study?

You will be promptly notified of any new information that develops during the conduct of this research study, which may cause you to change your mind about continuing to participate.

Will my insurance provider or I be charged for the costs of any procedures performed as part of this research study?
None of the procedures you receive during this research study will be billed to you or your health insurance. If you get a bill or believe your health insurance has been billed for something that is part of the study, notify a member of the research team or UPMC Patient Billing Services.

Will I be paid if I take part in this research study?
You will be paid $40 for completion of all components of the study. You will receive $10 as partial compensation for the screening visit in the event that you are deemed ineligible to participate in the remainder of the study. You will receive your payment after you complete the study at the final study visit or after the screening procedures. None of the services and/or procedures (measuring vocal cord vibration, measuring air pressure, and measuring gas volumes and concentrations) you receive during this research study will be billed to you or your health insurance. If you get a bill or believe your health insurance has been billed for something that is part of the study, notify a member of the research team or UPMC Patient Billing Services.

Who will pay if I am injured as a result of taking part in this study?
University of Pittsburgh researchers and their associates who provide services at the UPMC recognize the importance of your voluntary participation in their research studies. These individuals and their staffs will make reasonable efforts to minimize, control, and treat any injuries that may arise as a result of this research. If you believe that you are injured as a result of the research procedures being performed, please contact immediately the Principal Investigator or one of the co-investigators listed on the first page of this form.

Emergency medical treatment for injuries solely and directly related to your participation in this research study will be provided to you by the hospitals of the UPMC. It is possible that the UPMC may bill your insurance provider for the costs of this emergency treatment, but none of these costs will be charged directly to you. If your research-related injury requires medical care beyond this emergency treatment, you will be responsible for the costs of this follow-up care unless otherwise specifically stated below. You will not receive any monetary payment for, or associated with, any injury that you suffer in relation to this research. There is no plan for monetary compensation. You do not, however, waive any legal rights by signing this form.

Who will know about my participation in this research study?
Any information about you obtained from this research will be kept as confidential (private) as possible. All records related to your involvement in this research study will be stored in a locked file cabinet. Your identity on these records will be indicated by a code rather than by your name, and the information linking these codes with your identity will be kept separate from the research records. You will not be identified by name in any publication of the research results unless you sign a separate consent form giving your permission (release). Note also that it is a University

policy that all research records must be maintained for at least 5 years following final reporting or publication of a project.

Will this research study involve the use or disclosure of my identifiable medical information?
No, this study does not involve the use or disclosure of identifiable medical information.

Is my participation in this research study voluntary?
Your participation in this research study is completely voluntary. Whether or not you provide your consent for participation in this research study will have no effect on your current or future relationship with the University of Pittsburgh. Whether or not you provide your consent for participation in this research study will have no effect on your current or future medical care at a UPMC hospital or affiliated health care provider or your current or future relationship with a health care insurance provider.

May I withdraw, at a future date, my consent for participation in this research study?
You may withdraw, at any time, your consent for participation in this research study. To formally withdraw your consent for participation in this research study you should provide a written and dated notice of this decision to the principal investigator of this research study at the address listed on the first page of this form. Your decision to withdraw your consent for participation in this research study will have no effect on your current or future relationship with the University of Pittsburgh. Your decision to withdraw your consent for participation in this research study will have no effect on your current or future medical care at a UPMC hospital or affiliated health care provider or your current or future relationship with a health care insurance provider.

If I agree to take part in this research study, can I be removed from the study without my consent?
It is possible that you may be removed from the research study by the researchers if they feel it is not in your best interest or it has become vocally or otherwise unsafe for you to continue.

****

VOLUNTARY CONSENT

All of the above has been explained to me and all of my current questions have been answered. I understand that I may always request that my questions, concerns or complaints be addressed by a listed investigator. I understand that I may contact the Human Subjects Protection Advocate of the IRB Office, University of Pittsburgh (1-866-212-2668) to discuss problems, concerns, and questions; obtain information; offer input; or discuss situations in the event that the research team is unavailable

By signing this form, I agree to participate in this research study. A copy of this consent form will be given to me.

_____ _____

Participant's Signature Date

CERTIFICATION of INFORMED CONSENT

I certify that I have explained the nature and purpose of this research study to the above-named individual(s), and I have discussed the potential benefits and possible risks of study participation. Any questions the individual(s) have about this study have been answered, and we will always be available to address future questions, concerns or complaints as they arise. I further certify that no research component of this protocol was begun until after this consent form was signed

_____	_____
Printed Name of Person Obtaining Consent	Role in Research Study
_____	_____
Signature of Person Obtaining Consent	Date

APPENDIX G

IN-CLINIC SCREENING FORM

Subject Number: _____ Start time: End time:

	Yes	No	
1. Sex			Male Female
2. Age 18-35?			Age: DOB:
3. English speaker?			
4. Recreationally active? If yes, how often? (90-150 min)			Min/wk: Days/wk:
5. Contraindications:			
• Cardiovascular disease? (cardiac, periphery artery, CVA)			
• Neurologic disease? (MG, MS, ALS)			
• Pulmonary disease? (COPD, asthma, interstitial lung)			
• Metabolic disease? (DM 1 or 2, thyroid, renal/liver)			
• Orthopedic complications (arthritis, heal spurs)			
• Eating disorder? (anorexia or bulimia)			
6. Currently pregnant or pregnant within last 6 months?			Pregnancy Test Result + / -
7. Regular monthly menstruation? (none if birth control)			
8. Non-smoker for past 5 years?			
9. Voice problems? (dysphonia > 2 wks or > 3 times/year)			VHI-10 score:
10. History of voice therapy or vocal fold surgery?			

11. History of voice training (choir ok, > 1yr formal)?
12. Medications? (except for menstrual cycle)
13. Allergic response to anesthesia?
14. Signs/symptoms of active allergies, URI, or other illness on day of participation?
15. Normal voice? (<10 on overall severity of CAPE-V)
16. Normal larynx?
17. Normal hearing? (30 dB @ 250, 500 Hz, 1, 2, 4, 8 kHz in better ear)
18. Normal BMI? (will accept 17.5 - 25.9 kg/m2) BMI:
19. Normal blood pressure and heart rate? (systolic <120 mm Hg; diastolic < 80 mm Hg; HR < 80)
20. Normal pulmonary function? (80% of age-predicted)
21. Match pitch (men: C3/female: A3, 3 secs [+/- 1 ST])?

CAPE-V score

Left:

Right:

Height: Weight:

Systolic: Heart Rate:
Diastolic:

FVC: FEV_1:

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