THE EFFECTS OF STRESS REACTIVITY ON EXTRALARYNGEAL MUSCLE TENSION IN VOCALLY NORMAL PARTICIPANTS AS A FUNCTION OF PERSONALITY

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The "theory of the dispositional bases of vocal nodules and functional dysphonia" (Roy & Bless, 2000) represents an important step toward the understanding of the relation between personality and voice disorders. However, experimental tests of this theory are widely lacking.

In this study, female healthy and vocally normal adults between the ages of 18-35 years were divided into two groups, introversion (n = 27) and extraversion (n = 27), based on results on the *Eysenck Personality Questionnaire- Revised* (*EPQ-R*). Both groups underwent a stress reactivity protocol, which involved a simulated public speaking stressor in addition to baseline speech, rest, and recovery phases.

Participants in the introversion group had significantly higher scores on voice handicap and depression than participants in the extraversion group. The introversion group exhibited significantly greater infrahyoid muscle activity (surface electromyography) and perceptions of vocal effort throughout the protocol. However, although greater stress reactivity was also descriptively observed for those measures in the introversion group, the phase by personality interaction was non-significant as were group differences for submental and anterior tibialis muscle activity. Voice fundamental frequency and intensity significantly decreased during stressor exposure for both groups. The stress induction was effective and significantly increased

systolic blood pressure (SBP) and negative affect in everyone. The degree of rumination was not different between groups, but the participants with introversion experienced significantly more fear of public speaking. Neither SBP, negative affect, nor fear of public speaking were significantly correlated with SEMG activity in the participant pool. Instead the trait introversion was significantly and positively correlated with infrahyoid muscle activity in the total sample.

This psychobiological study is first examination of the trait theory of voice disorders to focus on the role of stress reactivity. Results indicated that persons with introversion had a disposition towards increased infrahyoid extralaryngeal muscle activity combined with greater perceptions of vocal effort during speech, which were both magnified under conditions of psychological stress. Findings were tentatively interpreted to be consistent with behavioral inhibition in individuals high on introversion as predicted in the trait theory of voice disorders, which may constitute a risk factor for muscle tension dysphonia.

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PREFACE

My dissertation was a journey – figuratively and geographically. To many it may appear like a very lonely and winded path yet in essence it was a path that would not have been possible without continuous interactions. In retrospective, the supposedly winded path appeared less and less winded as the picture, the dissertation, became complete. I am happy and grateful for the opportunity to have worked on a project that was very fulfilling and close to my heart and this is only the beginning. This project would not have been possible without the unlimited support, and equal enthusiasm for this research endeavor, of my advisor Kittie Verdolini. My heartfelt thank-you goes to Kittie, who is an absolutely inspiring mentor and scientist, excellent thinker as well as an amazing human being and friend. Her boundless positive energy is truly contagious.

Summa summarum, this dissertation was a collaborative effort across several fields. I am indebted to my dissertation committee for their collective support and sharing of knowledge: Susan Shaiman and Scott Yaruss as well as Richard Jennings and Anna Marsland from the Department of Psychology. I am thanking Kevin Kim for statistical advice. Further, Tanja Schultz and Stan Szu-Chen Jou from the Language Technologies Institute and International Center for Advanced Communication Technologies at the School of Computer Science at Carnegie Mellon University and Universität Karlsruhe (TH) provided critical hard ware and logistic support. A thanks also goes to Rosemarie Cooper from the UPMC Center for Assistive

Technology. My colleagues at the University of Pittsburgh Voice Center -- Jackie Gartner-Schmidt, Clark Rosen, and Rita Hersan -- never let me lose sight of the clinical implications of my research. Moreover, interactions and discussions with colleagues outside of Pittsburgh shaped and honed my thinking with notable mention of Miriam van Mersbergen, Nelson Roy, and Julie Barkmeier-Kraemer. Last but not least, my new colleagues at the University of Kentucky deserve a big thank-you as they were available during the final period of my dissertation with constructive feedback while getting me started on my new journey as independent researcher: Joseph Stemple, Richard Andreatta, Rita Patel, Ashwini Joshi, and Anysia Ensslen.

I would like to thank my labmates past and present. Foremost, I am thanking Nicole Yee-Key Li and Chaya Nanjundeswaran for their friendship. They spent the most time with me throughout the PhD program and we grew together in many ways that are lasting. The original PhD group senior to me inspired me -- Ryan Branski, Elizabeth Urban Grillo, and Douglas Roth -- and the new group -- Aaron Ziegler and Amanda Gillespie -- is refreshing and surely will have a bright future. A separate special thanks goes to Jean Skeffington and Katherine Richardson. Further, this study would not have been possible at all without the help of the many students who assisted me with the practical execution of the experiments throughout the various stages and who were the ones who actually had to look the participants in the eye during the experimental stressor: Rebecca Bird, Caitlin Hughes, Nicole Li, Chaya Nanjundeswaran, Rina Patel, April Scott, Adrianna Shembel, Michelle Sokolsky, Shuba Sriram, and Katherine White.

I will not forget my time in Pittsburgh. I have made wonderful friends who are smart and kind. I will only mention a few: Sigrid Streit who is the one person who continuously and loyally accompanied me since the beginnings of my USA journey; my former roommate Min Cao who

always had an open ear; and finally, Rebecca Gilbert who brought my life full circle in Pittsburgh. The final and most important thank-you is reserved for my family. Words will never be able to express my love and appreciation for my parents. This journey could not have happened without them. They always believed in me and the power of education. They have laid the groundwork early on and provided patient and continued support throughout the years as did my brother Raimund and his wife Stefanie. I wish my nephew and niece, Max and Maya, a bright future in this world with less barriers and endless possibilities.

1.0 INTRODUCTION

"Why do only certain persons react to environmental stress and interpersonal conflict by developing an abnormal voice? Why is it that others who have emotional reactions to stress and conflict never have voice problems? Why is it that still others express their personal problems through some organ system other than the larynx?"(Aronson, 1990, p. 121)

For many people, psychological stress[1] appears nearly omnipresent in work and life, and may pose a direct or indirect health risk (Lovallo, 2005; National Institute for Occupational Safety and Health [NIOSH], 2004). Although stress certainly affects both genders it is striking that 90% of women report high levels of stress in their lives and that mostly female, White, and non-Hispanic workers, age 25-54 years, missed work in 2001 due to stress, anxiety, and/or neurotic disorders (National Institute for Occupational Safety and Health [NIOSH], 2004; National Institutes of Mental Health [NIMH], 2001; National Women's Health Resource Center, 2003). In addition to mental correlates of stress, also a large array of physical correlates has been reported, including those involving musculoskeletal disorders of interest for the present research (Lundberg, 2002). In sum, it is unquestioned that stress is a serious public health concern. However, despite numerous claims about its relation to voice disorders, to date few systematic data are available about the link between stress and voice problems (Verdolini, Rosen, & Branski, 2005).

In this document, voice disorder is broadly defined as a voice-related condition that affects an individual's daily *functioning* (Verdolini & Ramig, 2001). However, the term "pathology

[1] In the remainder of this document, stress will refer to *psychological* stress unless otherwise noted.

affecting voice" will be used as well in this document, because laryngeal muscle tension as a manifestation of stress may *acutely* affect voice production in many individuals without prompting them to consider themselves as having a voice disorder. Yet selected literature around patients with voice disorders implicates psychological factors such as personality and stress, as contributory to an array of *chronic* voice conditions. Of particular interest in the case of primary muscle tension dysphonia (MTD I) (Roy, 2003; Roy & Bless, 2000b). MTD I involves a dysphonia or other voice abnormality in the *absence* of known organic condition whereas secondary MTD (MTD II) indicates a dysphonia or voice abnormality in the *presence* of an underlying organic condition (Verdolini, et al., 2005). MTD I occurs primarily in women and accounts for 10-40% of clinical caseloads at a voice center (Roy, 2003). However, MTD more generally may be present in some form in nearly all patients with voice problems. In addition to stress's suggested role in the development of MTD I, stress may also play a critical role in compromising long-term treatment efficacy for MTD or other voice-related conditions (Roy, 2003). The proposal has been made that a convergent psychological-biological perspective around MTD would have value as an approach to understanding and treating MTD (Dietrich & Verdolini Abbott, 2008).

Chapter One of this document will provide a review of the literature pertinent to MTD and will highlight gaps in the literature. Chapter Two introduces a psychobiological framework for studying stress and its relation to voice disorders (Dietrich & Verdolini Abbott, 2008), and which lays the groundwork for the present research as well as future research. Thereafter, research questions and hypotheses will be outlined. Research methods will be discussed in Chapter Three and results will be presented in Chapter Four. The document will conclude with a Discussion in Chapter Five.

1.1 MUSCLE TENSION DYSPHONIA

Primary muscle tension dysphonia (MTD I) is the condition affecting voice that most often has been linked to certain personality traits as well as to stress (Roy, 2003; Seifert & Kollbrunner, 2005). As noted, MTD I generally refers to a condition in the *absence* of known organic pathology and contrasts with MTD II, which co-occurs with an existing organic condition, often in response to it (Verdolini, et al., 2005). The lack of organic findings for MTD I has kindled speculations about the condition's underlying etiology (Roy, 2003). As a result, MTD I has assumed many different names depending on one's bias about its origins. At the broadest levels, those biases can be subdivided into proposals around (1) a hyperfunctional, hyperkinetic, or muscle "misuse" origin, or (2) a "functional," psychogenic, conversion, or hysterical origin (Altman, Atkinson, & Lazarus, 2005; Koufman & Blalock, 1982; Morrison & Rammage, 1993; Roy, 2003). The first bias focuses directly on the local phenomenon of muscle tension, and the second bias focuses on putative upstream psychological factors as the primary roots of MTD I. A debate is ongoing about whether these approaches characterize qualitatively different classes of dysphonia, as discussed in detail shortly (Aronson, Peterson, & Litin, 1966; Roy, 2003; Roy, McGrory, et al., 1997). Unfortunately, difficulties exist around the interpretation of various data related to MTD I due to disagreements about the definition of MTD as a pathology affecting voice and lack of reliability data. The subsequent background discussion ignores this problem in any explicit fashion, but it should be kept in mind as a potential further confound to already confusing data.

Recently, MTD became the preferred label for this class of unexplained dysphonias (Roy, 2003; Verdolini, et al., 2005). According to the *Classification Manual for Voice Disorders*

(*CMVD-1*) (Verdolini, et al., 2005), MTD I (*hyperfunctional voice disorder*, *hypoadducted hyperfunction*, etc.) is a "dysphonia in the absence of current organic vocal fold pathology, without obvious psychogenic or neurologic etiology, associated with excessive, atypical or abnormal laryngeal movements during phonation" (p.249). Close examination of this definition reveals that MTD I does not necessarily manifest as any single profile. The definition's umbrella term is hyperfunction, but it accommodates both hyper- and *hypoadducted* hyperfunction, which in fact are observed clinically (Hillman, Holmberg, Perkell, Walsh, & Vaughan, 1989). Consequently, the clinical presentation of MTD I may vary widely, according to the *CMVD-1*'s definition of it.

However, further examination of the *CMVD-1* definition is required. This manual is a consensus document, and thus presumably its definition of MTD I represents a broad consensus among clinicians and researchers. In fact, personal communication indicates that the consensus process around the definition of MTD I was fraught with challenge (Verdolini Abbott, personal communication, January 12, 2008), and finding a satisfactory definition continues to be difficult. Relevant background is as follows. *Hyper*adducted laryngeal muscle patterns are thought to be causally related to the development of vocal fold lesions via increased *impact stresses* between the vocal folds (Berry, et al., 2001; J. J. Jiang & Titze, 1994). In this scenario, the hyperfunctional type of MTD I—which does *not* initially present with organic lesions according to the definition-- would be a precursor to phonotrauma. This clinical course for MTD I has been envisioned in the *CMVD-1* as a complication, which brings up the question if MTD I is ever truly non-organic. This question is however beyond the scope of this study. By definition, once a lesion has been laid down, the same hyperadducted laryngeal behavior that caused the lesion would be considered MTD II rather than MTD I. However, an alternative form of MTD I, for

which particular psychological profiles have been suggested as discussed shortly, involves vocal fold *hypo*adduction, and phonogenic lesions typically do not ensue due to *decreased* inter-vocal fold impact stress (Morrison & Rammage, 1993). Instead, increased vocal fold stiffness and possibly co-contraction of laryngeal adductors and abductors may be observed, which obstructs easy voice production (Hillman, et al., 1989; Morrison & Rammage, 1993). This topic will be pursued in greater detail in a subsequent discussion of muscle tension in MTD I. An emphasis in this document will be that excessive, atypical, sustained, and widespread contractions in the intra- or extralaryngeal muscles are thought to prevail in hypoadducted MTD I without causing mucosal changes in the vocal folds. In the present research, the interest is hypoadducted MTD I as a pathology affecting voice, and "MTD I" will further refer to this form of the condition unless otherwise indicated.

A word of caution is indicated regarding the role of psychological factors in MTD I. The *CMVD-1* emphasizes that no assumptions should be made about psychological involvement in MTD I or other conditions in the absence of specific, relevant observations. In other words, the diagnosis of a "psychogenic" voice disorder is not a diagnosis of exclusion. It should be used when specific *Statistical Manual of Mental Disorders-IV* classification criteria can be satisfied in conjunction with a voice disorder (e.g., conversion disorder, generalized anxiety disorder, etc.) (Verdolini, et al., 2005). It is also true that in some conditions affecting voice, psychological states may be present such as those linked to stress, which fall short of frank psychopathology but may nonetheless affect voice. In fact, psychological problems in the average patient with MTD I have been found to have more to do with anxieties that do not surpass the threshold for clinical diagnosis than with actual psychiatric problems (Aronson, et al., 1966; House & Andrews, 1987; A. House & H. B. Andrews, 1988; Seifert & Kollbrunner, 2005).

In conclusion, a reasonable stance is that MTD I should be considered a multifactorial voice disorder along a graded psychological and physiological continuum (Altman, et al., 2005; Bauer, 1991; Freidl, Friedrich, Egger, & Fitzek, 1993; Kinzl, Biebl, & Rauchegger, 1988; Roy & Bless, 2000b; Roy, Bless, Heisey, & Ford, 1997a; Roy & Leeper, 1993b; Seifert & Kollbrunner, 2005; Verdolini, et al., 2005). Such a continuum may encompass extremes such as "psychogenic" *a*phonia on one end (i.e., loss of voice; highest degree of psychological etiology) and MTD I as a purely musculogenic phenomenon on the other end (e.g., post upper respiratory infection).

Turning to the question of etiology for MTD I more specifically, in the past, research has widely investigated personality traits as predisposing factors. Further, the role of stressful life events and the nature of muscle tension have been discussed. The next paragraphs will present a discussion of existing literature on the potential roles of personality, stress, muscle tension, and psychobiological processes in MTD I.

1.1.1 The theory of the dispositional bases of vocal nodules and functional dysphonia (MTD I)

Based on psychometric evidence, certain traits have been found to characterize at least a subset of patients with MTD I, hereafter simply called MTD. A general trend has been noted toward elevated levels of introversion and anxiety (in the wider sense including neuroticism and trait and social anxiety) (Roy, 2003; Roy & Bless, 2000a; Seifert & Kollbrunner, 2005). A detailed list of characteristics that have been associated with MTD, as extracted from studies that used psychometric measures, includes: trait anxiety (Baker, Ben-Tovim, Butcher, Esterman, &

McLaughlin, 2006; Freidl, Friedrich, & Egger, 1990; Goldman, Hargrave, Hillman, Holmberg, & Gress, 1996; House & Andrews, 1987; Roy, Bless, & Heisey, 2000b), neuroticism (Gerritsma, 1991; Roy, Bless, & Heisey, 2000a; Roy, et al., 2000b), introversion (Gerritsma, 1991; Roy, et al., 2000b), social anxiety (Gerritsma, 1991; Roy, McGrory, et al., 1997; van Mersbergen, Patrick, & Glaze, 2008), constraint (Roy, et al., 2000a; van Mersbergen, et al., 2008), stress reactivity (Roy, et al., 2000a; van Mersbergen, et al., 2008), "conflict over speaking out" (Baker, et al., 2006; A. O. House & H. B. Andrews, 1988), depression (Aronson, et al., 1966; Baker, et al., 2006; Kinzl, et al., 1988; Roy, et al., 2000b; Roy, McGrory, et al., 1997), and somatic complaints (Aronson, et al., 1966; Gerritsma, 1991; Kinzl, et al., 1988; Millar, Deary, Wilson, & MacKenzie, 1999; Roy, McGrory, et al., 1997). Direct comparisons of results across the studies are challenging because of methodological differences and shortcomings such as differing inclusion criteria, the lack of adequate control groups, and small study samples. Hence, a certain degree of variability exists in the results. However, the most comprehensive and cohesive research on links between personality and voice disorders has brought forward the "theory of the dispositional bases of vocal nodules and functional dysphonia," described in the next paragraphs (Roy & Bless, 2000b).

This theory represents a framework that allows for the generation of hypotheses around processes that may lead to the development of vocal fold lesions and MTD (Roy & Bless, 2000a, 2000b; Roy, et al., 2000a, 2000b; Roy, McGrory, et al., 1997). Research conducted within this framework is seminal in terms of theoretical foundation. In short, the theory maintains that personality predisposes individuals to respond with conditioned and thus predictable emotional, cognitive and *vocal* behavioral response patterns to trait-specific environmental cues (Roy & Bless, 2000b). The theory is adapted from Newman and colleagues' synthesis of Eysenck's

biological theory of personality and Gray's neuropsychological model of the nervous system (Eysenck, 1967; Eysenck & Eysenck, 1985; Gray, 1975, 1982, 1985, 1987; Newman & Wallace, 1993a, 1993b; Patterson & Newman, 1993; Wallace & Newman, 1991 all cited in Roy & Bless, 2000b). The proposition is that extraverts and introverts are sensitive to different stimuli that are thought to be linked to behavioral response biases, as discussed shortly. Those differences in behaviors may promote the occurrence of either vocal fold lesions or MTD. In line with Gray's (1987) model, a Behavioral Activation System (BAS), a Behavioral Inhibition System (BIS), and a Nonspecific Arousal System (NAS) were mapped onto extraversion, introversion, and neuroticism respectively (Figure 1-1).

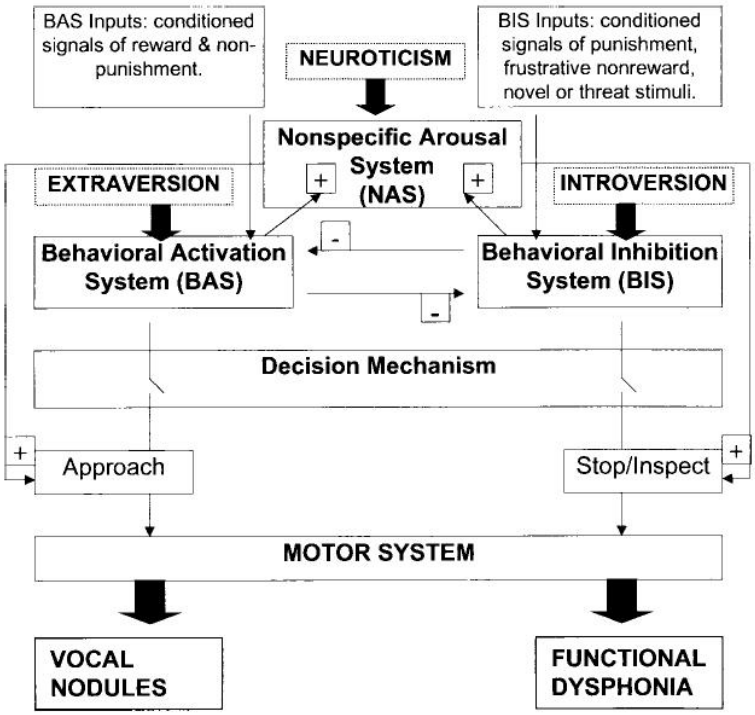


Figure 1-1. The theory of the dispositional bases of vocal nodules and functional dysphonia (in Roy & Bless, 2000b, p.474). From KENT/BALL. *Voice Quality Measurement*, 1E. © 2000 Delmar Learning, a part of Cengage Learning, Inc. All rights reserved. Reproduced by permission. Text/images may not be modified or reproduced in any way without prior written permission of the publisher. www.cengage.com/permissions.

The BAS is driven by conditioned signals of reward and non-punishment and promotes goal-directed *approach* behavior, escape, and active avoidance (response activation). In contrast, the BIS is driven by conditioned signals of punishment, frustrative non-reward, novelty or threat, and innate fear stimuli and promotes the *inhibition* of behavior, inspection of the environment, and passive avoidance (response suppression). The NAS represents neuroticism (personality trait reflecting low threshold for arousal and propensity to negative affect and worry) and becomes proportionally triggered with either activation of the BAS or BIS increasing general arousal and reinforcing approach or avoidance tendencies. The theory's attractiveness lies in its association of personality traits with neural correlates. Both the BAS and BISs are presumed to be related to neural structures in the septohippocampal system (septum, amygdala, hippocampus and fornix), which maintains connections to the prefrontal cortex. The NAS is thought to be linked to phasic autonomic changes. Thus, the theory provides a window into potential mechanisms that may link personality and pathologies affecting voice. Specifically, the implicit proposal is that extraverted persons may be at risk for vocal fold lesions, because their tendency towards behavioral and communicactive *activation* may bring forward vocal production patterns with high vocal fold impact stresses unopposed by inhibition. On the flip side, introverted persons may be at risk for MTD, because their tendency towards behavioral *inhibition* may interfere with the goal of vocal activation and promotes voice production patterns that are strained and effortful.

Within the voice domain, the trait theory of voice disorders was complemented by clinical research on a total of 70 participants with MTD using the *Minnesota Multiphasic Personality Inventory* (*MMPI*), the *Eysenck Personality Questionnaire* (*EPQ*), and the *Multidimensional Personality Questionnaire* (*MPQ* and *MPQ-Brief Form*). In addition, trait anxiety and depression were investigated in 45 of those participants. The research was further

strengthened by the incorporation of control groups of participants with other conditions affecting voice as well as vocally healthy participants (Roy & Bless, 2000a, 2000b; Roy, et al., 2000a, 2000b; Roy, McGrory, et al., 1997). The findings indicated that more patients with MTD were introverted (71%) and more patients with vocal fold lesions were extraverted (76%) as compared to controls (Roy, et al., 2000b). Further, 49% of patients with MTD could be characterized by both introversion and neuroticism (Roy, et al., 2000b). Overall, this series of studies revealed the following characteristics for patients with MTD in comparison to healthy controls (Roy, et al., 2000a, 2000b; Roy, McGrory, et al., 1997): increased hypochondriasis-depression-hysteria (neurotic triad), paranoia, psychasthenia, schizophrenia, introversion, trait anxiety, negative emotionality (in particular stress reactivity), and constraint.

Within the trait theory of voice disorders, individuals with MTD are seen to fall into the category of BIS-dominant neurotic introverts who are sensitive to threat, punishment, and novel situations, a constellation that in turn may induce anxiety and inhibited motor behavior (Roy & Bless, 2000b). Two scenarios for inhibited motor behavior, which may lead to increased laryngeal muscle tension, are proposed. First, an individual may exhibit selective internal and external hypervigilance for potential threats. Minor laryngeal *sensory* changes - from organic or emotional causes - may be registered as unusual and threatening. Such sensations may trigger *motor inhibition* or the *interruption of ongoing behavior* as controlled by the phylogenetically older septohippocampal system, which may temporarily override the phylogenetically newer neocortical control of vocalization and speech. Second, anticipation of punishment or non-reward (frustration) for *speaking out* may play a role.

At this point the relationships among personality (introversion/stress reactivity), behavioral inhibition, and muscle *tension* dysphonia may need to be clarified. In this document

tension refers to muscular contractile tension above resting tension unless otherwise indicated. Behavioral inhibition is not only suggestive of the suppression of behavior. Within the theory on the dispositional bases of functional dysphonia, laryngeal muscle *tension* may occur if (1) an active interest in vocal expression is opposed by laryngeal muscular inhibition (conflicting inputs), (2) or vocal behavior is ongoing and inhibitory tendencies interfere with voice production resulting in the interruption of ongoing behavior. Increased and widespread muscle tension is thought to be the result in both scenarios and may include the activation of antagonistic muscle groups (e.g., opposing laryngeal elevation versus depression; vocal fold adduction versus abduction).

Although the available psychometric data are promising, Roy and Bless (2000a) also acknowledged limitations. The cross-sectional nature of the studies prevented statements about cause and effect and about actual mechanisms involved in psychobiological factors' putative mediation of laryngeal behavior (Roy, et al., 2000b; Roy, McGrory, et al., 1997). Moreover, yet unexplored interactions among personality as risk factor, voice use patterns, and anatomical and physiological vulnerability may better reflect clinical reality (Roy, et al., 2000b). Nonetheless, the trait theory of voice disorders did lay the psychobiological foundation for future *experimental psychobiological* research that holds promise to aid in our fuller understanding of the links between personality and pathologies affecting voice (Dietrich & Verdolini Abbott, 2008).

1.1.2 Stress and MTD

The presence of stress and its potential role in MTD is a pervasive theme in the voice disorders literature (Dietrich & Verdolini Abbott, 2008; Dietrich, Verdolini Abbott, Gartner-Schmidt, &

Rosen, 2008). For example, evidence points to elevated life stress surrounding the onset of voice problems in persons with MTD or high levels of trait stress reactivity in that population as compared to populations with other conditions affecting voice or healthy voice (Aronson, et al., 1966; Baker, et al., 2006; Freidl, et al., 1990; Freidl, et al., 1993; Goldman, et al., 1996; House & Andrews, 1987; A. House & H. B. Andrews, 1988; Kinzl, et al., 1988; Morrison, Nichol, & Rammage, 1986; Morrison & Rammage, 1993; Roy, et al., 2000a; Roy, Bless, et al., 1997a; Roy & Leeper, 1993b; van Mersbergen, et al., 2008). Of note, current stress research carefully examines *perceived* stress and environmental stress in the light of *context* in order to explain individual responses to stressors (Cohen, Kessler, & Underwood Gordon, 1995; Endler & Kocovski, 2001).

One study investigated the frequency of *perceived* stress (Cohen & Williamson, 1988; Perceived Stress Scale, PSS-10), anxiety, and depression (Zigmond & Snaith, 1983; Hospital Anxiety and Depression Scale, HADS) in 160 new patients in a voice center with common pathologies affecting voice (Dietrich, et al., 2008). The pathologies under investigation were (1) MTD I, vocal fold lesions, and paradoxical vocal fold movement disorder (PVFMD), which have been variably linked to psychological factors, and (2) glottal insufficiency (e.g., vocal fold paralysis), where psychological factors should not play a causal role. Across all subjects, more females than males reported elevated stress, anxiety, and depression scores. The following results refer to gender-adjusted *z*-scores with a cut-off set at ≥ 1 *SD* above the mean[2]. Patients with PVFMD generally reported the highest *frequency* of perceived stress, anxiety, and depression and patients with glottal insufficiency the lowest frequencies for all scores except

[2] That is, "elevated" stress means that stress scores were one or more *SD*s above the gender-adjusted *z*-score mean.

depression (Figure 1-2). Patients with MTD and vocal fold lesions ranked similar on anxiety, stress and depression. However, although differences were small, patients with MTD scored marginally higher on scores for perceived stress than patients with vocal fold lesions.

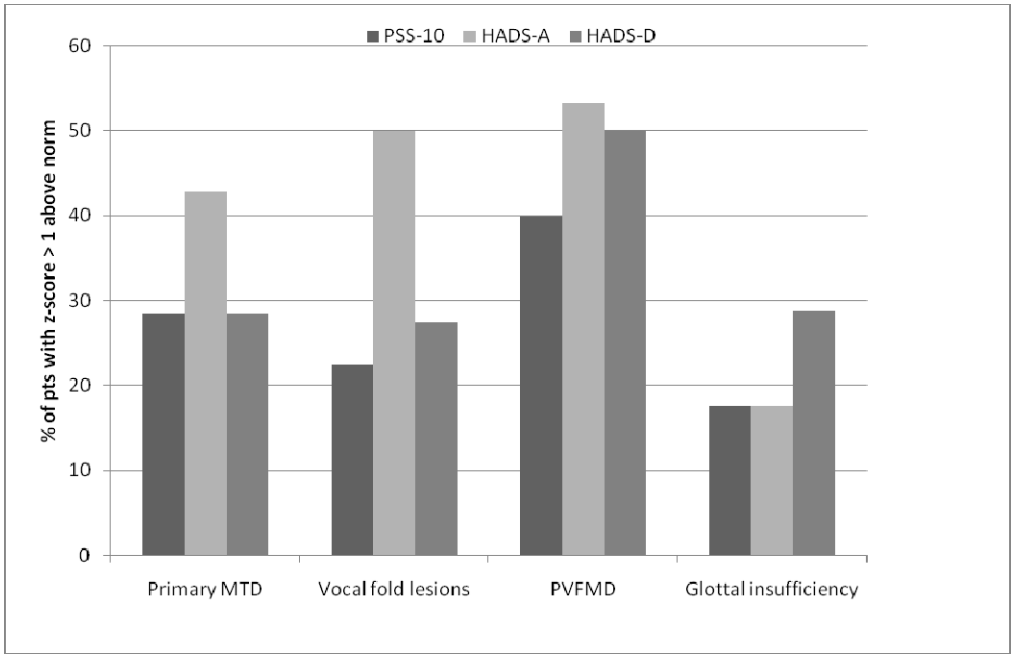


Figure 1-2. The distribution of stress, anxiety, and depression scores by pathology affecting voice (in Dietrich et al., 2008, p. 11). Reprinted from Journal of Voice, Volume 22 (4), Dietrich, M., Verdolini Abbott, K., Gartner-Schmidt, J., & Rosen, C.A., The Frequency of Perceived Stress, Anxiety, and Depression in Patients with Common Pathologies Affecting Voice, 472-488, Copyright (2008), with permission from Elsevier.

Unfortunately, the data do not in any way illuminate how perceived stress, anxiety, and depression may have influenced or will influence the course of various conditions affecting voice. That is, cause-effect relations remain unclear (Dietrich, et al., 2008). Again, similarly to the conclusion about the trait theory of voice disorders, *experimental* research will be crucial to tease out stress-related individual differences that may be relevant in MTD (Dietrich & Verdolini Abbott, 2008).

A different approach to the study of stress consisted of a focus on the *quality* of perceived stress using the *Life Events and Difficulties Schedule* (*LEDS*), a contextual measure that follows

an interview schedule (Brown & Harris, 1989). Patients with MTD indicated significantly more stressful life events and difficulties of at least moderate severity than participants with other conditions affecting voice or healthy controls (Baker, et al., 2006; A. O. House & H. B. Andrews, 1988). Moreover, the studies found that half of all patients with MTD reported stress in the form of a "conflict over speaking out" (COSO), a term that was coined by House and Andrews (1988). COSO represents (1) a situation in which a person is strongly involved and committed, and (2) a conflict in which a person feels the pressure to speak out, but yet refrains from doing so because of *anticipated punishment*. The notion of COSO in relation to MTD is in fact a recurring theme in the literature. In Aronson's (1990) words, for some patients with MTD the conflict consisted of a (1) breakdown in communication with someone important, and (2) a conflict for and against the expression of emotions, because of *fear* or shame. Last but not least, Roy and Bless's (2000b) theory on the dispositional basis *for MTD* suggested potential mechanisms involved in COSO. For example, elevated laryngeal muscle tension is predicted as the result of competition between older and newer neurological processes linked to emotion, vocalization, and speech, which compete for the inhibition or expression of emotion and speech respectively in a situation where, for example, anticipated punishment or fear plays a role. Overall, the notion of a conflict over speaking out is theoretically appealing relative to MTD, because it illustrates that emotional, cognitive, and behavioral conflicts may directly interfere with smooth vocal behaviors. Of note, partial support for the role of COSO in MTD comes from the domain of health psychology indicating that women typically feel more committed in interpersonal situations, which may be a source of stress as outlined above (Kiecolt-Glaser & Yehuda, 2005)—and MTD is indeed more common among women than men (Roy, 2003).

In conclusion, research on stress and MTD is thought-provoking. However, the questions outweigh the answers by far. Perhaps the most central question is why some individuals develop *muscle tension dysphonia* as a result of stress whereas others may not or respond to stress through some other organ system (Aronson, 1990). Researchers point out that while personality tendencies may exist in patients with MTD as previously summarized, personalities are generally complex and unique. Other factors must be considered, such as the nature and the degree of stress experienced, life experience, and support and coping strategies (Aronson, et al., 1966; Baker, et al., 2006; Freidl, et al., 1990; Kinzl, et al., 1988). Yet, common ground appears to be that most patients with MTD experienced their personal situation as *overtaxing* (Freidl, et al., 1990; A. O. House & H. B. Andrews, 1988; Kinzl, et al., 1988). Consequently, one assumption is that if stress surpasses a certain individual threshold, it produces symptoms at the place of least resistance (Adler, 1927; Kemper, 1954 cited in Kinzl et al., 1988). The larynx, an organ with special sensitivity to emotional influences, may be such a "locus minoris resistentiae" for some individuals (Aronson, 1990; Seifert & Kollbrunner, 2005). Aronson (1990) suggested that "laryngoresponders" may exist implying that each person has a unique way of reacting through a particular neuromuscular or visceral system, and for laryngoresponders, the place of least resistance may be the larynx. However, such notions have not been pursued with systematic *experimental psychobiological* research (Dietrich & Verdolini Abbott, 2008). In simple terms, stress has been thought to be associated with general tension states and such states may also increase or imbalance laryngeal muscle activity in some individuals (Aronson, 1990; Baker, et al., 2006; House & Andrews, 1987; Verdolini, et al., 2005). The next section reviews research on muscle tension in MTD.

1.1.3 Muscle tension and MTD

There is wide agreement that a range of excessive, dysregulated, or sustained intra- and extralaryngeal muscle activity is the proximal cause for MTD (Angsuwarangsee & Morrison, 2002; Aronson, 1990; Koufman & Blalock, 1982; Morrison, et al., 1986; Morrison & Rammage, 1993; Roy, 2003; Roy, Bless, Heisey, & Ford, 1997b; Roy & Leeper, 1993a). Palpable *extra*laryngeal muscular tension such as suprahyoid tension, laryngeal elevation, and decreased thyrohyoid space during rest and phonation has been described (Angsuwarangsee & Morrison, 2002; Aronson, 1990; Morrison, et al., 1986; Morrison & Rammage, 1993; Roy, Bless, et al., 1997b; Roy & Leeper, 1993a). Paradoxically, *objective* validation of muscle tension in this condition called *muscle tension* dysphonia has not been extensively pursued (Verdolini, et al., 2005). A handful of studies used laryngeal surface electromyography (SEMG) to measure *extra*laryngeal muscle tension. However, most studies focused on patients with phonotrauma or amassed patients with and without vocal fold lesions together as a single "hyperfunctional" voice disorders group (Andrews, Warner, & Stewart, 1986; Hočevar-Boltežar, Janko, & Žargi, 1998; Redenbaugh & Reich, 1989; Stemple, Weiler, Whitehead, & Komray, 1980). Further, most studies placed EMG electrodes only on the thyroid lamina or thyrohyoid space of the larynx, thus neglecting to test for co-contractions of laryngeal elevator versus depressor muscles during voice and speech production. In short, deviant laryngeal muscle *patterns* were not sufficiently explored.

As an exception, Hočevar-Boltežar et al. (1998) performed a study that sampled *both* suprahyoid and infrahyoid SEMG activity in patients with hyperfunctional voice disorders. However, MTD cases with and without vocal fold lesions were mixed. Manifestations of MTD

included posterior glottal chink, false vocal fold adduction, and anteroposterior contraction. During rest, patients with MTD and healthy controls did not differ in the magnitude of EMG signals. Just before the onset of sustained vowel phonation, SEMG activity increased in most patients and control participants, in particular in the muscles overlying the thyrohyoid space. However, in 6/11 patients, that muscle activity was higher than activity for other participants before and during voice production. Submental and infrahyoid muscle activity (below the cricoid cartilage) was similar across patients and control participants, but in 2/11 patients in comparison to controls infrahyoid muscle activity simultaneously increased with submental muscle activity during phonation. Overall, the study showed some evidence of higher laryngeal muscle *reactivity* in anticipation of as well as during phonation in patients with MTD as well as a tendency for extralaryngeal co-contractions in a subset of patients.

Overall, only limited objective data are available that quantify and compare intra- and extralaryngeal muscle activation in individuals with MTD in comparison to healthy controls during phonatory, speech, *and* non-speech tasks. The results from studies based on subjective methods of extralaryngeal muscle tension (palpation) or SEMG data are difficult to compare because of small and heterogeneous subject pools and methodological differences. Nonetheless, results generally show a trend towards greater extralaryngeal muscle activity during phonation in patients with MTD in comparison to controls as well as during the anticipation of phonation.

An implicit clinical assumption is that corresponding patterns of tension exist in the *intrinsic* laryngeal muscles. Objective methods for measuring intrinsic laryngeal muscle activity would involve needle EMG, but the physiological study of the intrinsic laryngeal muscles is challenging due to problems of accessibility and small muscle sizes (Ludlow, 2005). Instead, clinical attempts have been made to classify intralaryngeal muscle tension based on visual

inspection (Angsuwarangsee & Morrison, 2002; Morrison, et al., 1986; Morrison & Rammage, 1993). In the discussion that follows, the focus will be on intrinsic laryngeal muscle tension patterns that are typically not expected to result in mucosal vocal fold changes.

Patients with underlying psychological problems such as stress, anxiety and tension states were predominantly found to present with hypoadducted vocal folds or supraglottic squeezing (ventricular fold approximation) (Morrison, et al., 1986). In the case of hypoadduction, the vocal folds were abnormally held in an *ab*ducted posture just short of sufficient adduction for normal phonation. This laryngeal posture, possibly related to the co-contraction of laryngeal adductor and abductor muscles, may create a sensation of perceived tension during voice production (Colton, Casper, & Leonard, 2006; Hillman, et al., 1989; Morrison & Rammage, 1993). In fact, frequent related symptoms are the perception of *vocal effort*, laryngeal pain, and vocal fatigue (Morrison, et al., 1986; Morrison & Rammage, 1993; Roy & Bless, 2000b; Roy & Leeper, 1993b; Verdolini, et al., 2005). At the same time, vegetative phonation is typically normal (e.g., cough, laughter) (Colton, et al., 2006). Although extralaryngeal muscle tension is often described in association with MTD, palpated suprahyoid tension and laryngeal elevation were noted to be less frequent for the hypoadducted type of MTD I than for MTD II associated with vocal fold lesions. Supraglottic squeezing (lateral or anteroposterior) may be another presentation of MTD in the absence of organic changes, but clinical data indicate that the likelihood of developing vocal fold lesions is higher for this pattern than for the hypoadducted pattern (Morrison, et al., 1986).

Of particular interest for the present research is the proposal that inhibited or dyscoordinated laryngeal muscle activity may result in a loss of necessary laryngeal flexibility for phonation, or rigidity of the laryngeal framework (Morrison, et al., 1986). Many accounts of

patients with MTD have focused on suprahyoid tension. However, it has also been noted that the maintenance of a low laryngeal posture during voice production would demand more energy from a speaker than allowing the laryngeal mechanism to move freely (Shipp, Guinn, Sundberg, & Titze, 1987). Regardless of the exact pattern, widespread muscle tension may create a phonatory situation similar to "driving with the brakes on" (van Mersbergen, personal communication, October 21, 2005).

As a word of caution, there are no grounds to claim that any one constellation of MTD is exclusively related to stress or psychological factors. Individuals may present with a variety of behaviors and laryngeal tension patterns in response to stress. Partial support comes from neurolaryngology. The same learned speech-related vocal fold movement can be achieved by a number of combinations of laryngeal muscle activations (usually both intrinsic and extrinsic muscle activations), a concept often referred to as "motor equivalence" (Ludlow, 2005). Future research in laryngeal neurophysiology will be necessary to answer many outstanding questions regarding laryngeal muscle tension and vocal fatigue. In the next and final section on pathophysiological mechanisms in MTD, psychobiological evidence will be discussed that may illuminate the relation between stress and MTD.

1.1.4 Psychobiology and MTD

Although many researchers claim that stress is linked to MTD via the autonomic nervous system (ANS), there is a blatant lack of *experimental psychobiological* research to substantiate the claims (Butcher, Elias, & Raven, 1993; Demmink-Geertman & Dejonckere, 2002; Milutinović, 1991; Nichol, Morrison, & Rammage, 1993; Roy & Bless, 2000b; Roy, et al., 2000b). To date,

studies found that patients with MTD reported more psychosomatic complaints than healthy controls (Demmink-Geertman & Dejonckere, 2002; Freidl, et al., 1993; Goldman, et al., 1996; Kinzl, et al., 1988; Millar, et al., 1999). However, those studies were cross-sectional in nature. Further, a question is whether these patients were merely somatically hypersensitive and more prone to report symptoms or if the complaints were physiologically valid (Demmink-Geertman & Dejonckere, 2002).

Notably, recent work has made some forays into the exploration of psychobiological processes in MTD. A well-thought-out study was reported by van Mersbergen et al. (2008), in which participants with MTD (no organic changes) (n = 12) were compared to those with social anxiety (without voice problems) (n = 19), and healthy controls (n = 12). Participants were compared on parameters related to psychometric, cardiovascular, mood, vocal, and extralaryngeal SEMG measures in response to mood induction. No significant differences could be found between the MTD group and healthy group on any of the subscales of the *Multidimensional Personality Questionnaire – Brief Form* (Patrick, Curtin, & Tellegen, 2002). However, participants with MTD had numerically the highest score on the trait constraint as compared to the other groups and scored numerically between healthy participants (lowest) and participants with social anxiety (highest) on stress reactivity and introversion. In contrast, all groups differed significantly from each other on social anxiety. Healthy participants scored within normal limits, participants with MTD scored in the range considered consistent with a social anxiety disorder, and participants with social anxiety had a generalized social anxiety disorder, i.e. had the highest scores (Mennin, et al., 2002). Last, participants with social anxiety scored highest on state and trait anxiety and depression. Although having numerically higher scores, participants with MTD did not differ from controls on state and trait anxiety and

depression. In sum, participants with MTD were characterized by greater tendencies towards introversion, stress reactivity, constraint, trait anxiety, social anxiety, and depression in comparison to healthy controls yet social anxiety was the only statistically significant comparison.

Experimental procedures involved the exposure of participants to mental imagery with all combinations of aversive, positive, or neutral imagery and either nonverbal or verbal mental imagery. Autonomic arousal was assessed by heart rate (HR), and laryngeal responses were assessed by SEMG of the submental and thyrohyoid muscle sites. Participants with MTD had the lowest resting SEMG and participants with social anxiety had the highest. HR at baseline was comparable for participants with MTD and healthy controls, but higher for participants with social anxiety. Further, HR increased for all participants during positive and negative affective scripts; however, HR increases were lowest for MTD patients during aversive scripts and highest during positive scripts.

SEMG results for participants with MTD were counterintuitive and will be discussed shortly. Participants with MTD showed the lowest extralaryngeal SEMG activity during any affective and communication scripts as compared to the other groups. But ratings of *perceived vocal effort* that were elicited after reading a sentence after each scene were significantly higher for participants with MTD than for healthy controls, in particular for the aversive speech condition. Further, subjective ratings of arousal were significantly higher for the aversive, positive, and communicative conditions in the group of participants with MTD.

Findings for the social anxiety group were intriguing as well. Those participants did not have a self-reported voice disorder, yet their resting SEMG activity was highest for all muscle groups: HR and laryngeal SEMG activity increased the most during aversive, positive, and

communication scripts; and perceived vocal effort was higher than for healthy controls but lower than for participants with MTD. Hence, participants with social anxiety were physiologically (HR) *and* behaviorally (SEMG) more reactive than participants with MTD and even reported vocal complaints. In contrast, participants with MTD reported the highest vocal effort, but the lowest EMG activity.

The comparison of participants with MTD, social anxiety, and healthy controls is a worthwhile one. The results are at odds with current assumptions about patients with MTD. The data did show a clear *dissociation* between objectively reduced laryngeal EMG activity and subjective reports of increased vocal effort, despite engagement in the mental imageries as evidenced by increased HR and subjective increase in arousal. As a side note, the participants with MTD had previously received or were still receiving voice therapy at the time of the study. However, they were still symptomatic and many of them continued to complain about perceived laryngeal muscle tension, yet the same participants did not appear to have much awareness of laryngeal tension or lack thereof (van Mersbergen, personal communication, May 16, 2006). Thus, it is unclear if voice therapy did account for the reduced SEMG muscle tension.

In summary, the findings by van Mersbergen et al. (2008) are challenging to interpret. The author suggested that behavioral response *suppression* may have occurred in patients with MTD. On the other hand, it should be noted that EMG activity was not recorded *during* voice production but only during *mental imagery*, yet vocal effort was rated in reference to voice production. One consideration may be that imagery may not have been strong enough to differentiate the MTD group from the healthy group. Finally, the data may be spurious; that is, they may not represent findings for the population of individuals with MTD at large. Nonetheless, this study is a prime example for the importance of simultaneous recordings of

subjective and objective measures of laryngeal functioning. The findings are thought-provoking, but a more intensive test of emotions and their consequences on laryngeal behavior may be achieved with a stress reactivity protocol that would also focus on the collection of data for non-speech *and* speech tasks. Possibly, a clearer picture would emerge under exposure to a stressor.

Last but not least, research should explore the relation between personality and laryngeal behavior in the ostensibly *healthy, non treatment-seeking population*. It is striking that participants with a generalized social anxiety disorder exhibited extralaryngeal tension, reported vocal effort, and also scored remarkably high on the *Voice Handicap Index* (M = 53.80). It seems conceivable that *subclinical processes affecting voice* exist as a function of personality that are not registered as a voice disorder. Such thinking would be in line with Roy's theory of the dispositional bases of functional dysphonia (Roy & Bless, 2000a). In fact, participants with social anxiety had a tendency towards introversion and neuroticism, which is the combination of personality traits that would predict strong behavioral inhibition in response to threat. In comparison, patients with MTD were less introverted and neurotic than participants with social anxiety, which could at least partially account for the behavioral differences encountered. The final section of this chapter will summarize the gaps in the MTD literature.

1.2 GAPS IN THE LITERATURE

Past research investigating the relation between personality and MTD is interesting and important. The literature is replete with accounts of psychological *profiles* of patients with MTD that highlight introversion and anxious traits including heightened stress reactivity. However,

past research has been cross-sectional and thus, cause and effect relationships between personality and MTD remain speculative (Dietrich & Verdolini Abbott, 2008). Unexplored are in particular the *effects* of exposure to a stressor on laryngeal muscle activity and the specific *mechanisms* that may link introversion and perceived stress with MTD. Although researchers readily refer to the ANS as one potential mechanism, systematic data to support this claim are missing (Dietrich & Verdolini Abbott, 2008). Meanwhile, the "theory of the dispositional bases of vocal nodules and functional dysphonia" has provided valuable *theoretical* input (Roy & Bless, 2000b). According to this theory, introverts are particularly sensitive to threat and possess a predisposition to react with behavioral inhibition to it, which may also translate to laryngeal muscle tension. Unfortunately, this theory has not been *experimentally* tested. From a programmatic standpoint, it would seem valuable to examine laryngeal functioning while manipulating exposure to stress as a function of personality in *vocally healthy* participants first in order to learn more about normal laryngeal behavior under stress before investigating disordered laryngeal behavior under stress.

To summarize, four substantial gaps are noted in the current literature on MTD (Dietrich & Verdolini Abbott, 2008): (1) to date, although claims are made about the relevance of personality and psychological factors such as stress for MTD, research has failed to evaluate actual *laryngeal behavior* in response to presumed precipitators; (2) assuming that personality and psychological factors are relevant for MTD, research has similarly failed to adequately address the actual *causal* role of these variables; (3) although the *sympathetic nervous system* (SNS) has been widely implicated as a mechanism mediating relations between personality/stress reactivity and MTD, thus far data to this effect are lacking in the literature; and (4) theoretically motivated *intervention* studies have not been conducted around these issues.

In short, the literature is lacking *experimental psychobiological* research that could substantiate claims about relations among personality, stress reactivity and increased or dysregulated laryngeal muscle tension (Dietrich & Verdolini Abbott, 2008). We are still in the realm of speculation as to whether psychological processes may be considered causal, predisposing, exacerbating, maintaining, or consequential in MTD (Aronson, 1990; Dietrich & Verdolini Abbott, 2008; Freidl, et al., 1993; Morrison & Rammage, 1993; Roy, 2003; Roy, et al., 2000a, 2000b; Roy, Bless, et al., 1997b; Roy & Leeper, 1993a; Seifert & Kollbrunner, 2005). To complicate matters further, a vicious cycle of stress and voice problems may be common (Morrison, et al., 1986; Morrison & Rammage, 1993; Seifert & Kollbrunner, 2005) as conditions affecting voice are known to potentially affect a person's occupational, social, psychological, physical, and communicative functioning (Aronson, 1990; Krischke, et al., 2005; E. Smith, et al., 1996). Of note, stress can also influence bodily perceptions by increasing attention to physical states, which would explain somatic complaints in the absence of patent, verified disease (Costa & McCrae, 1985a; Pennebaker, 1983).

Research on mechanisms involved in MTD would be clinically significant. MTD is considered an etiologically unexplained dysphonia that is, however, often modifiable with behavioral voice therapy aimed at the reduction of laryngeal hyperfunction (Butcher, Elias, Raven, Yeatman, & Littlejohns, 1987; Elias, Raven, Butcher, & Littlejohns, 1989; Verdolini, et al., 2005). Nonetheless, despite optimism about the general short-term success of voice therapy for MTD, long-term benefits continue to be questionable, presumably because underlying psychological factors are not addressed (Gerritsma, 1991; Morrison & Rammage, 1993; Nichol, et al., 1993; Roy, 2003; Roy & Bless, 2000b; Roy, et al., 2000a, 2000b; Roy, Bless, et al., 1997b; Roy, McGrory, et al., 1997; Verdolini, Ramig, & Jacobson, 1998). In fact, remarkably consistent

across studies are characteristics of patients who were more prone to relapses: severe or chronic psychosocial stress, excessive anxiety, increased respect for social norms, conflict over speaking out, and persistent muscle tension post therapy (Butcher, et al., 1987; Günther, Mayr-Grafl, Miller, & Kinzl, 1996; Milutinović, 1991; Roy, Bless, et al., 1997b). Thus, stress appeared to be an overarching theme that impeded long-term treatment efficacy.

At a simple level, stress might interfere with voice therapy, because voice therapy requires compliance with exercises and clinical as well as medical advice (Anderson, Kiecolt-Glaser, & Glaser, 1994; Verdolini, et al., 1998). At a more complex level, the findings from psychobiological research will hopefully help to improve and tailor therapy approaches for patients with MTD as we discover more about mechanisms at the root of laryngeal tension (Dietrich & Verdolini Abbott, 2008). For instance, it may be that some patients would benefit from a more cognitive approach to stress reduction (e.g., Mindfulness-Based Stress Reduction) whereas other patients would benefit from a more physiological approach (e.g., biofeedback).

In conclusion, a psychobiological framework for studying stress and its relation to voice disorders has been mapped out to guide the present research project (Dietrich & Verdolini Abbott, 2008). Its components will be outlined in the following chapter.

2.0 A PSYCHOBIOLOGICAL FRAMEWORK FOR STUDYING STRESS AND ITS RELATION TO VOICE DISORDERS

"It is much more important to know what sort of a patient has a disease, than what sort of disease a patient has." -William Osler

There is a conspicuous lack of *psychobiological* research that targets (1) the actual effects of perceived stress on laryngeal function and (2) the mechanisms that may mediate relations between (a) stress and laryngeal tension and (b) stress and MTD (Dietrich & Verdolini Abbott, 2008). To guide thinking, a psychobiological framework for studying stress and its relation to voice disorders has recently been mapped out, which emerged from consideration of both the health psychology and MTD literature (Dietrich & Verdolini Abbott, 2008). The framework, which is a work in progress as new knowledge will be incorporated, describes the potential links between stress and laryngeal functioning, and ultimately the relevance of stress for various voice disorders (Figure 2-1). In the present context however, the focus will be on implications of stress for the occurrence of MTD as a pathology affecting voice. The framework is characterized by complex and multiple reverberations among all levels of the model, not all of which are explicitly shown. In the next paragraphs, first, an overview of the model will be presented. Then, a short review of each of the model's components will follow and each section will conclude

with relevant findings or speculations with regard to MTD or the larynx. The chapter will end with research questions and hypotheses to be pursued in the present study.

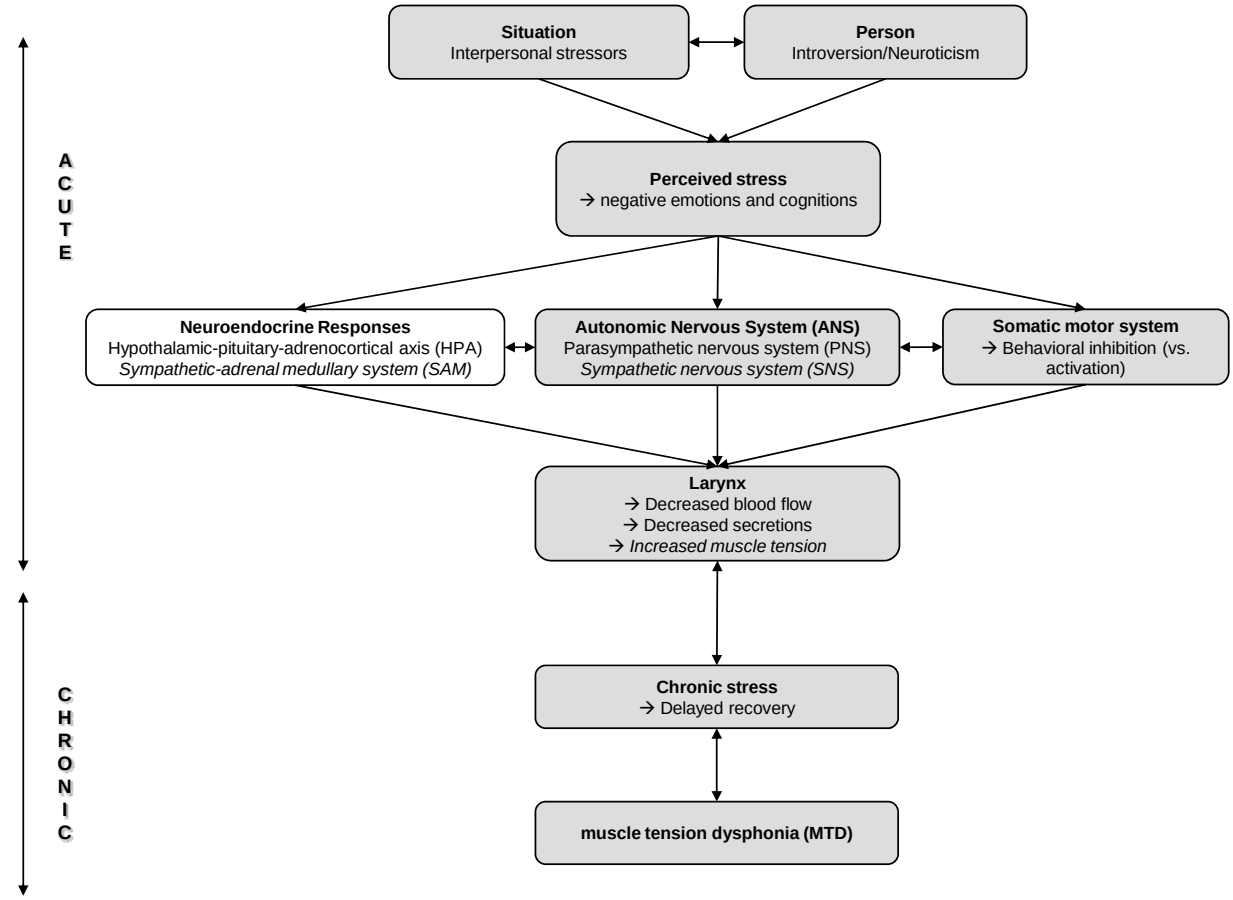


Figure 2-1. A psychobiological framework for studying stress and its relation to voice disorders. From Dietrich, M. & Verdolini Abbott, K. (2008). Psychobiological framework for stress and voice: A psychobiological framework for studying psychological stress and its relation to voice disorders. In: K. Izdebski (Ed.), *Emotions in the Human Voice* (Vol. II, Clinical Evidence, pp. 159-178). Copyright © 2008 by Plural Publishing, Inc. All rights reserved. Used with permission.

2.1 OVERVIEW

2.1.1 Definitions

2.1.1.1 Perceived stress

Before embarking on a strictly linear discussion of the model, there would be value to laying out definitions of key terms and concepts first. The first case in point regards the term "stress." Stress clearly plays a central role in the proposed framework, as the key questions are (a) to what degree and in what form stress affects laryngeal muscle tension as a function of personality and (b) *how* such changes may take effect. Stress can be variably defined. The essence of many definitions is captured by the comment that stress is a "process in which environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological and biological changes that may place persons at risk for disease" (Cohen, et al., 1995, p.3). Indeed, within the health psychology literature, the concept of stress is traditionally geared towards negative health outcomes (Cohen, et al., 1995). In more specific terms, *stressors* are acute or chronic life experiences or conditions that can be of a psychological, physical, or biological nature (Dickerson & Kemeny, 2004), and according to the foregoing definition, place demands on an individual that tax his/her adaptive capabilities. *Distress* is the negative psychological response to stressors, which can include a variety of affective and cognitive states (Kemeny, 2003). For instance, the classic fight-or-flight response has been noted to be associated with *negative emotions* of either aggression (fight: anger, rage, frenzy) or *fear* (flight: anxiety, terror, panic) (Goldstein, 2001; Lovallo, 2005). Of all emotions, in particular anxiety has been highlighted as a "stress emotion" (Endler, 1997; Endler & Kocovski, 2001; Lazarus, 1999; Scherer, 1986).

In contrast to the environmental perspective (number of life events), the psychological stress perspective, which is the perspective of choice for the present research proposal, focuses on the *perception* of stress arising from the subjective evaluation of stressors (Cohen, et al., 1995). Appraisal theory has been influential in this regard, because it accounts for individual differences in stress responses and specific emotions experienced (Lazarus, 1999; Lazarus & Folkman, 1984). Regarding the emotional impact, some authors have considered stress as the result of prolonged emotional arousal (Scherer, 2000). According to appraisal theory, individuals evaluate the personal significance of human interactions and categorize them as harm, threat or challenge. Such categorization is based on the outcome of an environment (demands, constraints, opportunities, culture) by person interaction (goals and goal hierarchies, beliefs about self and world, personal resources) (Lazarus, 1999). Appraisal theory is widely accepted, but has been criticized for the over-emphasis on *cognitive* process and the under-emphasis of other processes such as emotions. As a rebuttal, it has been pointed out that appraisal does not necessarily imply awareness, thus expanding the view of "cognitive processes" in the model (Lazarus, 1984; Zajonc, 1984). However, neuroscience points to the relative independence of emotion from cognition for at least a subset of emotions, e.g. by pointing to a direct connection between fear and the amygdala (Zajonc, 1984).

2.1.1.2 Stress reactivity

A central concept in stress research has to do with stress *reactivity*. For example, cardiovascular stress reactivity refers to an increase in physiological activity from baseline in reference to a stressor and investigates the notion that some individuals may exhibit "exaggerated" responses (Manuck, Kaplan, Adams, & Clarkson, 1989). Stress reactivity is investigated for its potential to

predict negative health outcomes by exploring the specific mechanisms linking individuals with disease (Linden, Gerin, & Davidson, 2003). Such exploration is complicated by differences across individuals at one or several levels of the stress response, including appraisal and emotion, hypothalamic and brainstem activation, peripheral responses, coping, and health behavior (Lovallo, 2005). However, differences in appraisal seem to be the primary force for the perception of stress and subsequent stress reactions (Cohen, et al., 1995; Dickerson, Gruenewald, & Kemeny, 2004). Specifically, negative emotions seem to play a key role in stress-related physical and psychological disease states (Cohen, et al., 1995).

2.1.2 Overview of the framework

This section on the orientation to the central concept of perceived stress will be concluded with a brief outline of the entire framework proposed here (Figure 2-1) (Dietrich & Verdolini Abbott, 2008). Thereafter, components of the framework will be discussed in more detail, as divided into the following parts: (1) The person-by-situation interaction fueling a person's emotional and cognitive stress response, (2) the physiological stress response, (3) the behavioral stress response, (4) the stress response in the larynx, and (5) chronic stress.

First, it cannot be overemphasized that *stress* is the potential result of an environment by person *interaction*. If a person is not well-equipped to handle a situation, a threat may be *perceived,* triggering the experience of a set of *negative emotions and cognitions*. The internal reaction to threat further elicits an acute psychobiological response, the fight-or-flight response, which coordinates changes in the body in order to meet the needs of the situation. Such changes arise from the involuntary autonomic nervous system (ANS) and the voluntary *somatic system*.

Physiological adjustments are orchestrated by interacting *autonomic* and *neuroendocrine* operations. Stress may also induce shifts in immunological functioning, which will be set aside for the moment. Autonomic function is integrated with actual and potential *behavior* (*somatic system*) and typically induces increased muscular contractile levels. In the *larynx*, the stress response is thought to exert the same physiological changes as in other body parts. The primary focus in this document will be on laryngeal muscle activity, because of its immediate clinical significance for MTD. Finally, it is proposed that *inadequate recovery* may play an important role in creating *chronic* laryngeal muscle tension that may be related to various voice pathologies including MTD.

2.2 THE PERSON-BY-SITUATION INTERACTION

Common sense tells us that not everybody reacts the same way in stressful situations. Research to date agrees that a person-by-situation interaction may best explain interindividual variability. The quality of stress responses may depend on a person-by-situation match or mismatch (Dickerson, et al., 2004; Endler, 1997; Lazarus, 1999; Linden, et al., 2003). In other words, people react worst in situations in which their traits are a mismatch to the situation, e.g. as with an introverted individual being put on the spot to be a group leader. Therefore, not surprisingly, individual responses appear to be moderately reliable across time, but only within tasks (Cohen & Hamrick, 2003).

Personality and dispositional emotionality are inextricably linked. There is robust evidence that extroverts have a tendency towards positive emotionality and that neurotics have a

tendency towards negative emotionality (Gross, Sutton, & Ketelaar, 1998; Zelenski & Larsen, 1999a). In fact, neuroticism and negative affect are often used synonymously, and the opposite of positive emotionality is often considered introversion (LaRowe, Patrick, Curtin, & Kline, 2006). Relevant for the present research program is the notion that the traits Positive Emotionality (PEM) and Negative Emotionality (NEM) are considered emotion and temperament constructs that are thought to map onto underlying biological and brain systems, thus creating linkages across psychological processes, neurobiology, and ultimately behavior (Patrick, et al., 2002). The link to behavior is that PEM and NEM are thought to be driven by motivations such as appetitive-approach and defensive-withdrawal, respectively, resulting in opposite behavioral responses to a situation (Watson, Wiese, Vaidya, & Tellegen, 1999). Gray's (1987) Behavioral Inhibition System (BIS), a focus in the present research because of potential relations to exaggerated muscle tension, has been linked to anxiety and neuroticism, but also to introversion and constraint (Patrick, et al., 2002; Roy & Bless, 2000a; Zelenski & Larsen, 1999b). In the next paragraphs, traits related to anxiety will be discussed further, because anxiety is considered central in stress research (Endler, 1997; Endler & Kocovski, 2001; Lazarus, 1999; Scherer, 1986).

2.2.1 Neuroticism

Neuroticism, which is inter-related with anxiety (Fullerton, 2006), reflects a predisposition for a wide range of *negative affect* including stress reaction, alienation, and aggression (Costa & McCrae, 1985a; Patrick, et al., 2002; Suls & Martin, 2005; Tellegen, 1982, in press). Specifically, the notion is that neurotics have a tendency to (1) hyperreact emotionally, (2)

selectively process or elaborate negative affect or information that has a potentially negative content, thus provoking additional encounters that may have negative aspects, (3) appraise more situations as threatening, (4) recover more slowly from negative affect and ruminate, and (5) exhibit a lack of habituation to a repeated stressor, possibly reflecting inadequate coping strategies (Suls & Martin, 2005). This "neurotic cascade" is proposed to consist of distinct and interrelated mechanisms that reinforce and amplify each other (Suls & Martin, 2005).

A vicious cycle becomes apparent in which neuroticism may provide a fruitful ground for the experience of stress. Clearly, elevated *emotional* stress reactivity has been documented for neurotics as compared to non-neurotics, but the evidence around concurrent greater *physiological* stress reactivity has been less convincing. A meta-analysis of studies that looked at personality as a possible source of individual variability for blood pressure showed weak results (Jorgensen, Johnson, Kolodziej, & Schreer, 1996). Chronically elevated blood pressure and essential hypertension were associated with more defensiveness, negative affect, and less affective expression, in particular in interpersonal contexts. However, the effect sizes were small and the variability was large (Jorgensen, et al., 1996). Research to date concludes that individuals high in trait negative affect are *emotionally* more stress reactive, but are comparable to other individuals in terms of *physiological* stress reactivity, and if a trend for exaggerated cardiovascular reactivity exists within this group of people, differences with respect to non-neurotic individuals may be weak (Costa & McCrae, 1985a; Schwebel & Suls, 1999; Suls & Martin, 2005). Of note, neurotics often *report* a high number of somatic complaints in the absence of organic disease (Costa & McCrae, 1985a). However, of relevance for the clinician and researcher, these complaints may be partially the result of a dispositional style of perceiving, recalling, and

reporting of bodily events, which includes a general oversensitivity to somatic concerns (Costa & McCrae, 1985a).

2.2.2 Social anxiety

Individuals high in *social anxiety* are thought to be particularly sensitive to *social-evaluative threat,* according to "Social Self Preservation Theory" (Dickerson, et al., 2004). This theory proposes that perceived threats to the fundamental human goal of preserving the social self (social esteem and status, acceptance, and potential or explicit rejection) typically elicit negative self-evaluative states, especially shame. Concurrent cognitive and behavioral states involve withdrawal and disengagement (Dickerson, et al., 2004). Biologically, task performance of normal participants under conditions of imposed social-evaluative threat has been shown to be significantly correlated with higher cortisol responses post stress reactivity and recovery, and only marginally correlated with higher cardiovascular reactivity, in comparison to responses during tasks in the absence of social-evaluative threat (Gruenewald, Kemeny, Aziz, & Fahey, 2004). Greater increases in shame and decreases in social self-esteem, more than differences in state anxiety, appeared to mediate the cortisol changes (Gruenewald, et al., 2004). Of note, cortisol changes in response to social-evaluative stress could not be consistently found as a function of neuroticism although neuroticism may be related to low self-esteem (Costa & McCrae, 1985a; Kirschbaum, Bartussek, & Strasburger, 1992; van Eck, Nicolson, Berkhof, & Sulon, 1996). Findings from this research underline the importance of a person-by-situation *interaction* for explaining interindividual differences in physiological responses to stress.

2.2.3 Person-by-situation interactions in MTD

Person-by-situation interactions that increase the susceptibility to stress may be at play in some individuals with a complaint of MTD. Personality variables that characterize a substantial subset of persons with MTD include introversion, neuroticism, and social anxiety (Roy, et al., 2000a; van Mersbergen, et al., 2008). With regard to situational factors, individuals with MTD appeared to have accumulated life stress, in particular of an interpersonal nature, surrounding the onset of their voice disorder (House & Andrews, 1987; A. House & H. B. Andrews, 1988). Based on knowledge from the health psychology literature, it is conceivable that stressful and overly taxing person-by-situation interactions may affect a person's well-being including vocal functioning. However, beside individual differences related to trait-specific appraisal, emotionality, and consequently perceived stress, individuals may also differ in their basal physiology (Lovallo, 2005). Turning to the next level in the model, physiological systems involved in stress reactions will be reviewed.

2.3 THE PHYSIOLOGICAL STRESS RESPONSE

2.3.1 Autonomic nervous system

The autonomic nervous system (ANS) is well known for its regulation of the classic "fight-and-flight" response and hence is considered a biological stress response system (Lovallo, 2005). The ANS is a division of the peripheral nervous system and it is extraordinarily well suited to

respond in a quick and coordinated way, because it is well-connected to every organ in the body (Iversen, Iversen, & Saper, 2000). Specifically, the ANS is a visceral sensory and motor system that innervates cardiac muscle, smooth muscle (e.g., skin, blood vessels), and glandular tissues, and regulates a range of reflexes such as ocular, cardiovascular, glandular, and gastrointestinal reflexes. It acts widely involuntarily in parallel with the voluntary somatic sensory and motor system in order to prepare the body for action (Iversen, et al., 2000). The autonomic innervation of skeletal muscle has been established (Goldstein, 2001).

Commonly, two tonically active divisions are distinguished within the ANS: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). Phasic changes in the SNS involve a short-lived, and thus adaptive, arousal reaction. At the neural autonomic level, the neurotransmitter involved is norepinephrine, which increases the rate and force of contraction of target muscles (e.g., cardiovascular effects such as increased heart rate and blood pressure), induces vasoconstriction in nonworking muscles and the viscera, and inhibition to most glands. On the flip side, the PNS is known for the "rest and digest" response that ensures homeostasis or return to such a state from phasic arousal. The vagus nerve plays a central role in the PNS and vagal tone controls normal cardiovascular, esophageal, respiratory and gastrointestinal functioning (Iversen, et al., 2000). The neurotransmitter involved is acetylcholine, which downregulates cardiovascular functioning (e.g., decreases heart rate), induces vasodilation, and promotes glandular secretions (Iversen, et al., 2000).

Sympathoneural activity is supported by hormones released into the bloodstream via the sympathetic-adrenal medullary system (SAM) and the hypothalamic-pituitary-adrenocortical (HPA) axis (Goldstein, 2001). Epinephrine is released from the adrenal medulla and cortisol from the adrenal cortex as controlled by the hypothalamus (Iversen, et al., 2000). Both hormones

reinforce actions of the SNS and assume responsibility to provide the body with adequate energy (e.g., glucose, fat) to meet changed metabolic demands (Kemeny, 2003).

In summary, physiological responses to perceived stress typically include (a) an increase in cardiovascular function as driven by the SNS, (b) vasoconstriction in nonworking muscles and the viscera and redistribution of blood flow to exercising muscles, (c) a decrease in glandular secretions, and (d) an increase in muscular contraction. Both autonomic cardiovascular and endocrine measures have been used as biological indicators of perceived stress (Cohen, et al., 1995). However, the time course of these measures differs substantially. Whereas autonomic indicators respond rapidly on the order of seconds and return to baseline within minutes (Linden, Earle, Gerin, & Christenfeld, 1997), cortisol responds slowly with a peak usually 20-40 minutes post stressor exposure and recovery lasting up to one hour (Dickerson & Kemeny, 2004).

2.3.2 Physiological stress reactivity

In the context of stress and disease, most research focuses on cardiovascular reactivity in response to acute laboratory stressors, and its prognostic relationship for coronary heart disease (Linden, et al., 2003). Stress reactivity research is appealing, because results have moderate stability over time and may serve as a window into complex psychobiological processes (Cohen & Hamrick, 2003; Kamarck & Lovallo, 2003; Linden, et al., 2003). In the 1990s, the hyperarousal of the SNS was regarded as the primary mediator in cardiovascular reactivity (Linden, et al., 2003). However, both blood pressure and heart rate reflect a mix of sympathetic and parasympathetic control, and now research investigates phasic contributions of both the SNS and PNS more carefully (Linden, et al., 2003). Further, a shift has been seen in the literature

away from assumptions that the physiological stress response is generalized and non-specific, to acknowledgements that some specific stress response patterns may exist (Goldstein, 2001; Kemeny, 2003; Lovallo, 2005). The patterning is thought to reflect differences in the (behavioral) responses required by different stressors, inter-individual differences in responding to the same stressor, and varying inter-individual responses across different stressors (Cacioppo, et al., 1998; Ekman, 1999; Lazarus, 1999; Scherer, 2000).

At least three factors have been identified that may drive differentiated psychobiological response patterns to stressors (Kemeny, 2003): (1) threat versus challenge, (2) uncontrollability, and (3) negative social evaluation. For example, although cardiac performance increases during the experience of both threat and challenge, a difference may emerge with regard to peripheral resistance to blood flow. The perception of threat can induce increased peripheral resistance to blood flow and thus increased blood pressure whereas peripheral resistance may be unchanged or reduced during perceived challenge, which would be interestingly similar to processes occurring during aerobic exercise (Kemeny, 2003). Comparing neural and hormonal responses, during fight or anger the SNS response would be expected to be larger than the SAM response, whereas during flight or anxiety, both the SAM system and the SNS would be expected to be equally activated (Goldstein, 2001). For both scenarios, skeletal muscle tone would be anticipated to increase. Further, research to date points to evidence for differential HPA reactivity as a function of stressor (Cacioppo, et al., 1998; Kemeny, 2003). Suggestions have been made that motor and cognitive effort drive SAM system activation, with accompanying rises in catecholamines, muscle tension, cardiac output and blood pressure, whereas affective stressors specifically activate the HPA axis (Linden, et al., 1997). For example, both uncontrollability or a threat to the social self have been shown to drive increases in cortisol (Dickerson & Kemeny, 2004). In fact, a

stressor that engages an individual in an active performance task such as public speaking under a time constraint with the potential for negative judgment by others, has been found to elicit the largest and longest lasting cortisol responses in the laboratory, in addition to strong cardiovascular and subjective stress responses (Dickerson & Kemeny, 2004).

2.4 THE BEHAVIORAL STRESS RESPONSE

The ANS coordinates the stress response and its processes aim to support (potential) behavior arising from the appraisal of the stressor (Iversen, et al., 2000; Lovallo, 2005). Control over organic function is hierarchically structured with central as well as local reflexive control mechanisms (Lovallo, 2005). Sensory information is evaluated in the prefrontal cortex. From the prefrontal cortex, extensive connections exist to the limbic system, which in turn has connections to the hypothalamus. The central command centers including the frontal lobe, hypothalamus, and brainstem finally prime motor output (Kamarck & Lovallo, 2003). As part of the stress response, cardiovascular responses perfuse tissues with oxygen and glucose for the execution of behavior such as fleeing or fighting, and blood flow may be shifted from the viscera and skin to large muscles (Goldstein, 2001).

Psychobiological theories of personality have linked affective predispositions to behavioral tendencies (Eysenck, 1967; Gray, 1982). Neuroticism is thought to be related to an overactive limbic system and an overactive ANS (Zelenski & Larsen, 1999a). Further, as previously discussed, Gray (1982) distinguished two systems that reflect appetitive and aversive motivation respectively. The Behavioral Activation System (BAS) is associated with approach

and active avoidance behaviors resulting in impulsivity, whereas the Behavioral Inhibition System (BIS) is associated with passive avoidance and extinction of non-reward behaviors resulting in anxiety (Zelenski & Larsen, 1999a). A factor analysis of personality taxonomies (Eysenck, Gray, Cloninger) revealed three factors that appeared to map best onto the three-factor model of personality proposed by Tellegen (1982) (Zelenski & Larsen, 1999a): (1) Impulsivity-thrill seeking: impulsiveness, novelty seeking, psychoticism, fun seeking, and venturesomeness; (2) Reward sensitivity (positive emotions): reward expectancy, persistence, reward responsiveness, drive, and extraversion (BAS); and (3) Punishment sensitivity (negative emotions): neuroticism, harm avoidance, punishment expectancy, and reward dependence (BIS).

2.4.1 Muscular behavior

Generally, stress is associated with an increase in muscle tension (Goldstein, 2001). Skeletal muscle sympathoneural activity (SMSA) increases with a rise in blood pressure and induces systemic neurocirculatory changes. For example, studies investigated the effects of perceived stress on muscle tension in the upper extremities (e.g., neck and shoulders), which is a common work-related disorder (Lundberg, 2002; Lundberg, et al., 1994). Findings indicated that not only physical load (e.g., manual labor), but also psychological stress may induce muscle tension in the upper body, especially in the trapezius muscle (Krantz, Forsman, & Lundberg, 2004; Lundberg, 2002; Lundberg, et al., 1994). Such increase in muscle tension was correlated with an increase in BP and HR. The BP increase was further correlated with an increase in norepinephrine. However, it is not clear to what extent the increase in norepinephrine was directly related to muscle tension or just generally related to arousal (Lundberg, et al., 1994). In other words, to

what degree the SMSA is *directly* related to muscle tension during stress is less clear and there is controversy about whether EMG responses under stress reflect global activation, task-specific activation, or idiosyncratic activations (Goldstein, 2001; Tassinary & Cacioppo, 2000).

In contrast to physical stress (e.g., manual labor, exercise), which is usually well-defined in the temporal domain, emotional stress may persist beyond the termination of exposure to the stressor and may unduly sustain elevated muscle tension. Inadequate recovery and rest of muscles may be a risk factor for developing and maintaining musculoskeletal disorders (Lundberg, 2002). Moreover, women are thought to be more susceptible to musculoskeletal disorders because they are more prone to worry and are generally less able to "wind down" after work (Kiecolt-Glaser & Yehuda, 2005; Lundberg, 2002). Relevant hypotheses about mechanisms causing muscle pain and fatigue converge to the notion that a lack in rest and recovery from either physical or psychological load may induce degenerative processes in muscles. According to the Cinderella Hypothesis, low threshold motor units may be kept active under stress, may be overloaded, and consequently react with metabolic disturbances, degenerative processes, and pain (Hägg, 1991; Sjøgaard et al., 2000, cited in Lundberg, 2002). Such chronic low levels of muscle tension may also contribute to inflammatory processes in poorly vascularized areas of the muscle (Sjøgaard et al., 2000, cited in Lundberg, 2002). Knowledge about alterations in muscle biology invoked by stress may be informative in the context of laryngeal functioning, as discussed next.

2.5 THE STRESS RESPONSE IN THE LARYNX

Our voice provides not only a window into the status and functioning of the larynx, but also into the state of our mind and body (Aronson, 1990; Titze, 1995). Of interest for the present discussion is that stress typically results in an increase in cardiovascular functioning and ANS-mediated (a) vasoconstriction in nonworking muscles and the viscera and redistribution of blood blow to exercising muscles, (b) a decrease in glandular secretions, and (c) an increase in muscle activation. Although these findings are well established for the soma in general, research has been minimal with respect to the larynx. Histological studies indeed indicate that the structures for autonomic innervation are in place for the SNS to exert these effects in the larynx (Hisa, 1982; Hisa, et al., 1999; Hisa, Matsui, Fukui, Ibata, & Mizukoshi, 1982; Hisa & Sato, 1991). Noradrenergic fibers have been found to run along with blood vessels and muscle fibers and close to submucosal glands.

2.5.1 Blood flow regulation

Both physical and emotional stressors may temporarily affect the distribution of blood flow in the larynx (Lyon & Barkmeier-Kraemer, 2004). Blood flow is regulated by the ANS and endocrine systems. Vasoconstriction typically occurs in non-working muscles whereas vasodilation would occur in working muscles that have an increased need of nutrients (Iversen, et al., 2000; Lovallo, 2005). Further, actual variations in laryngeal blood flow may depend on a multitude of factors including muscle fiber type and activity level, metabolites, and tissue

integrity and morphology (Lyon & Barkmeier-Kraemer, 2004). The relations between changed vocal fold vascular supply and voice disorders have not been extensively researched and most research thus far focused on factors related to vocal fold lesions (Lyon & Barkmeier-Kraemer, 2004). However of greater interest for the present research, it has been pointed out that a lack of oxygen in the laryngeal muscles, possibly due to local vasoconstriction, may be related to the occurrence of vocal fatigue (Švec & Šram, 2001).

2.5.2 Regulation of glandular secretions

The amount and quality of glandular secretions changes during a stress reaction (Iversen, et al., 2000). Specifically, less and more viscous secretions would be expected (Iversen, et al., 2000). Such changes may also affect the larynx, because numerous submucosal glands are located in the human laryngeal and tracheal mucosa (Sato & Hirano, 1998). Of relevance for voice production, a relative loss of mucosal secretions and increase in viscous secretions may amount to a decrease in local hydration. The relevance of reduced surface or systemic hydration to the vocal folds has long been under investigation. Research has indeed shown that experimental interventions aimed at decreasing hydration may increase the phonatory threshold pressure, that is the subglottal pressure required to initiate and sustain vocal fold vibrations (Fisher, Ligon, Sobecks, & Roxe, 2001; J. Jiang, Verdolini, Aquino, Ng, & Hanson, 2000; Verdolini, Titze, & Fennell, 1994) and in turn may increase an individual's perceived vocal effort during voicing (Verdolini, et al., 1994). Less relevant to the present research, but pertinent to hydration and voice in general, dehydrating interventions may also increase the risk of phonotraumatic lesions, at least

theoretically (Titze, 1981) and empirically, hydrating interventions may have potential to reverse the same lesions (Verdolini-Marston, Sandage, & Titze, 1994).

2.5.3 Laryngeal muscle control

Interpreting the functioning of the larynx against the backdrop of evolution should be informative in laryngeal psychobiological research. Such an approach may inform our understanding of the differences in laryngeal functioning under various conditions such as emotional stress. The larynx is phylogenetically much older than the neocortex which, for instance, contains the neuromuscular systems for articulation and language (Aronson, 1990). The larynx's primary and vital function is to protect the airway from foreign substances by way of laryngeal closure. Many primitive reflex arcs subserve this purpose, e.g., cough reflex and swallowing arcs (Ludlow, 2005). Further, impounding air in the thoracic cage by laryngeal closure stabilizes the thorax to support activity with the upper extremities (Aronson, 1990). Consequently, the larynx's role in speech production and expression of thought is an evolutionary recent and secondary one (Aronson, 1990).

Similar to the control of volitional physical behavior in general, vocal behavior is generally thought to be hierarchically organized comprising control mechanisms ranging from lower-level reflexive to higher-level cortical operations (Jürgens, 2002)[3]. Mechanisms of laryngeal muscle control may vary for (1) breathing, swallowing, and cough, (2) emotional expression, and (3) voice for speech communication (Ludlow, 2005). The vagus nerve,

[3] There are arguments that want to attenuate the emphasis on hierarchical control and increase the emphasis on reciprocal, interactive control – as for example in dynamical systems theory (Haken, Kelso, & Bunz, 1985). However, further discussion is beyond the scope of this particular venue.

originating in the nucleus ambiguus of the brainstem, is the primary nerve that supplies the intrinsic muscles of the larynx (Colton, et al., 2006). Of note, the vagus nerve is also the primary nerve in the parasympathetic nervous system (Iversen, et al., 2000). An interaction between higher cortical control and subcortical circuits is likely during voice production, but is not well understood (Ludlow, 2005). For the purpose of speech, it is assumed that laryngeal motoneurons receive fairly direct input from the laryngeal motor cortex in the primary motor cortex (Ludlow, 2005). However, reflexive central pattern generators in the periaqueductal grey (PAG) and nucleus retroambiguus are thought be involved as well. In contrast, during vocal expression of emotions, the evolutionary older vocalization system composed of the anterior cingulate cortex and PAG will be primarily activated to connect with the laryngeal motoneurons via the reticular formation (Jürgens, 2002; Ludlow, 2005). Of relevance for the discussion of stress, input to the PAG comes from a number of limbic structures such as the anterior cingulate cortex, amygdala, and hypothalamus (Jürgens, 2002), which are structures that are shared in stress and emotion regulation (Lovallo, 2005).

2.5.3.1 Laryngeal muscle control during perceived stress

Clearly, the larynx stands at the crossroads of primitive influences from subcortical circuits and higher cortical control. It is believed that under emotional arousal, phylogenetically older neural mechanisms dominate over higher cortical functions, leading to the disintegration of not only fine-tuned voice production, but also respiration, resonance, and articulation (Aronson, 1990). During emotional arousal *such as fear*, lower functions that promote withdrawal and inhibition may compete with higher functions that strive for communicative expression, possibly creating a basis for MTD (Aronson, 1990). Moreover, speculations have been put forward that stress may

induce firm adduction of the vocal folds to support the thorax or wide abduction to facilitate an increased volume and flow of oxygen in order to meet the body's increased metabolic demands (Aronson, 1990). Others have regarded generalized laryngeal muscle tension as a defensive reflex mechanism to threat (Bradley & Lang, 2000 cited in van Mersbergen et al., 2008). Indeed laryngeal elevation during emotional arousal could be an automatic and defensive maneuver given that this movement employs muscles of swallowing whose primary function it is to protect the airway (van Mersbergen, et al., 2008). In essence, the laryngeal muscles are thought to be exquisitely sensitive to stress and emotion. In other words, any psychological disequilibrium may have the potential to interfere with normal volitional control over the larynx and phonation (Aronson, 1990).

The detailed investigation of laryngeal muscle control under various conditions--especially central control mechanisms--requires functional imaging techniques (Ludlow, 2005). Using a simpler approach, the feasibility of a stress reactivity protocol aimed at exploring *extra*laryngeal muscle tension changes with surface EMG technology was investigated. In a single-subject design, a vocally healthy female adult was exposed to a social-evaluative stressor (Dietrich, Verdolini, & Barkmeier-Kraemer, 2005). The experimental protocol itself consisted of a baseline rest phase, a baseline speech phase (counting), and a stress reactivity phase modeled after the *Trier Social Stress Test* (public speaking and verbal mental arithmetic) (Kirschbaum, Pirke, & Hellhammer, 1993). The stress tasks were performed in front of a four-member audience while the subject was videotaped. The following muscle sites were sampled in order to represent laryngeal elevators and depressors: (1) submental; (2) thyrohyoid (area *overlying* the thyrohyoid muscle); and (3) infrahyoid. Muscle activity was also sampled from the anterior tibialis of the leg to include a non-speech control site to test whether stress-induced muscular

activation is task-specific (speech) or more general to the entire body. In addition, BP and HR were tracked. Voice fundamental frequency (F_0) and intensity were also recorded, however, those data turned out to be unusable.

Stress was successfully induced as seen by increases in BP and HR with the onset of the stressor (Figure 2-3). In terms of laryngeal muscle activity, average SEMG activity was overall lowest during the rest phase (Figure 2-4). During comfortable counting, SEMG activity increased slightly for the submental muscle group only. With the onset of the stressor, SEMG activity increased for laryngeal muscles in the following order of magnitude: (1) thyrohyoid (least), (2) infrahyoid, and (3) submental (most). The increases in muscle activation were paralleled by increases in cardiovascular reactivity. Muscle activity in the leg varied minimally across the protocol. Results were consistent with the hypothesis that stressors may increase laryngeal muscle tension in association with cardiovascular and emotional arousal. Interestingly, the laryngeal muscle activity *pattern* changed with the onset of stressed speech as opposed to comfortable speech. A characteristic of stressed speech was a notable increase in infrahyoid muscle activity that was not seen during comfortable speech, along with a further increase in submental activity as compared to baseline speech. This difference in muscle activation pattern may be an indication that the co-contraction of extralaryngeal muscles, as seen by the increased activation of laryngeal elevator *and* depressor muscles, may be a task-specific sign of speaking under stress.

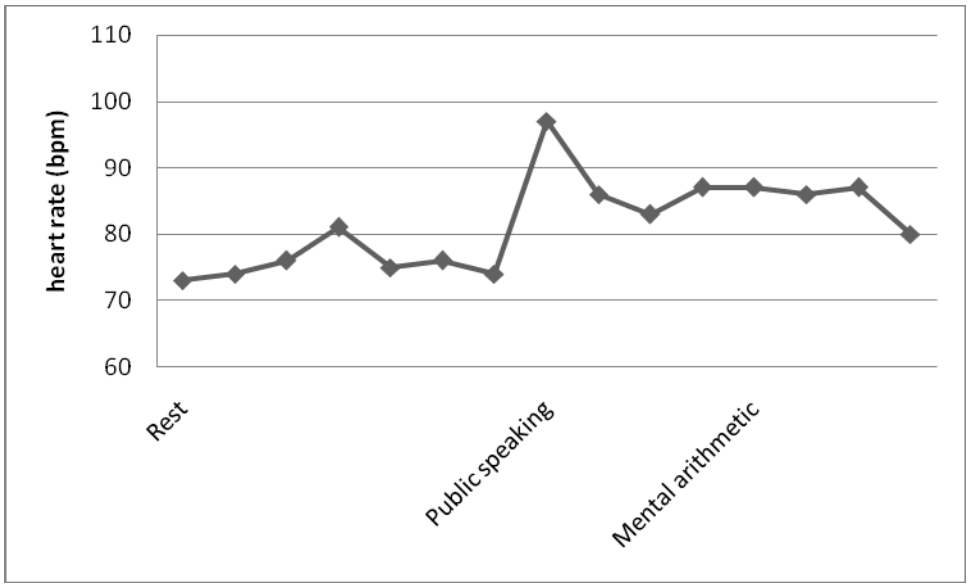


Figure 2-2. Heart rate (HR) activity in beats per minute (bpm) as a function of exposure to stress

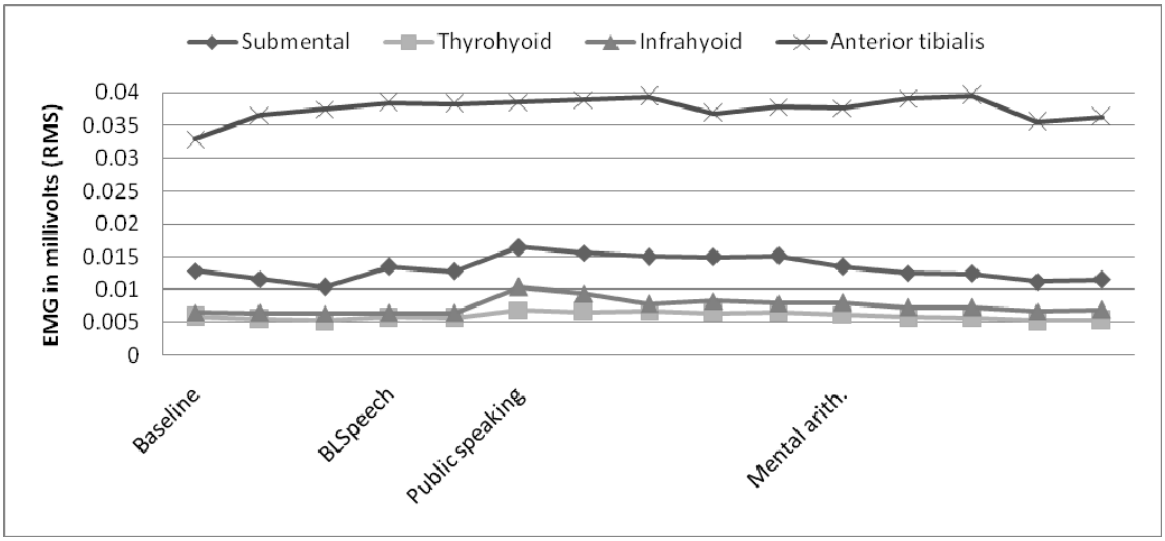


Figure 2-3. SEMG activity in millivolts (RMS) as a function of exposure to stress

This case study investigated the effects of exposure to a stressor on extralaryngeal muscle tension and *indirectly* investigated the processes that may be involved in the links between stress and increased laryngeal tension such as sympathetically mediated cardiovascular reactivity.

Limitations included that this was a single-subject design and that therefore the pattern of results could be entirely idiosyncratic. Further, the non-stressed speech baseline task and the stressed public speaking should be of similar duration for comparison purposes. Finally, a recovery phase would yield additional valuable information especially with regard to the SEMG recovery slope and potential individual differences.

2.5.4 Acoustic changes

A few notes about acoustic changes as a result of stress are indicated. Although listeners are often accurate in detecting emotion, acoustic analysis has failed to decompose reliably the relevant acoustic cues indicative of specific emotions. This failure may be partly due to poor validity of acoustic analysis with respect to perception (Scherer, 1986; Titze, 1995). At a basic level, an increase in voice fundamental frequency (F_0) has been widely accepted as a universal indicator of stress and has been also the most frequently investigated acoustic measure in that regard (Tolkmitt & Scherer, 1986; Wittels, Johannes, Enne, Kirsch, & Gunga, 2002). Further, evidence points to an additional increase in vocal intensity as a non-specific indicator of arousal (positive and negative). A controversy exists in research on vocal expression of emotion as to whether there are qualitatively different vocal profiles for discrete emotions or if voice primarily conveys arousal in a more quantitative sense (Juslin & Scherer, 2005).

According to Juslin and Scherer (2005), both anxiety and stress are associated with an increase in mean F_0, jitter (pitch perturbation), standard deviation of intensity, high frequency energy, speech rate, and decrease in rhythmic regularity. Predictions differ for the direction of F_0 (*SD*) change (decrease for fear and increase for stress) and mean intensity (unchanged for fear

and increased for stress). Overall however, predictions for vocal cues are fairly similar for fear/anxiety and stress/arousal. In an effort to summarize the current literature, Juslin & Scherer (2005) proposed the following set of voice cues in order to measure affective arousal: F_0 (floor, i.e. lowest 5% of F_0 values), F_0 standard deviation (*SD*), intensity (*M*), speech rate (syllables per minute), and high frequency energy (total intensity above 500 Hz). However, if the goal were to distinguish various emotions through the vocal signal, the following parameters should be added based on empirical evidence (Banse & Scherer, 1996; Juslin & Scherer, 2005; Scherer, 1986): F_0 contour, jitter, voice intensity (*SD*), pauses, rhythmic regularity, and first formant (*M*, precision).

Unfortunately, voice cues are rarely reliable, because of (a) intra- and interindividual differences, (b) interactions that involve linguistic contents, (c) degradation of acoustic signals in the natural environment, (d) interactions between physiological effects and strategic artificial posing of emotions, and (e) cues similarly associated with more than one emotion (intercorrelations among cues) (Juslin & Scherer, 2005). Further, results are difficult to compare, because protocols vary having a range of combinations of (different) sustained vowels, different task repetition numbers, the absence or presence of an all-voiced sentence (to test F_0 control independent of adductory control) and control for loudness (Titze, 1995).

2.6 CHRONIC STRESS

A final and central question in stress research surrounds the issue how stress reactions transform from an evolutionary adaptive response to a maladaptive one for some individuals and, in the context of this research program, how this may happen at the level of the larynx (Dietrich &

Verdolini Abbott, 2008). Theories linked to the concept of *allostatic load*, which broadly relates to chronic stress may provide valuable guidance (McEwen, 1998). Allostatic load refers to the long-term effect of the physiologic response to stress such as the chronic overactivity or underactivity of physiological stress response systems, be they neural, hormonal, or immunological. The idea is that stress should be short-lived and any deviation may result in a strain for the physiological system. Four scenarios of allostatic loads have been described (McEwen, 1998): loads from (1) frequent acute stress; (2) lack of adaptation to the same stressor if repeated; (3) loads from delayed recovery; and (4) loads from inadequate responses by some physiological stress response systems that trigger compensatory responses by other systems, e.g., inadequate counter-regulation of inflammation. This framework would be useful for voice disorders research, because it bridges the gap between laboratory stress reactivity and its significance for everyday life (Dietrich & Verdolini Abbott, 2008).

One of the most promising trends for future psychobiological research may be a revitalized focus on stress *recovery* beyond the sole focus on stress *reactivity* (Kamarck & Lovallo, 2003; Linden, et al., 1997; Linden, et al., 2003). Background is that quick recovery after arousal is thought to reflect adaptive functioning and effective coping, a notion that never has been challenged (Linden, et al., 1997). In contrast, maladaptation may consist of affective and cognitive factors such as ruminations sustaining the arousal beyond the acute stress situation (Linden, et al., 1997; Linden, et al., 2003; McEwen, 1998). Even the mere *anticipation* of stress such as anticipatory worry may result in the activation of physiological stress response systems (McEwen, 1998). The notion of delayed recovery is not trivial, because stressful real-life situations rarely are as time-limited as an acute laboratory stressor. Keeping ecological validity in mind, research on recovery characteristics may be inherently more powerful to predict

negative health outcomes than a simple focus on reactivity (Linden, et al., 1997). Finally, from a methodological point of view, the slope of recovery may be more important than the actual time to recovery for the investigation of maladaptive processes, because different individuals and different physiological systems may exhibit differential recovery patterns (Linden, et al., 1997).

2.6.1 Chronic stress and MTD

In summary, the perception of stress is linked to a complex psychobiological and behavioral chain reaction that may affect laryngeal functioning (Dietrich & Verdolini Abbott, 2008). In an effort to explain what makes some individuals but not others develop MTD, one hypothesis is that chronic stress in the form of *delayed recovery* may be a relevant mechanism. Emotional and cognitive hyperreactivity may sustain the stress response after the termination of the stressor. Individuals with MTD prone to tension and worry may evidence "laryngeal inertia" and may unconsciously sustain stressor-induced counterproductive laryngeal patterns, which would put them at risk for laryngeal complaints such as pain or vocal effort. Muscles need rest and recovery and such tension states may cause vocal fatigue. Thus, a shift may be seen from temporary laryngeal changes affecting voice that are not registered as a voice disorder, towards changes in vocal functioning that affect a person's life, which may be eventually registered as a voice disorder. A chronic muscle tension *habit* may develop, which may maintain MTD.

2.7 STATEMENT OF PURPOSE, SPECIFIC AIMS, EXPERIMENTAL QUESTIONS, AND HYPOTHESES

It is clear that research into the laryngeal response to stress, and the pathways that mediate it, is still in its infancy. Gaps in the literature are many. In light of the existing literature in MTD in particular, the present research's goal was to highlight four substantial gaps that are held to deserve particular attention as points of departure (Dietrich & Verdolini Abbott, 2008): (1) to date, although claims are made about the relevance of personality and psychological factors such as stress for MTD, research has failed to evaluate actual *laryngeal behavior* in response to presumed precipitators; (2) assuming that personality and psychological factors are relevant for MTD, research has similarly failed to adequately address the actual *causal* role for MTD; (3) although the *sympathetic nervous system* (SNS) has been widely implicated as a likely mechanism mediating relations between personality and psychological factors and MTD, thus far data to this effect are lacking in the literature; and (4) theoretically motivated *intervention* studies have not been conducted around these issues.

The present study represented a first step in a longer-term research program that will address these gaps. The overarching proposal was that a psychobiological model, described herein, provided a useful theoretical framework within which to couch the individual research questions. The long-term research program will systematically address vocally normal as well as vocally impaired participants. The primary short-term goal was to investigate the question of whether exposure to a stressor actually influences end-organ *extralaryngeal muscle tension* (magnitude and pattern) as a function of personality. Secondary outcomes focused on the effects of exposure to a stressor on voice characteristics, such as perceived vocal effort, voice

fundamental frequency (F_0), and voice intensity, which should covary with laryngeal activation as a function of personality. The ultimate goal will be to elucidate specific mechanisms that underlie a relation between stress and laryngeal muscle tension, such as autonomic nervous system (versus voluntary somatic system) and emotional and cognitive mechanisms. However, the question of *mediation* was only addressed at a *preliminary level* at this point and data collected will help to guide future in-depth research on this question. Clinically, the long-term goal will be to enhance intervention techniques and outcomes for a range of individuals with voice problems using advanced psychobiological knowledge coming from the voice domain. In the interest of clarity, a synopsis of the long-term plan is as follows:

Table 2-1. Overview of short-term and long-term research goals

Goal	Addressed
1. Investigate *effects* of exposure to an acute stressor on laryngeal and vocal function (magnitude and pattern);	Present study → Specific Aims 1 and 2
2. Identify individuals at *risk* for psychobiologically mediated voice disorders, e.g. laryngeal muscle tension disorders;	Present study → Specific Aims 1 and 2
3. Identify psychobiological *mechanisms* that may underlie changes in laryngeal function subsequent to exposure to the stressor, *thereby to*	Exploratory observations in present study → Specific Aims 3-7
4. Generate and evaluate appropriate *education* and *prevention* programs, based on data from the foregoing series; and	Future studies
5. Improve *clinical* services and outcomes for patients with existing muscle-tension related voice disorders.	Future clinical outcome

The present series systematically addressed the first and second of these goals with vocally normal participants. The third goal was addressed at an exploratory level. The data generated from this series will ultimately provide a springboard for a refined causal model and

framework relevant to the fourth and fifth goals, i.e. intervention and clinical outcomes. Specifically, the main focus in the present series was the effect of a stressor on laryngeal activation as a function of personality (risk factor personality). The goal pertaining to mechanisms could only be addressed at a rudimentary level at this stage of inquiry due to methodological constraints and lack of prior data to guide thinking along these lines.

The background and societal relevance of the project is that voice disorders in general, and stress-related complications in voice disorders specifically, are common. The research focused on *personality* and *stress reactivity* to identify individuals *at risk* for laryngeal muscle tension and subsequent pre-clinical and clinical voice problems. This approach was a necessary extension of an existing theory that maintains that individuals with certain personality traits (introversion and extraversion) may have a disposition to develop specific vocal pathologies (primary MTD versus vocal fold lesions) by way of differing laryngeal responses to external stimuli (behavioral inhibition versus behavioral activation) (Roy & Bless, 2000b). Hence, this research's primary focus was on the comparison of introverted individuals versus extraverted individuals and the quantity and quality of their respective extralaryngeal behavior under exposure to a stressor. As an additional layer, the theory maintains that the added presence of the trait stress reactivity (i.e. neuroticism) will magnify any ongoing response tendencies experienced by persons with introversion or extraversion. The role of neuroticism *within* the personality traits introversion and extraversion in relation to extralaryngeal activity was investigated within the limits of state negative emotion experienced during the stress reactivity protocol. Of particular interest was the notion that neuroticism may have a strong influence on a participant's perceptual processes, e.g. emotional reactivity or perceived vocal effort, more than physiological processes (Suls & Martin, 2005).

The theory of the dispositional bases for vocal nodules and functional dysphonia (Roy & Bless, 2000a) is geared toward clinical voice disorders, but the issue of subclinical or mild voice complaints deserves attention as well. Mild voice complaints have been all but neglected in the literature, although they may also limit effective communication in the general – non-treatment seeking – population. Such conditions may place individuals at risk for clinically relevant voice disorders in the future as a function of personality type. The proposed research provided the first systematic test of a psychobiological stress reactivity *risk* model for voice disorders and indirectly investigated potential mechanisms at a preliminary level using a modification of standardized stress reactivity protocol, the *Trier Social Stress Test* (TSST; Kirschbaum, et al., 1993). Ultimately, psychobiological research aims to understand who is vulnerable to disease by way of searching for individual markers of causality or, at least, risk (Cacioppo, et al., 1998; Kamarck & Lovallo, 2003).

The specific aims and experimental questions, posed shortly, were asked in reference to the causal model under investigation, shown graphically in Figure 2-6. According to this model, personal factors, specifically trait emotionality (introversion versus extraversion) interact with situation (stressor) to influence emotional and cognitive reactions, which in turn drive ANS response, which further influence laryngeal as well as vocal responses. Not shown in the figure, but implicit in the model are feedback loops such that ultimately, the model becomes non-linear. However, for our purposes, the focus was the model's multiple tiers, and thus its causal complexity. The short-term goal was to provide preliminary information that will be useful towards the ultimate goal of complex model building. Specifically, the present series assessed the assumption that person x situation interactions in effect *do* influence laryngeal behavior (primary outcome, I) as well as corollary voice output parameters (secondary outcome, II). At a

tertiary and exploratory level (III), the series provided preliminary data around possible mechanisms that may mediate this effect, anticipated from preliminary data, in terms of (III) possible person x situation influences on (IIIa) emotional and cognitive reactions and (IIIb) ANS reactions, (IIIc) emotion and cognition's possible effects on laryngeal responses, and (IIId) associations between ANS functions and laryngeal responses.

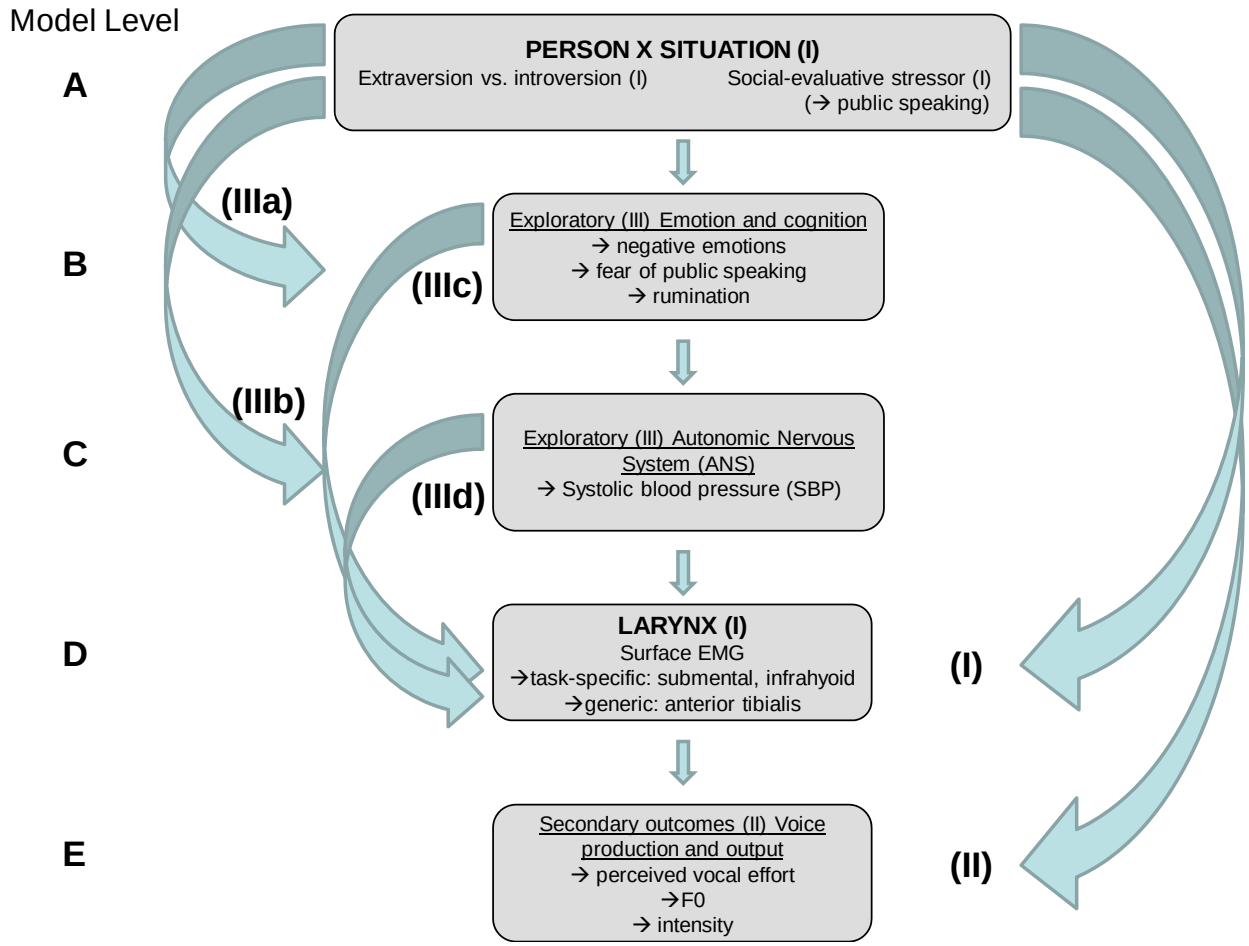


Figure 2-4. Causal model under investigation pertaining to the effects of exposure to a stressor on laryngeal activation as a function of personality (Dietrich & Verdolini Abbott, 2008). The Roman numbers relate to primary (I), secondary (II), and exploratory (III) outcomes

2.7.2 Primary outcome (I)

Specific aim 1 (addresses goals 1 and 2 above):

To assess the influence of exposure to a stressor on the magnitude and pattern of extralaryngeal muscle activation, as compared to muscle activation in a non-speech control site, as a function of personality (extraversion vs. introversion). Muscle activations were measured using SEMG. Extralaryngeal measurement sites included submental and infrahyoid muscle groups and the remote control site was the anterior tibialis of the leg.

Overview:

A modification of standard stress reactivity protocol was chosen to induce stress in participants relative to this aim. The *Trier Social Stress Test* (TSST; Kirschbaum, et al., 1993) is an established stress reactivity protocol, which has been used extensively in psychobiological research and thus allows for a replication of known effects (e.g. emotional and cardiovascular responses). The following facts further supported the use of the *TSST*. The *TSST* is considered a *social-evaluative* stress reactivity protocol and has been shown to be among the most powerful in eliciting stress responses (Dickerson & Kemeny, 2004), the public speaking task is ecologically valid, and interpersonal and social-evaluative stress is the type of stress that is often referenced in the voice disorders literature (Aronson, 1990; A. House & H. B. Andrews, 1988). In addition, based on the theory of the dispositional bases for vocal nodules and functional dysphonia (Roy & Bless, 2000a), the personality traits introversion and extraversion were examined for their proposed *differential* influence on laryngeal behavior, i.e. behavioral inhibition versus behavioral activation, and how these responses played out in the extralaryngeal network.

*Extra*laryngeal behavior was under investigation, because of easy accessibility of extralaryngeal muscles and the opportunity to sample opposing extralaryngeal muscle groups involved in voice production (submental – laryngeal elevation; infrahyoid – laryngeal depression). Primarily, the magnitude of the SEMG responses was investigated during speech and non-speech phases of the experimental task. Secondarily, the *pattern* of the laryngeal responses was screened and summarized descriptively, i.e. the relation between submental and infrahyoid SEMG activity as a function of personality and exposure to public speaking. Last, a limb muscle was chosen as a non-speech specific muscular control site to test if the stress response was general to the whole soma or task-specific. The comparison of all sites was descriptive due to the challenge of quantitatively comparing muscle groups that are different in size and composition of the muscle group.

Experimental question:

Will there be a significant interaction between the effects of experimental phase (baseline speech, rest, anticipation, public speaking, recovery, and recovery speech) and personality (extraversion vs. introversion) on peripheral SEMG behavior, and if so, are the changes task-specific (extralaryngeal SEMG: submental and infrahyoid) or general to the whole soma (leg SEMG: anterior tibialis)? The relevant dependent and independent variables were the following:

Table 2-2. Dependent and independent variables for Specific Aim 1

Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
Submental SEMG (% MVC)	Phase	Personality (extraversion/introversion)	All
Infrahyoid SEMG (% MVC)	Phase	Personality (extraversion/introversion)	All
Anterior tibialis SEMG (% MVC)	Phase	Personality (extraversion/introversion)	All

H_0: There will be no significant interaction between the effects of personality and phase on the magnitude of SEMG activity (submental, infrahyoid, anterior tibialis). A null result would fail to produce evidence consistent with the model's prediction that exposure to a stressor will alter the participants' magnitude of SEMG activity as a function of personality, i.e. the prediction of differential stress reactivity with respect to extralaryngeal function.

H_1: There will be a significant interaction between the effects of personality and experimental phase on the magnitude of each SEMG activity. Specifically, based on logic and pilot data, submental SEMG will be greatest for extraverts during the stressor phase, and infrahyoid and anterior tibialis SEMG will be greatest for introverts during the stressor phase. The results would be consistent with the model's prediction that differential extralaryngeal stress reactivity exists as a function of the traits introversion and extraversion. Specifically, positive results would strengthen the theory of the dispositional bases of vocal nodules and functional dysphonia and would invite in-depth research (Roy & Bless, 2000a).

Note magnitude and pattern of SEMG responses:

The following *pattern* of extralaryngeal muscle activity was hypothesized: introverted participants will show a relatively greater increase in infrahyoid SEMG activity than submental activity under stress, whereas the opposite pattern will be observed for extraverted subjects. In more detail, the described behavioral *activation* pattern is supposed to reflect unrestricted laryngeal activation for speech whereas the behavioral *inhibition* pattern is thought to reflect laryngeal activation for speech that is *impeded* by the disproportionate presence of laryngeal depression, which may restrict laryngeal flexibility thought to be necessary for healthy voice production. At the same time, the *degree* of introversion and extraversion may influence the magnitude of the responses as well as other associated features outlined in the secondary and

exploratory outcomes below. In addition, the following general progression was expected for the magnitude of the SEMG responses as a function of phase: rest < recovery < anticipation < baseline speech < recovery speech < stressor (public speaking). This progression basically reflects a continuum from least stressful to most stressful including nonverbal (rest, anticipation, recovery) and verbal tasks (baseline speech, public speaking stressor, recovery speech).

2.7.3 Secondary outcomes (II)

Specific aim 2 (offshoot of goals 1 and 2, to gain supplementary data around laryngeal activation and vocal consequences thereof):

To assess the influence of stressor exposure (baseline speech, public speaking, recovery speech) and personality (extraversion vs. introversion) and their interaction on key *voice* characteristics that should covary with laryngeal activation, and that is perceived vocal effort, voice fundamental frequency (F_0) and voice intensity.

Overview:

Perceived vocal effort was investigated in parallel as the *subjective* counterpart to objective extralaryngeal SEMG activity. This approach provided a window into the sources of commonly self-reported vocal effort or vocal fatigue in patients with voice disorders. Specifically, it was investigated if the exposure to a stressor merely altered the *perception* of laryngeal functioning as a function of personality or if the perceptions were in agreement with SEMG changes. For example, it is widely known in the psychology literature that neuroticism can magnify bodily perceptions and increase symptom reporting (Costa & McCrae, 1985a; Suls & Martin, 2005). Moreover, changes in F_0 and voice intensity were tracked, because acoustic analyses in emotion

research have pointed to an increase in both F_0 and voice intensity as global indicators of stress and arousal (Juslin & Scherer, 2005). However, acoustic changes during a specific stressor such as public speaking were rarely, if at all, investigated as a function of personality. Finally, it was important to track those acoustic parameters, in order to examine if any increases in SEMG activity produced changes in voice output parameters that are regularly and readily assessed clinically (in difference to SEMG).

Experimental question:

Will there be a significant interaction between the effects of experimental phase (baseline speech, public speaking, recovery speech) and personality (extraversion vs. introversion) on changes in voice characteristics (perceived vocal effort; F_0; voice intensity)?

Table 2-3. Dependent and independent variables for Specific Aim 2

Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
Vocal effort	Phase	Personality (extraversion/introversion)	All
F_0	Phase	Personality (extraversion/introversion)	All
Intensity	Phase	Personality (extraversion/introversion)	All

H_0: There will be no significant interaction between the effects of phase and personality on vocal effort, F_0, and voice intensity. A null result would fail to produce evidence consistent with the model's prediction that exposure to a stressor will alter the participants' perception of vocal effort as well as F_0 and voice intensity as a function of personality, i.e. the prediction of differential stress reactivity with respect to perceived and objective voice characteristics.

H_1: There will be a significant interaction between the effects of phase and personality on vocal effort, F_0, and voice intensity. Specifically, based on logic expressed in the theory of the dispositional bases for vocal nodules and functional dysphonia (Roy & Bless, 2000a) and pilot

data, perceived vocal effort will increase the most for introverts during the stressor phase as a result of the hypothesized experience of laryngeal muscle tension. F_0 and voice intensity will increase the most for extraverts during the stressor phase (behavioral activation). Contrary to accounts in the literature that F_0 and voice intensity increase as global indicators of stress (Juslin & Scherer, 2005), in line with Roy and Bless's theory (Roy & Bless, 2000a) and based on logic, it was hypothesized that F_0 and voice intensity will actually decrease or stay the same under stress in the case of introverts. Presumably, this would be the result of behavioral inhibition and laryngeal depression as opposed to behavioral activation (Colton, et al., 2006). Last, it has been found that acoustic parameters may shift in different directions depending on whether one considers the effects of stress/arousal or fear/anxiety (see Table 2-1). Redirected to the present context, potential interindividual differences may be explained, because introverts may perceive fear whereas extraverts may perceive arousal in response to the public speaking stressor. Overall, positive findings would add support to the psychobiological model under investigation.

2.7.4 Exploratory outcomes (III)

These questions attempted to garner preliminary information about pathways that *mediated* relations between the stressor and peripheral responses. The ultimate goal, for which the present data provided a platform, was to provide information about the validity and utility of the proposed model, across all levels of its multi-tiered structure (Figure 2-6). The present study gathered data relevant to future studies with that goal. In the present context, pairs of model levels – as opposed to the model as a whole-- were examined for their potential validity (e.g. Levels A and B (IIIa); Levels A and C (IIIb); Levels A and D (I); Levels A and E (II); etc.). The

exception was the comparison between Levels B and C of the model, which was not explored here. Conceptually, the candidates of interest in this study, to explore mechanisms that mediated relations between a person-by-situation interaction, specifically stressor exposure (Level A) and laryngeal/vocal reaction (Levels D and E), were subdivided into (1) physiological (Level C of the model), (2) emotional (Level B), and (3) cognitive (Level B) mechanisms. The ANS represented the physiological mechanism under study and was (incompletely) reflected by systolic blood pressure (SBP; Level C). State negative emotions, fear of public speaking, and rumination represented the emotional and cognitive mechanisms under investigation (Level B). Besides the question of whether exposure to a stressor affected physiological, emotional, and cognitive parameters, the corollary question was whether these variables also influenced SEMG by acting as *mediators.*

- A → ***B*** → D (Level B: negative emotions; fear of public speaking)
- A → ***C*** → D (Level C: SBP)

If so, there will be motivation to pursue a multi-tiered causal model at a preliminary level using Structural Equation Modeling and further empirical studies, based on the findings (see Chapter five, future directions).

Physiological mechanism (assessment of model Levels A versus C, IIIb):

Specific aim 3 (addresses goal 3, IIIb):

To assess the influence of the manipulation of exposure to a stressor (baseline speech, rest, anticipation, public speaking, recovery, and recovery speech) and personality (extraversion vss. introversion) and their interaction on systolic blood pressure (SBP).

Overview:

In the voice disorders literature, the ANS has been frequently assumed as a relevant mediator for increased laryngeal tension during stress. However this assumption has not yet been adequately investigated (Dietrich & Verdolini Abbott, 2008). At the same time, it is known that a stress reactivity protocol generally increases autonomically mediated cardiovascular activity (Cacioppo, et al., 1998; Gruenewald, et al., 2004; Kirschbaum, et al., 1993) and thus it was a suitable set-up to examine the possible relation of cardiovascular reactivity with extralaryngeal tension. SBP has been chosen as dependent measure, because it is a common marker of autonomic arousal albeit an indirect and vague one. SBP represents influences from *both* the parasympathetic and sympathetic nervous systems, but the sympathetic influences outweigh the parasympathetic ones (Cacioppo, et al., 1998). At this stage of inquiry, the investigation of SBP in relation to laryngeal tension under exposure to a stressor guided further thinking.

Experimental question:

Will there be a significant interaction between the effects of a stressor condition (baseline speech, rest, anticipation, public speaking, recovery, and recovery speech) and personality (extraversion vs. introversion) on SBP?

Table 2-4. Dependent and independent variables for Specific Aim 3

Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
SBP	Phase	Personality (extraversion/introversion)	All

H_0: There will be no significant interaction between the effects of phase and personality on SBP. The null hypothesis was supported, because the health psychology literature indicates that interindividual differences in cardiovascular reactivity as a function of personality are minimal at best (Jorgensen, et al., 1996). While the interaction was expected to be non-significant (the focus

of this project), a main effect of phase was expected. That is, based on previous stress reactivity research using the *TSST* or similar social-evaluative stress protocols (Gruenewald, et al., 2004; Kirschbaum, et al., 1993), it was anticipated that results will show significantly higher SBP during the stressor phase. A null result for the interaction would fail to produce evidence consistent with the current model's assumption that stressor-induced differential increases in SBP as a function of personality may be correlated with SEMG activity during the stressor phase.

H_1: There will be a significant interaction between the effects of phase and personality on SBP. Although the null hypothesis was favored due to the inconsistent literature, a direction for the alternative hypothesis was proposed. Specifically, it was hypothesized that introverted participants will exhibit significantly greater SBP than extraverted participants under stressor exposure. A significant effect would provide the basis for testing SBP as a mediator between personality and SEMG activity during exposure to stress as outlined below.

Emotional and cognitive mechanisms (assessment of model Levels A versus B, IIIa):

Negative emotions:

Specific aim 4 (addresses goal 3, IIIa):

To assess the influence of the manipulation of exposure to a stressor (rest, public speaking, recovery) and personality (extraversion vs. introversion), and their interaction on negative emotion.

Overview:

As discussed previously, "stress" is a general concept and can be variably defined (Cohen, et al., 1995). More precisely, "distress" often comprises negative emotional and cognitive states, in particular negative emotions such as fear. Individuals with personality traits that make them

susceptible to negative emotionality, i.e. neuroticism, or with traits that make them vulnerable to the social-evaluative stressor, e.g. introversion or social anxiety, would naturally react with stronger and longer-lasting negative emotions (Dickerson, et al., 2004; Suls & Martin, 2005). In fact, "stress emotions" are often assessed in lieu of measuring "perceived stress." Capturing *perceptions* was critical in this stress reactivity protocol, because "stress" is not only a physiological response, but a complex *psycho*biological response and emotional reactivity may or may not parallel physiological reactivity. At the same time, it was possible to confirm if the stressor manipulation was successful.

Experimental question:

Will there be a significant interaction between the effects of phase and personality on negative emotions?

Table 2-5. Dependent and independent variables for Specific Aim 4

Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
Negative emotions	Phase	Personality (extraversion/introversion)	All

H_0: There will be no significant interaction between the effects of phase and personality on negative emotions. A null result would fail to produce evidence consistent with the model's prediction that exposure to a stressor will differentially increase the participants' negative emotional state as a function of personality. A main effect of stressor on negative emotions, however, was expected based on existing literature on similar stress reactivity protocols (Gruenewald, et al., 2004). A failure to detect a main effect of experimental phase will invalidate the effectiveness of the public speaking task as a stressor.

H_1: There will be a significant interaction between the effects of phase and personality on negative emotions. Specifically, based on pilot data and derived from the existing literature, increases in negative emotions during the stressor phase were expected to be greatest for introverts (Larsen & Ketelaar, 1991; Roy & Bless, 2000a). The results would be consistent with the model's central prediction that *perceived* distress is the result of a person-by-situation interaction, specifically the susceptibility to perceive threat in a stressful social-evaluative situation. Positive results would invite follow-up research focusing on the role of negative emotion as a mediator for stressor-induced SEMG changes in line with the proposed psychobiological model.

Fear of public speaking (assessment of model Levels A versus B, IIIa):

Specific aim 5 (addresses goal 3, IIIa):

To assess the influence of personality (extraversion vs. introversion) on fear of public speaking.

Overview:

As discussed in the previous section on negative emotions as a dependent measure, distress in form of negative emotions is common as a result of perceived stress. However, an increase in fear, for instance, does not instantly reveal the source of fear. Hence, as the stressor manipulation involved a public speaking task, it was appropriate to include a measure of *fear of public speaking* to narrow down the source of potential negative emotions. In fact, fear of public speaking or speech phobia is a common concept assessed in social anxiety or social phobia questionnaires (B.J. Ries, et al., 1998). According to Social Self Preservation Theory (Dickerson, et al., 2004), especially individuals, high in social anxiety or low in self-esteem will be vulnerable to threats to the social self as occurring during the social-evaluative stressor.

Experimental question:

Will there be a significant difference in fear of public speaking as a function of personality (extraversion vs. introversion)?

Table 2-6. Dependent and independent variables for Specific Aim 5

Dependent Variable	**Independent Variable 1**
Fear of public speaking	Personality (extraversion/introversion)

Relations of the hypotheses to the proposed psychobiological model under investigation were similar to the ones explained in the previous section on negative emotions.

H_0: There will be no significant difference in fear of public speaking as a function of personality.

H_1: There will be a significant difference in fear of public speaking as a function of personality. Specifically, based on existing literature (especially social introversion literature), increases in speech anxiety will be relatively greater for introverts although introversion does not necessarily have to go hand in hand with speech or social anxiety (MacIntyre & Thivierge, 1995; McCroskey, 1977). Positive results would invite follow-up research focusing on the role of state speech anxiety as a mediator for stressor-induced SEMG changes in line with the proposed psychobiological model.

Rumination (assessment of model Levels A and B, IIIa):

Specific aim 6 (addresses goal 3, IIIa):

To assess the influence of personality (extraversion vs. introversion) on rumination.

Overview:

A current trend in health psychology is the renewed focus on the *recovery period* in addition to a much highlighted stress *reactivity* phase (Linden, et al., 1997). Existing research indicates that anticipation of stress or delayed emotional or cognitive recovery from stress may sustain

physiological stress responses. This notion is an important one to be investigated in future research, due to its implications for voice disorders. In other words, the perception of chronic and sustained stress may sustain heightened physiological activity such as increased or imbalanced extralaryngeal SEMG activity, which in turn may put individuals at risk for vocal symptoms or voice disorders. Thus, the additional spotlight on rumination aided in collecting data on this important piece of the causal chain linking perceived stress and voice disorders.

Experimental question:

Will there be a significant difference in rumination as a function of personality (extraversion vs. introversion)?

Table 2-7. Dependent and independent variables for Specific Aim 6

Dependent Variable	**Independent Variable 1**
Rumination	Personality (extraversion/introversion)

H_0: There will be no significant difference in rumination as a function of personality. A null result would fail to produce evidence consistent with the model's prediction that exposure to a stressor will delay recovery from distress as a function of personality. Such a result would weaken the assumption within the proposed psychobiological model that delayed recovery may be a potentially relevant factor in the development of muscle tension dysphonia.

H_1: There will be a significant difference in rumination as a function of personality. Specifically, based on pilot data and existing literature, increases in rumination were expected to be relatively greater for introverts and will covary with negative emotions experienced during the stressor phase (Siegle, Moore, & Thase, 2004). The results would be consistent with the model's prediction that differential cognitive stress reactivity exists, extending into the recovery phase, as

a function of personality. Positive results would support the idea that chronic stress may be a relevant part of the model in relation to muscle tension dysphonia. Of note, it is conceivable that such delayed cognitive and emotional reactivity may come with or without concurrent delayed physiological recovery (e.g. sustained increased SEMG activity).

Potential mediation of foregoing variables for changes in laryngeal behavior (assessment of model levels A, B, C, and D, IIIa-d):

Specific aim 7 (addresses goal 3, IIIa-d):

To assess the role of the variables SBP, negative emotions, and fear of public speaking as potential mediators for changes in SEMG activity in extraverts versus introverts during exposure to a stressor. Follow-up mediation analyses were only performed on the variables where significant effects on SEMG were established as a function of personality and phase (Specific Aims 3-6).

Overview:

Ultimately, the goal was to illuminate the causal chain that links personality traits with increased (magnitude), altered (pattern), or sustained (magnitude during recovery) extralaryngeal tension under exposure to a stressor. Candidates of interest for mechanisms in light of a person-by-stressful situation interaction were autonomically mediated cardiovascular reactivity (SBP), negative emotions, and fear of public speaking. Evidence-based knowledge of mechanisms linking personality and perceived stress to laryngeal changes is severely lacking but would aid in refining and tailoring voice therapy programs for individuals complaining of muscle tension dysphonias (e.g., focus on biofeedback versus focus on perception of stress or a combination thereof). Although the effects of exposure to a stressor on cardiovascular, emotional, and cognitive reactivity have generally been documented, it is new territory to correlate such changes

with laryngeal muscle activity under stress. It was important to obtain preliminary data on the links between potential mechanisms and extralaryngeal activity to guide future research. For example, although cardiovascular reactivity is an integral part of any psychobiological stress reactivity model, it may not have direct effects on laryngeal activity under stress.

Experimental question:

Will the variables SBP, negative emotions, and fear of public speaking each mediate relations between phase and personality and their interaction (where appropriate) on SEMG?

Table 2-8. Dependent and independent variables and covariates for Specific Aim 7

Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Covariate**	**Interactions**
SEMG (% MVC)	Phase	Personality (extraversion/introversion)	SBP	All
SEMG (% MVC)	Phase	Personality (extraversion/introversion)	Negative emotions	All
SEMG (% MVC)	Phase	Personality (extraversion/introversion)	Fear of public speaking	All

H_0: None of the variables SBP, negative emotions, and fear of public speaking will be a significant mediator for relations between personality, phase, and their interaction (where appropriate) on SEMG. A null result would be at odds with the model's choices of potential mediators of stressor-induced altered SEMG activity as a function of personality. As a consequence, alternative mediators would have to be theoretically and experimentally explored.

H_1: Based on pilot data and theoretical input, negative emotions and fear of public speaking will be significant mediators of predicted SEMG activity during the stressor for introverts (Roy, et al., 2000a; van Mersbergen, et al., 2008). SBP will not be a significant mediator although numerical tendencies may exist that may warrant examination. As mentioned before, the SBP differences between personality groups such as introversion versus extraversion are traditionally

small. Significant results for negative emotions and speech anxiety as mediators would be consistent with the model's prediction that *perceived* distress may play a central role in the proposed psychobiological model in driving differential extralaryngeal stress reactivity as a function of personality as opposed to cardiovascular mediation. Such results would strengthen the theory of the dispositional bases of vocal nodules and functional dysphonia (Roy & Bless, 2000a) as well as the currently proposed model and would invite in-depth research.

3.0 RESEARCH METHODS

3.1 EXPERIMENTAL DESIGN

The primary experimental design was a 2x6 mixed-model design. The experimental design for the secondary outcomes was a 2x3 mixed-model design. Exploratory follow-up analyses were used to investigate potential mediators for the effects. The dependent and independent variables are listed in Table 3-1. Two experimental groups (between-subjects independent variable personality with two levels, extraversion versus introversion) were studied before, during, and after exposure to a social-evaluative stressor (within-subjects variable experimental phase: baseline speech, rest, stressor anticipation, stressor (public speaking), recovery, and recovery speech, i.e. repeated baseline speech). The stressor was *modeled after* the *Trier Social Stress Test* (TSST; Kirschbaum, et al., 1993), which is a well-established laboratory protocol to induce moderate psychosocial stress that typically produces significant increases in cardiovascular parameters and in subjective stress ratings. The stress involved a *public speaking* task performed under social-evaluative conditions and time pressure and was preceded by an anticipation period functioning as a *nonverbal stressor*. The design was complemented by a rest period before and a recovery period after the stressor and by a baseline non-stressor speech task before the stress protocol proper and a recovery speech (repeated baseline speech) at the end of the stress protocol all representing reference points. The primary dependent variable was surface electromyography

(SEMG: submental, infrahyoid, anterior tibialis). Secondary outcome variables were perceived vocal effort, voice fundamental frequency (F_0), and voice intensity (dB). Finally, potential mediators under investigation were systolic blood pressure (SBP), negative emotional state, and state fear of public speaking.

Table 3-1. Experimental variables

Primary outcome (I) (Specific Aim 1)			
Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
Submental SEMG (%MVC)	Phase[1]	Personality (extraversion vs. introversion)	All
Infrahyoid SEMG (%MVC)	Phase[1]	Personality (extraversion vs. introversion)	All
Anterior tibialis SEMG (%MVC)	Phase[1]	Personality (extraversion vs. introversion)	All
Secondary outcomes (II) (Specific Aim 2)			
Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
Vocal effort	Phase[2]	Personality (extraversion vs. introversion)	All
F_0	Phase[2]	Personality (extraversion vs. introversion)	All
Intensity (dB)	Phase[2]	Personality (extraversion vs. introversion)	All
Exploratory outcomes (III) (Specific Aims 3-6)			
Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
SBP	Phase[1]	Personality (extraversion vs. introversion)	All
Negative emotional state	Phase[3]	Personality (extraversion vs. introversion)	All
Fear of public speaking	N/A	Personality (extraversion vs. introversion)	N/A
Rumination	N/A	Personality (extraversion vs. introversion)	N/A
Examination of mediation (Specific Aim 7)			
Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Covariate**
SEMG (%MVC)	Phase	Personality (extraversion vs. introversion)	SBP
SEMG (%MVC)	Phase	Personality (extraversion vs. introversion)	Negative emotions
SEMG (%MVC)	Phase	Personality (extraversion vs. introversion)	Fear of public speaking

Phase[1]: baseline speech, rest, anticipation, stressor (public speaking), recovery, recovery speech (repeated baseline speech)
Phase[2]: baseline speech, stressor (public speaking), recovery speech
Phase[3]: rest, stressor, recovery

3.2 PARTICIPANTS

All participants were vocally normal female adults between the ages of 18 and 35 years divided into two groups: extraversion and introversion. A total of n = 54 participants were enrolled with n = 27 per group. The mean age for the extraversion group was 23.26 (SD = 3.77, range 18-32 years) while the mean age for the introversion group was 22.67 (SD = 3.50, range 19-35 years). The distribution of race and ethnicity was fairly similar between groups, however, racial diversity was more prominent in the group with extraversion (Table 3-2). Participants were primarily recruited from the student population in order to form coherent social groups and to reduce extraneous variability in the data. In particular, it is known that laryngeal and cardiovascular aging processes may introduce variability in the data (Colton, et al., 2006; Guyton & Hall, 2005; Kahane, 1987). Only female adults were enrolled because the research questions addressed were relevant to only one gender in the context of voice disorders research. Most literature indicates that women are about twice as likely to experience voice problems, or at least to report them and seek treatment, as compared to men (M. K. Miller & Verdolini, 1995). All participants passed the inclusion and exclusion criteria listed below. Inclusion and exclusion criteria are also specified in table-format in Appendix A with detailed rationales.

Table 3-2. Distribution of race and ethnicity in the study sample

Race and ethnicity	Extraversion	Introversion
White	18 (66.7%)	22 (81.5%)
African-American	4 (14.8%)	2 (7.4%)
Asian	4 (14.8%)	2 (7.4%)
More than one race (White and American Indian/Alaska Native)	1 (3.7%)	1 (3.7%)
Hispanic	0	0

3.2.1 Inclusion criteria

General *inclusion* criteria included female adults, ages 18 to 35 years. The participants were native speakers of English with normal speech and language skills as determined independently by the Principal Investigator (PI) and a second certified speech-language pathologist based on conversational speech and reading. Similarly, the participant's speaking voice was rated as normal by the PI and a second certified speech-language pathologist based on independent auditory-perceptual ratings of conversational speech, reading, and voice production (*Consensus Auditory Perceptual Evaluation of Voice* (*CAPE-V*); Rainbow Passage; Appendix B). Of note, a distinction was made between normal voice and healthy voice. Although glottal fry is generally not considered a healthy phonation pattern yet a common one (Gottliebson, Lee, Weinrich, & Sanders, 2007), participants with glottal fry in their voices were included as long as other vocal inclusion and exclusion criteria were met.

Specific inclusion criteria for the selection of personality-specific subgroups were based on scores on the *Eysenck Personality Questionnaire – Revised* (*EPQ-R*) (Eysenck & Eysenck, 1994) obtained during the screening, but additional data were also collected with the

Multidimensional Personality Questionnaire – Brief Form (*MPQ-BF*) (Patrick, et al., 2002) obtained during the screening as well (see also the section on measures for more information). Requiring participants to score at least 0.5 *SD* above or below the norm was necessary to ensure a minimum of appropriate differentiation between the groups (Figure 3-1).

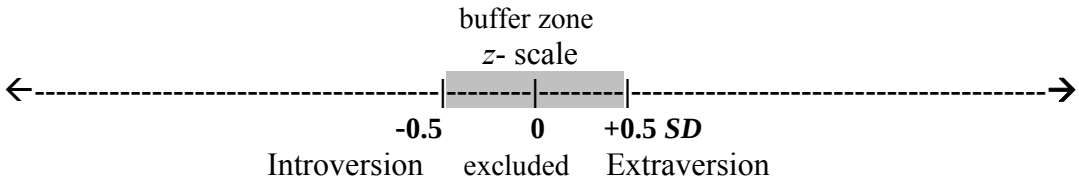


Figure 3-1. Inclusion criteria personality

3.2.2 Exclusion criteria

All participants complied with the following general *exclusion* criteria based on information obtained from participants' self-reports if not otherwise noted: current smoking; acute or chronic upper respiratory infection at time of testing; history of cardiac, pulmonary, or neurological problems, specifically history or symptoms of systemic diseases known to affect the nervous or endocrine systems; allergies, laryngopharyngeal reflux disease, and asthma, which both the participant and the PI concurred *did affect* the voice at the time of study participation; the use of medications known to affect the nervous and endocrine systems in the week preceding the experimental session (contraceptives not included); controlled and uncontrolled hypertension per self-report and as assessed during screening (> 140 mmHg systolic and/or 90 mmHg diastolic blood pressure; American Heart Association); current psychiatric treatment including medications for treatment of a psychiatric disorder; moderate depression (score $\geq$ 16) as

determined by the *Beck Depression Inventory* (*BDI*) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961); hearing loss to 25 dB at 1000, 2000, 3000, and 4000 Hz as assessed by the PI; body mass index (BMI) in the obese range based on participants' self-reports of height and weight and as compared to tables provided by the NIH Department of Health and Human Services; history of laryngeal trauma or surgery; fatty or scarred anterior neck that prevented the identification of laryngeal landmarks; a current or lifetime history of a voice problem or voice disorder lasting two weeks or more; complaint of vocal effort (Direct Magnitude Estimation >100) or vocal fatigue while speaking including positive answers to questions #14 and #20 of the *Voice Handicap Index* (*VHI*) (Jacobson, et al., 1997) related to strain and effort during voice production; and previous voice therapy or professional singing, voice, or speech training.

Of note, no cut-off score was used for the *VHI*. Rather specific answers pertaining to vocal effort were screened as noted above and the Gestalt of *all* answers was judged and discussed as needed as part of the voice screening. All included, an informed decision on a participant's normal vocal status was made.

3.3 MEASURES

3.3.1 Independent variables

3.3.1.1 Personality

The *Eysenck Personality Questionnaire* (*EPQ*) (Eysenck, 1967) is one of the most widely used inventories in personality research today. It is a 90-item self-report questionnaire and is scored

on one validity scale and three personality scales. It has been used to identify three broad personality dimensions: extraversion, neuroticism, and psychoticism. The *EPQ* scales have been shown to possess good reliability (Eysenck & Eysenck, 1975) and validity (Eysenck & Eysenck, 1971, 1970; Verma & Eysenck, 1973). The *EPQ-Revised* was used during the screening as a traditional measure of extraversion and introversion (Eysenck & Eysenck, 1994). In addition, data on the *Multidimensional Personality Questionnaire – Brief Form* (*MPQ-BF*) were collected.

The *MPQ-BF* (Patrick, et al., 2002) is a self-report personality instrument designed to assess structural levels conceptualized in psychobiological terms such as Positive Emotionality, Negative Emotionality, and Constraint. The *MPQ-BF* consists of 155 self-reference statements and has been found to be an enhanced research tool for the investigation of the genetic, neurobiological, and psychological substrates of personality (Patrick, et al., 2002). The rationale for the development of the brief form was to facilitate the administration of multiple measures in psychophysiological studies - among other reasons - while maximally preserving the original structure and content of the full-version *MPQ* (Tellegen, 1982). The brief from has been shown to possess strong psychometric properties similar to the full version and even slightly superior to other available measures of normal personality such as the *EPQ* and *NEO* Big Five (Costa & McCrae, 1985b). Cronbach's α for the *MPQ-BF* primary trait scales ranged from .74 to .84.

3.3.1.2 Experimental phase (stressor manipulation)

The stress reactivity protocol was largely based on the *Trier Social Stress Test (TSST)* (Kirschbaum, et al., 1993). The *TSST* is a well-established laboratory protocol to induce moderate psychosocial stress, which in turn produces significant increases in cardiovascular parameters and in subjective stress ratings. Specifically, the stressor involved an impromptu

public speaking task performed under social-evaluative conditions and time pressure. Social-evaluative stress was created by the presence of two experimenters and concurrent videotaping. Various female (Under)graduate research assistants trained in the protocol presented the stressor task to the participants. The assistant was unfamiliar to the participant until the day of the experiment and maintained social distance during the experiment. During the stressor phase, the PI was outside the booth controlling the recordings, but within sight of the participant as well, because the booth had a glass door and glass window. The script for the stressor presentation is included in Appendix C. In addition to the public speaking task, the anticipation phase for the speech stressor was used to represent a speech-specific but *nonverbal* stressor.

The protocol was further complemented by adequate reference points for the stressor. Both tonic and phasic extralaryngeal muscle activity was captured throughout the protocol. The stressor was preceded by a rest phase and followed by a recovery phase, both in silence. As for active speech, a baseline speech and an identical recovery speech (repeated baseline speech) were used as reference points for stressed speech. The purpose was to elicit speech in the absence of acute stress for comparison purposes. Tasks from the *Consensus Auditory-Perceptual Evaluation of Voice* (CAPE-V) (American Speech-Language-Hearing Association Division 3, Voice and Voice Disorders, 2003; Appendix B) together with the phonetically balanced Rainbow Passage (Fairbanks, 1960) were used for the baseline and recovery speech and partly for the stressed speech. The reasoning was two-fold. First, the combination of the *CAPE-V* and the Rainbow Passage allowed for a speech sample that was roughly of similar length (5 min.) as the public speech (Appendix B). Secondly, a portion of the *CAPE-V* (sustained /a/ and all-voiced sentence “we were away a year ago”) was elicited *immediately* prior to the onset of public speaking in order to obtain a phonetically controlled speech sample under *maximum stress* that

could be compared to the baseline speeches. Of note, the same speech tasks were also used during the voice screening.

3.3.2 Dependent variables

3.3.2.1 Physiological measures

Surface EMG (primary)

Surface EMG electrodes were placed on the anterior neck and the leg. A ground electrode was placed on the bony prominence of the elbow (De Luca, 2002). Activation from two different surface extralaryngeal muscle groups was recorded to achieve an exploratory sampling of laryngeal elevator muscles (suprahyoid) versus laryngeal depressors (infrahyoid) related to voice production. Although the extralaryngeal muscles primarily position and support the larynx, they also have been shown to influence voice F_0, specifically an increase in F_0 with thyrohyoid muscle activity (assuming laryngeal elevation when the hyoid is fixed) and a decrease in F_0 with sternothyroid activity (laryngeal lowering) (W.R. Zemlin, 1998). However, the thyrohyoid muscle is covered by the omohyoid and sternohyoid muscles and also extremely small and thus cannot be exclusively sampled. Laryngeal electrodes were positioned as previously described in the literature (Ding, Larson, Logemann, & Rademaker, 2002; Hočevar-Boltežar, et al., 1998; van Mersbergen, et al., 2008) and parallel with the direction of the muscle fibers. Specifically, two electrodes for the submental muscle group were placed circa 1 cm from the midline in the left submandibular area and superior to the hyoid (Colton, et al., 2006; Ding, et al., 2002; van Boxtel, 2001; E. M. Yiu, Verdolini, & Chow, 2005). This location recorded activity from the anterior

belly of the digastric, mylohyoid, and geniohyoid. The infrahyoid electrodes were placed on the left side over the thyroid cartilage circa 1 cm off midline. This location recorded activity mainly from the sternohyoid, omohyoid, and thyrohyoid (Colton, et al., 2006; Ding, et al., 2002). It is acknowledged that muscle activity of the platysma may have been recorded as well (van Boxtel, 2001). In general, it cannot be overemphasized that SEMG does not allow for the sampling of individual extralaryngeal muscles in the same way as it is possible for limb muscles due to small muscle sizes and the multilayered structure of extralaryngeal muscles. Instead SEMG activity from muscle *groups* was captured. In the end, exact electrode positions varied depending on each participant's laryngeal anatomy. All electrode cables were taped to a participant's shirt or pants in order to avoid movement artifact. The absence of movement artifact was further checked by having the participants rotate their head during set-up.

Two electrodes were placed on the right leg to sample muscle activity from the anterior tibialis. The first electrode was placed one third lead line length from the patella (lead line length equals distance from lower margin of patella to lateral ankle) (Soderberg, 1992). The data from the leg were used as control data from a muscle site that is not related to speech production.

Gel-filled surface silver-silver chloride electrodes with a 5 mm diameter detection surface were applied with double-sided adhesive disks after cleaning skin with rubbing alcohol. Bipolar recordings were obtained based on a 1.5 cm distance between electrode centers (Fridlund & Cacioppo, 1986). All data were acquired following guidelines for EMG recordings (Fridlund & Cacioppo, 1986; van Boxtel, 2001). Guidelines for EMG recordings have been put forward in the literature that, if followed, should substantially increase reliability (Fridlund & Cacioppo, 1986). The guidelines specify the technical aspects of recordings and the adequate positioning of electrodes. The electrodes were exclusively placed by the PI who is a certified Speech-Language

Pathologist with experience in laryngeal anatomy and physiology. The adequate placement of the laryngeal electrodes was verified before data were collected. Recordings for a swallow (Ding, et al., 2002), sustained /i/ with high pitch (submental) (Borden, Harris, & Raphael, 2003), and sustained /u/ with low pitch (infrahyoid) (Borden, et al., 2003) were screened for the activation of the targeted muscle groups. To test muscle activity of the anterior tibialis, the participant was asked to tense the leg muscle.

The extralaryngeal and limb raw SEMG data were normalized relative to the participant's (sub)maximal voluntary contraction (MVC) of the targeted muscles (isometric force), i.e. a reference level. This procedure is recommended in order to allow for comparisons between muscles and muscle groups (e.g., muscle geometry), speakers (e.g., subcutaneous fat), and time points (Fridlund & Cacioppo, 1986; Lundberg, 2002). However, it has also been noted that 100% MVC frequently achieves lower intraclass correlation coefficients than submaximal MVC, e.g., 50% (De Luca, 1997; Soderberg, 1992) (Ma, personal communication, April 7, 2008). Further, the orofacial and laryngeal muscles usually operate below 20% MVC and rarely above 50% (Netsell, 1982). Therefore, the focus was on submaximal voluntary contractions although maximal recordings were obtained as well. Visual feedback of the MVC SEMG recordings was provided for the participants, the participants were motivated to perform their best, and they received practice trials. Maximal EMG tasks included an isometric resistive mandible depression task (depression of jaw against manual resistance; submental and infrahyoid) (Juul-Kristensen, Laursen, Pilegaard, & Jensen, 2004; W. R. Zemlin, 1998), and dorsiflexion of the foot against manual resistance (LeVeau & Andersson, 1992). Each MVC was held five seconds and repeated three times per site with intermittent one-minute recovery periods to avoid muscle fatigue (De Luca, 1997; LeVeau & Andersson, 1992; Soderberg, 1992). For the 50% MVC participants were

asked to subjectively embody half the effort expended during the 100% MVC. The participants were verbally introduced to a self-rated scale where the 100% force exerted for MVC was given an arbitrary value of ten units and the participants were required to exert a force of five units for the 50% MVC (Ma, personal communication, April 7, 2008).

Cardiovascular reactivity (exploratory)

Blood pressure, in particular systolic blood pressure (SBP), was measured as an indicator of physiological arousal. First, an increase in SBP validates the effectiveness of the experimental task as a stressor and secondly, SBP acts as an *indirect* measure of activation of the sympathetic nervous system. While both heart rate and SBP are the result of influences of both the sympathetic and the parasympathetic nervous system on the sinoatrial node, SBP is a relatively better indicator of sympathetic nervous system activation (Allen, 2000). For example, large increases in heart rate during public speaking have been shown to be driven by beta-adrenergic and mixed beta- and alpha-adrenergic reactivity and a large parasympathetic withdrawal is thought to be typical for public speaking (Allen, 2000). Automated measurements of blood pressure were recorded every 90 seconds throughout the protocol with a Critikon Dinamap 8100 Vital Signs Monitor suitable for blood pressure and heart rate measurements. Measurements followed the guidelines provided by Shapiro et al. (1996).

3.3.2.2 Emotional and cognitive measures (exploratory)

Emotional state

Participants' changes in affective states during the protocol were tracked using the *Positive And Negative Affect Schedule-Expanded Form* (*PANAS-X*) (Watson & Clark, 1994). The *PANAS-X* is

a reliable 60-item questionnaire rated on a five-point scale to assess dimensions of emotional experience including valence and content (Cronbach's α = .85-.88 (general scales) and .72-.93 (specific scales)). A score was calculated for general *negative* emotion (fear, hostility, guilt) for the rest, stressor, and recovery phases in order to represent the degree of distress experienced.

State speech anxiety

Acute *fear of public speaking* was separately assessed to explore the nature of negative affect experienced as a consequence of the speech stressor. Fear of public speaking or speech phobia is a common concept assessed in social anxiety or social phobia questionnaires (B.J. Ries, et al., 1998). The *Personal Report of Confidence as a Speaker* (*PRCS*) (Paul, 1966) was used to assess the degree of confidence and fear of public speaking experienced by the participants during the speech. Participants answered 30 true or false statements regarding emotions, cognitions, and perceptions of bodily changes during the public speaking task. The instrument demonstrates good concurrent, convergent and predictive validity (Lombardo, 1988; Tarico, van Velzen, & Altmaier, 1986) and adequate internal consistency (Daly, 1978).

Rumination

A rumination questionnaire was used to assess negative cognitive states during the recovery phase as a result of the stressor (Marsland, unpublished measure). The questionnaire contains five questions and each question is rated on a seven-point scale. This rating scale has been adapted from other rumination questionnaires to fit the specifics of a stress reactivity protocol. Reliability and validity data are not available at this point but forthcoming.

3.3.2.3 Voice production and output (secondary)

Perceived vocal effort

Participants judged the degree of vocal effort experienced after any of the speech tasks (baseline speech, public speaking stressor, recovery speech). The participants were instructed to use the Direct Magnitude Estimation method where 100 represents comfortable amount of effort, 200 represents twice as much effort as comfortable and so forth or any number in between (Wright & Colton, 1972). Those vocal effort ratings were the subjective counterpart of objective measures of SEMG activity during the protocol.

Acoustics

A professional headmounted condenser hypercardiod microphone (Shure Beta 54) was used to transduce audio signals during any verbal phases. Voice pitch derived from fundamental frequency in Hertz (F_0) and voice intensity in dB SPL were extracted from audio recordings during experimental phases involving voice and speech (baseline speech, public speaking, recovery speech). Production of sustained vowels and of an all-voiced standard sentence was used as the basis for acoustic analyses using Praat software version 5.0.32. The acquisition of acoustic voice signals followed published recommendations, i.e. a mouth-to-microphone distance of 3 cm and 45^0 to 90^0 off-axis positioning of the microphone (Titze, 1995). Adobe Audition software (version 1.5) was used for recordings with a sampling rate of 44.1 kHz and a 16-bit resolution (Deliyski, Shaw, & Evans, 2005). Loudness calibration was performed and recorded.

3.4 PROCEDURES

Participants were recruited by way of IRB-approved advertisements in University settings (University of Pittsburgh, University of Pittsburgh Medical Center (UPMC), Carnegie Mellon University (CMU), Chatham University, Carlow University) and community settings (Post Gazette, Tribune Review, Craigslist). All participants provided informed consent before going through any screening or experimental procedures and the PI was the only person to consent the participants. An IRB-approved pre-screening over the phone was performed as needed. The sole purpose of the pre-screening was to obtain scores on the personality trait Positive Emotionality (extraversion vs. introversion) of the *MPQ-BF* (Patrick, et al., 2002). The goal was to increase the efficiency of screening for introverts, which are traditionally more difficult to recruit and to avoid having to invite everyone for a longer face-to-face screening.

In the consent form, the title of the study deviated from the title of the research protocol for the purpose of a planned deception. The title on the consent form was "Automatic Speech Recognition Based on Surface Electromyographic Signals Generated by Facial and Extralaryngeal Muscles." Collaborators from the School of Computer Science at Carnegie Mellon University (Tanja Schultz and Szu-Chen (Stan) Jou) were collaborators and interested in recording SEMG activity from facial and extralaryngeal muscles during speech-related activity for speech recognition purposes (Jou, Maier-Hein, Schultz, & Waibel, 2006; Jou, Schultz, & Waibel, 2005; Walliczek, Kraft, Jou, Schultz, & Waibel, 2006). Hence, the speech recognition part of the study was emphasized during the consent process and the psychobiological part of the study was disguised until the debriefing at the end of the experimental session. SEMG recordings related to the speech recognition part of the study, i.e. recordings from facial muscles and

additional extralaryngeal muscles, were *only* turned on during separate speech phases for that purpose labeled CS1 and CS2 in Table 3-2 (CS: computer science). During each CS1 (after baseline speech) and CS2 (after recovery speech) participants read 50 sentences that came up sequentially on a computer screen. The participant was able to pace the recordings. CS2 was a replication of the same set of sentences with the only difference that the second time the participant was asked to mouth them.

Prior to the laboratory stress session, participants received a sheet with instructions including the request to abstain from alcohol (prior 12 hours), exercise (above that required during daily routine), food (30 minutes), and caffeine (3 hours) (day of experiment) (Shapiro et al., 1996). However, the participants were instructed to drink 64 ounces of water throughout the day of the experiment, but no water was provided during the experiment. All recordings took place in the Department of Communication Science and Disorders at the University of Pittsburgh in an acoustically treated sound booth. Each experimental session had a duration of approximately 2 hours and the sessions were scheduled between 9:00AM and 7:00PM. Time of day for the experimental sessions was kept comparable across groups.

Upon arrival at the laboratory, the participants filled out three forms: (1) an intake form to check the participant's adherence with recommendations to abstain from food, caffeine, alcohol, and exercise for the specific time frames and to double check that the participant was not sick or had taken exclusionary medications over the past week; (2) the *VHI* in order to have a record of a participant's perception of her quality of life pertaining to voice *for the day of the experiment*; and (3) the *Liebowitz Social Anxiety Scale* (*LSAS*) (Liebowitz, 1987) in order to obtain a record of the participant's trait level of social anxiety for future reference (Table 3-2). Then, all electrodes for SEMG were applied as previously described. The participant was

comfortably seated in the recording room in a powered wheelchair outfitted with a foot and head rest (courtesy of Rosemarie Cooper, MPT, ATP, Wheelchair Seating Clinician, Director UPMC Center for Assistive Technology and Department of Rehabilitation Science Technology). The head was further stabilized with an elastic headband around the headrest and the forehead to avoid extraneous movements. An occluding blood pressure cuff was placed on the non-dominant arm for automated measurement of blood pressure. A headset microphone was positioned for audio recordings during voice and speech tasks.

Then, for experimental procedures proper, first, the maximum muscle activity for the laryngeal and limb SEMG sites was determined as described above. Participants were then asked to remain seated quietly for a two-minute recovery rest period before proceeding to baseline speech recordings. A non-stressed speech baseline was recorded while participants sustained vowels and read sentences and paragraphs as outlined in the *CAPE-V* and the phonetically balanced Rainbow Passage (Appendix B). SEMG was recorded continuously for the duration of each phase if not otherwise noted and automated blood pressure samples were made every 90 seconds throughout the protocol if not otherwise noted. Audio recordings were made concurrently with any SEMG recordings.

Then, the participant rested for ten minutes (baseline rest). Automated cardiovascular recordings started at minute 3:30 of rest to yield an average of five readings for baseline (Shapiro et al., 1996). Blood pressure data were collected throughout the entire stressor phase and during the seven minute recovery phase (five readings) as well as during the baseline speeches. During the stressor phase, the naïve participants were asked to perform a simulated public speaking task, consisting of two minutes of silent and mental preparation for a simulated job interview (Appendix C) followed by five minutes of speech under social-evaluative conditions. A brief

reference speech sample was requested immediately at the *outset* of the free speech (sustained /a/ and all-voiced sentence "we were away a year ago" extracted from the *CAPE-V*) to improve the comparison of stressed and non-stressed speech among participants and across phases. A seven-minute quiet recovery phase began after the conclusion of the stressed speech. After the recovery, a final recovery speech sample (repeated baseline speech) was elicited.

In addition, participants filled out a questionnaire to track negative emotions relative to the baseline rest, stressor, and recovery phases (*PANAS-X*). Participants also rated their fear of public speaking (*PRCS*) with respect to the public speaking stressor and finally, participants concluded with a rumination questionnaire. Vocal effort ratings were elicited in written form *immediately* after any speech tasks. However, in the interest of capturing the critical and short-lived transition from the end of the stressor to the start of recovery, the *PANAS-X* related to the stressor phase and *PRCS* were only handed out *after* recovery and the participant was asked to answer retrospectively. At the end of the session participants were debriefed using the debriefing protocol in Appendix D. Participants were paid $20 dollars for completing the experimental session and $10 for the screening. The timeline of the experiment can be found in Table 3-2. The full experimental lab protocol is available in Appendix D.

Table 3-3. Timeline of the experimental protocol

Pre-experiment	**Rest**	**Baseline speech**	**BASELINE**	**STRESS**	**RECOVERY**	**Recovery speech**
Questionnaires: *VHI* *LSAS* Set-up: SEMG BP Microphone, calibration MVC SEMG	No speech	*CAPE-V* Rainbow Passage Vocal effort rating CS1 (speech) Vocal effort rating	No speech; *PANAS-X*	Anticipation Sustained /a/, all-voiced sentence (portions *CAPE-V*) Public speaking Vocal effort rating	No speech *PANAS-X* (stressor rating) *PRCS* *PANAS-X* (recovery rating) Rumination questionnaire	*CAPE-V* Rainbow Passage Vocal effort rating CS2 (mouthing)
45 min.	2 min.	10 min.	10 min.	2+5 min.	7 min.	10 min.

3.5 INSTRUMENTATION AND DATA REDUCTION

All SEMG data were acquired by a computer-controlled 8-channel EMG data acquisition system (Varioport, Becker-Meditec, Germany). The recording interface has been developed by the Universität Karlsruhe (Technische Hochschule) (Brain Computer Interface). Technical specifications of the Varioport system include an amplification factor of 1170, 16 bits A/D conversion, a step size (resolution) of 0.033 microvolts per bit, and a frequency range of 0.9-295 Hz. The voltage full scale equals 2.16. Signals were sampled at 1010 Hz, which was the maximum sampling frequency possible with this system for this set-up. All data were analyzed using the MindWare EMG analysis software version 2.52 after converting raw SEMG ascii time series files to mindware files (MindWare Technologies) (courtesy of the Pittsburgh Mind and

Body Center). SEMG signals were rectified and filtered with a bandpass filter of 15-295 Hz (van Boxtel, 2001).

The root mean square (RMS) voltage (in microvolts) of the SEMG signals, which represents the effective amplitude of the signal (Fridlund & Cacioppo, 1986), was extracted from the following segments. One average was collected from the last minute of the baseline rest phase and one average was collected from the anticipation phase. From the baseline speech, public speaking stressor, and recovery speech, the first focus was on the all-voiced sentence "we were away a year ago" to ensure maximum comparability. In addition, up to 20 seconds of the "Rainbow Passage" (baseline speech and recovery speech) were compared to the same duration of public speaking. It was verified that only continuous, same-length, portions of public speaking were compared across groups (including up to two second speech pauses). The review of the public speaking samples revealed that beyond the first 20 seconds a continuous flow of speech could not be guaranteed for every speaker although many participants made a good effort to continue to speak for the most part of the allotted five minutes. The remaining four minutes of public speaking will be descriptively analyzed minute by minute as a habituation slope may become evident within the duration of public speaking. One average will be collected for the recovery phase, but the recovery phase will be also segmented into minute by minute intervals for descriptive purposes of tracking the recovery slope. Swallow activity will not be excluded but regarded as normally distributed across participants.

SBP values will be reduced to averages per phase. However, it will be examined how response values may be linked to baseline values and corrections for baseline differences among groups will only be made if a significant relation exists (Jennings & Gianaros, 2007).

3.6 STATISTICAL ANALYSIS

Statistical analyses were guided by the study's purpose. The questions were hierarchically organized as (I) primary outcome, (II) secondary outcomes, and (III) exploratory outcomes. All statistical analyses used analysis of variance (ANOVA). Significance level was set at $\alpha = .05$ for each equation. A priori comparisons as outlined in the hypotheses did not require adjustment for Type I error. Statistical analyses focused on the primary outcome. Although analyses were conducted on other outcomes, the investigation-wide alpha was not adjusted for those analyses given their secondary as well as exploratory nature. In addition, possible baseline differences between groups were examined with *t*-tests for the following variables: demographics (age, BMI), depression scores, and *Voice Handicap Index* (*VHI*).

3.6.1 Primary outcome (I)

Table 3-4. Dependent and independent variables for statistical equations pertaining to Specific Aim 1

Dependent Variable	Independent Variable 1	Independent Variable 2	Interactions
Submental SEMG (%MVC)	Phase	Personality (extraversion/introversion)	All
Infrahyoid SEMG (%MVC)	Phase	Personality (extraversion/introversion)	All
Anterior tibialis SEMG (%MVC)	Phase	Personality (extraversion/introversion)	All

Three separate 2x6 mixed analyses of variance (ANOVA) were performed on SEMG as a function of phase (baseline speech, rest, anticipation, stressor, recovery, recovery speech) and personality (extraversion vs. introversion). Two main effects (phase, personality) and one two-way interaction (phase x personality) were tested. In case of a main effect for phase (> 2 levels),

post hoc planned comparisons were performed to test the direction of the differences. If the interaction was significant, simple main effects were performed, e.g. simple main effect of personality for each phase. No simple comparisons were needed.

3.6.2 Secondary outcomes (II)

Table 3-5. Dependent and independent variables for statistical equations pertaining to Specific Aim 2

Dependent Variable	Independent Variable 1	Independent Variable 2	Interactions
Vocal effort	Phase	Personality (extraversion/introversion)	All
F_0	Phase	Personality (extraversion/introversion)	All
Intensity	Phase	Personality (extraversion/introversion)	All

Three 2x3 mixed ANOVA were performed separately on vocal effort, F_0, and intensity as a function of phase (baseline speech, stressor, recovery speech) and personality (extraversion vs. introversion). Two main effects (phase, personality) and one two-way interaction (phase x personality) were tested. Statistical analyses followed those for the primary outcome.

3.6.3 Exploratory outcomes (III)

Physiological mechanism:

Table 3-6. Dependent and independent variables for the statistical equation pertaining to Specific Aim 3

Dependent Variable	Independent Variable 1	Independent Variable 2	Interactions
SBP	Phase	Personality (extraversion vs. introversion)	All

A 2x6 mixed ANOVA was performed on SBP as a function of phase (baseline speech, rest, anticipation, stressor, recovery, recovery speech) and personality (extraversion vs. introversion). Two main effects (phase, personality) and one two-way interaction (phase x personality) were tested. Statistical analyses followed those for the primary outcome.

Emotional and cognitive mechanisms:

Table 3-7. Dependent and independent variables for the statistical equation pertaining to Specific Aim 4

Dependent Variable	Independent Variable 1	Independent Variable 2	Interactions
Negative emotions	Phase	Personality (extraversion vs. introversion)	All

A 2x3 mixed ANOVA was performed on negative affect as a function of phase (baseline, stressor, recovery) and personality (extraversion vs. introversion). Two main effects (phase, personality) and one two-way interaction (phase x personality) were tested. Statistical analyses followed those for the primary outcome.

Table 3-8. Dependent and independent variables for the statistical equations pertaining to Specific Aim 5

Dependent Variable	Independent Variable 1
Fear of public speaking	Personality (extraversion vs. introversion)

Table 3-9. Dependent and independent variables for the statistical equation pertaining to Specific Aim 6

Dependent Variable	Independent Variable 1
Rumination	Personality (extraversion vs. introversion)

Two one-way between-subjects ANOVAs were performed separately on the score on fear of public speaking and rumination as a function of personality with two levels (extraversion vs. introversion).

Mediation analyses:

Table 3-10. Dependent and independent variables for statistical equations pertaining to Specific Aim 7

Dependent Variable	Independent Variable 1	Independent Variable 2	Covariate	Interactions
SEMG (%MVC)	Phase	Personality (extraversion vs. introversion)	SBP	All
SEMG (%MVC)	Phase	Personality (extraversion vs. introversion)	Negative emotions	All
SEMG (%MVC)	Phase	Personality (extraversion vs. introversion)	Fear of public speaking	All

The proposed mediational model was examined as suggested by Stone (1992). A mediator was identified by following three steps. First, the predictor variable (personality) must have been associated with the proposed mediator (Specific Aims 3-6). Second, personality must have been associated with the primary outcome variable SEMG (Specific Aim 1). Third, the degree of association between personality and SEMG must have been remarkably reduced when the mediator of interest (SBP; negative emotions; fear of public speaking) was statistically controlled.

- Personality → SEMG
- Personality → *Mediator*
- Personality → *Mediator* → SEMG

The last step involved analyses of covariance (ANCOVA) where appropriate adjusting for SBP, negative emotions, and fear of public speaking where appropriate. If ANCOVA was not appropriate, a Pearson correlation matrix was generated to examine relations among all variables.

3.6.4 Power analysis

Power analysis was performed based on pilot data available for two groups of participants each with seven participants. Introverted subjects with neuroticism were contrasted with extraverted subjects without neuroticism during a stress reactivity protocol similar to the proposed one. Power analysis was performed on SEMG data for the submental and infrahyoid extralaryngeal sites.

1. Desired level of significance: α=0.05
2. Effect size of pilot data for the submental muscle group: partial η^2 =.020, f = .1428571
3. Effect size of pilot data for the infrahyoid muscle group: partial η^2 =.117, f = .3640094
4. Desired level of power: 0.80
5. Required total sample size (largest obtained number based on power analysis (G*Power) for the interaction effect): n = 54

4.0 RESULTS

In the next paragraphs, the experimental questions are revisited together with their respective planned statistical analyses. The questions were hierarchically organized in terms of (I) primary outcome, (II) secondary outcomes, and (III) exploratory outcomes. All statistical analyses used analysis of variance (ANOVA). The significance level was set at $\alpha = .05$ for each equation. *A priori* comparisons as outlined in the hypotheses were not adjusted for Type I error. Statistical analyses focused on the primary outcome. Although analyses were conducted on other outcomes, the investigation-wide alpha was not adjusted for those analyses either given their secondary as well as exploratory nature. All statistical analyses were performed with SPSS version 16.0 software. A summary of statistical results is provided in Table 4-21 at the end of this chapter.

All data were screened in order to determine if assumptions for each ANOVA were met. Most importantly, homogeneity of variance had to be demonstrated. The Brown-Forsythe test (α=.05) was used to test the homogeneity of variance for between-subjects ANOVAs, and results had to be non-significant. In addition, for any proposed *mixed* ANOVA compound symmetry, i.e. homogeneity of variance *and* covariance was tested. For that purpose, both Box's *M* (α=.001) and Mauchly's test of sphericity (α=.05) was used and both results should be non-significant. If either test was significant, the Huynh-Feldt adjustment was used. The Shapiro-Wilk test was used to test for normality (α=.05) and results should be non-significant as well. However,

ANOVA is known to be robust against violations of normality (Glass & Hopkins, 1996). Violations of normality were screened and reported, but no action was taken. Further, the independence of subjects was assumed, i.e. a response on the dependent variable was not influenced by another participant. Finally, the data were examined for outliers as reported by SPSS during data screening. Especially extreme outliers (> 3 *SD*) were examined. On the other hand, based on past experience and the composition of the current study sample, extreme responses, especially for the introversion group, were expected due to the within-group heterogeneity of the study sample in general and the range of personalities on a continuum specifically. It was chosen not to exclude any outliers unless specified and justified.

Finally, the results will be presented in both Table and Figure format in order to provide details around the actual scores obtained for the various outcome variables and secondly in order to provide a graphic illustration of the course of the data over time and the relation of the data between groups.

4.1.1 Group composition and baseline differences

To reiterate, personality groups were formed on the basis of a participant's score on extraversion on the *Eysenck Personality Questionnaire – Revised* (*EPQ-R*), which should be at least .5 *SD* above or below the norm on the trait extraversion for inclusion in the extraversion or introversion group respectively. Of note, the positive versus negative range of possible scores on extraversion on the *EPQ-R* test differs. The possible maximum *z*-score for extraversion on the *EPQ-R* is 1.54 whereas the possible maximum score for introversion is -2.54. The distribution of participants by personality in this study is provided in Table 4-1 and also in Figure 4-1. In addition,

corresponding *T* scores on the *Multidimensional Personality Questionnaire – Brief Form* (*MPQ-BF*) are presented in Table 4-1 to illuminate participant characteristics along other personality lines, as assessed by different personality instruments.

Table 4-1. The distribution of personality scores in the study sample

	Extraversion (*n* = 27)			Introversion (*n* = 27)		
Personality variable	*M*	*SD*	range	*M*	*SD*	range
Extraversion (*EPQ-R*)	0.98	0.27	0.52-1.54	-1.30	0.51	-2.54 to -0.50
Positive Emotionality (*MPQ-BF*)	64.22	8.64	51-78	46.33	8.76	22-61
Social Potency	59.96	6.78	45-71	45.30	7.20	34-62
Social Closeness	58.96	7.20	44-66	44.70	11.94	27-66
Neuroticism (*EPQ-R*)	-0.80	1.00	-2.49 to 1.16	-0.09	0.97	-2.31 to 1.70
Negative Emotionality (*MPQ-BF*)	43.96	11.24	27-65	48.74	8.37	31-71
Stress Reactivity	42.96	10.78	31-61	53.89	9.49	37-71
Psychoticism (*EPQ-R*)	.17	.92	-1.55 to 2.15	.44	1.2	-1.22 to 3.16
Constraint (*MPQ-BF*)	45.31[a]	11.00	22-61	44.56	9.12	22-63

Note. *EPQ-R z*-scores; *MPQ-BF T*-scores (50 = mean, +/- 10 = 1 *SD*)
MPQ-BF possible range of test scores: broad traits (positive and negative emotionality and constraint) = 22-78; social potency = 34-71, social closeness = 27-66, stress reactivity = 31-71
[a] *n* = 26

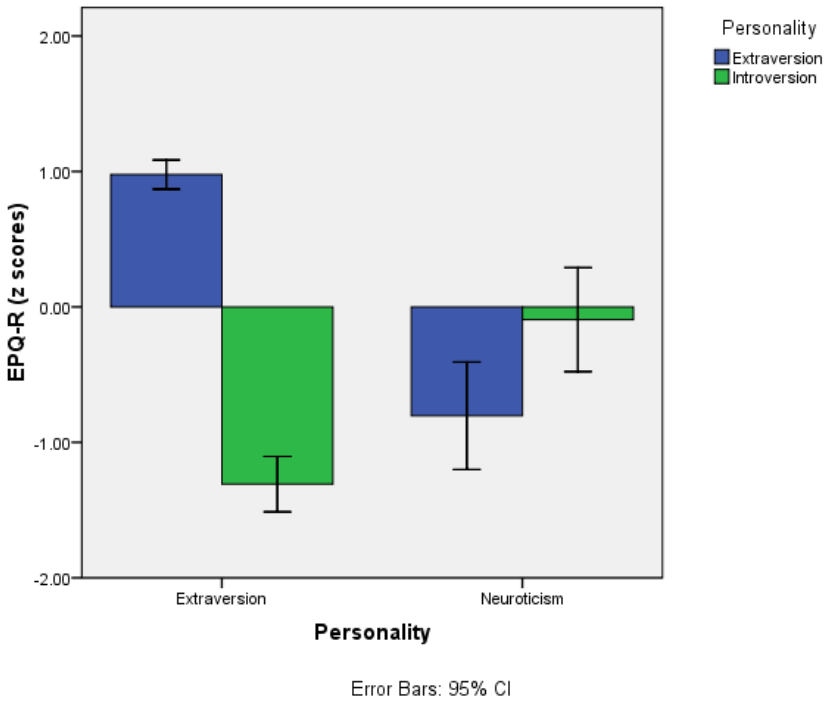


Figure 4-1. The distribution of personality in the study sample based on the *Eysenck Personality Questionnaire – Revised Form* (*EPQ-R*)

Further, as the trait neuroticism should be theoretically relevant to the research questions as well, a breakdown of extraverts and introverts by neuroticism is provided in Table 4-2.

Table 4-2. The frequency of neuroticism in the study sample

	Extraversion (n = 27)		Introversion (n = 27)	
Personality variable	+/- 0 *SD*	+/- .5 *SD*	+/- 0 *SD*	+/- .5 *SD*
w/ neuroticism	6 (22%)	12 (44%)	12 (44%)	7 (26%)
w/ stress reactivity	8 (30%)	9 (33%)	19 (70%)	12 (44%)
w/out neuroticism	21 (78%)	15 (56%)	15 (56%)	20 (74%)
w/out stress reactivity	19 (70%)	18 (67%)	8 (30%)	15 (56%)

Note. neuroticism = *EPQ-R*; stress reactivity = *MPQ-BF*

Potential baseline differences across groups were examined with independent-sample *t*-tests. Averages for age, body mass index (BMI), depression (*Beck Depression Inventory- BDI*), and *Voice Handicap Index* (*VHI*) are shown in Table 4-3. As the trait neuroticism may be theoretically relevant to the research questions as well, a *t*-test of group means on neuroticism on the *EPQ-R* was performed too. This test revealed a statistical difference, with the introversion group showing greater neuroticism than the extraversion group [$t(52) = -2.640$, $p = .011$ (equal variances assumed[4])]. The groups were not significantly different on age or BMI [age $t(52) = .599$, $p = .552$; BMI $t(52) = -.902$, $p = .371$ (equal variances assumed)]. However, the groups differed at baseline on mean scores for depression (*BDI*) and voice handicap (*VHI*) [*BDI* $t(40.619) = -3.258$, $p = .002$; *VHI* $t(41.240) = -3.767$, $p = .001$ (equal variances not assumed for either test)]. The introversion group reported more depression and voice handicap than the extraversion group. Demographic and general baseline data are summarized in Table 4-3 and illustrated in Figure 4-2.

Table 4-3. Demographic and general baseline data

	Extraversion (n = 27)			Introversion (n = 27)		
Variable	*M*	*SD*	range	*M*	*SD*	range
Age	23.26	3.77	18-32	22.67	3.50	19-35
BMI	21.67	2.51	17.00-26.60	22.26	2.28	17.40-28.30
BDI	2.22	2.29	0-8	5.19	4.13	0-15
VHI	7.48	6.42	0-26	16.89	11.28	0-41

Note. BMI = body mass index, *BDI = Beck Depression Inventory*, *VHI = Voice Handicap Index* (maximum score = 120)

[4] In the case of "equal variances assumed" homogeneity of variance was given while Welch's *t*-test was used in case homogeneity of variance was violated.

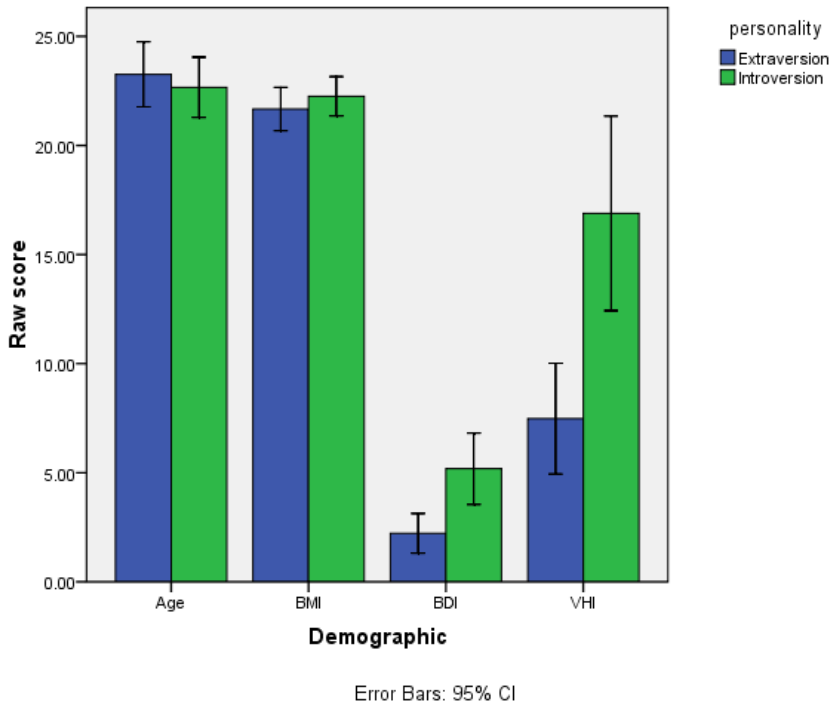


Figure 4-2. Demographic and general baseline data

4.1.2 Primary outcome (I)

Table 4-4 provides a snapshot of the experimental questions under investigation in this section. Of note, SEMG activity was investigated for two types of stressed speech samples: (a) comparison of a standard speech sample "we were away" across speech phases (baseline speech, stressed speech, repeated baseline during recovery); and (b) public speaking under conditions of stress with the reading of the Rainbow Passage used as baseline speech and repeated baseline during recovery. Data for both types of speech samples are presented.

Table 4-4. Dependent and independent variables for statistical equations pertaining to Specific Aim 1

Dependent Variable	Independent Variable 1	Independent Variable 2	Interactions
Submental SEMG (50% MVC)	Phase	Personality (extraversion/introversion)	All
Infrahyoid SEMG (50% MVC)	Phase	Personality (extraversion/introversion)	All
Anterior tibialis SEMG (50% MVC)	Phase	Personality (extraversion/introversion)	All

Note. MVC = maximum voluntary contraction

Submental SEMG

First, a summary of the descriptive data will be provided in Table 4-5 followed by a summary of the statistical results.

Table 4-5. Descriptive statistics for submental SEMG (50% MVC) (a) based on the standard speech sample "we were away;" (b) based on reading of the Rainbow Passage and public speaking (speech phases highlighted)

(a)

	Extraversion (*n* = 26)			Introversion (*n* = 27)		
Phase	*M* (%)	*SD*	Range	*M* (%)	*SD*	range
Baseline Speech	39.98	16.17	8.19-70.20	43.13	15.34	8.98-71.43
Rest	31.86	12.24	6.50-49.57	30.95	13.71	5.46-58.02
Anticipation	31.49	11.74	6.48-50.29	31.84	14.59	4.39-57.00
Stressed Speech	40.25	14.77	9.76-70.18	43.80	17.17	8.91-77.88
Recovery	31.57	11.65	6.54-49.56	33.45	13.19	6.80-57.00
Recovery Speech	39.76	15.03	8.33-66.13	42.23	15.56	7.73-71.43

Note. All speech data were based on the phrase "we were away"
Extraversion *n* = 26: one participant was excluded due to partial recording errors

(b)

	Extraversion (n = 26)			Introversion (n = 25)		
Phase	M (%)	SD	range	M (%)	SD	range
Rainbow Passage 1	44.92	15.85	9.88-70.71	43.46	16.16	8.92-78.11
Rest	31.86	12.24	6.50-49.57	30.65	13.76	5.46-58.02
Anticipation	31.49	11.74	6.48-50.29	31.64	14.75	4.39-57.00
Public Speaking	44.25	17.00	8.13-74.82	44.29	15.53	8.30-66.35
Recovery	31.57	11.65	6.54-49.56	33.40	13.21	6.80-57.00
Rainbow Passage 2	41.70	14.29	9.14-63.55	42.45	15.86	8.54-68.86

Note. Rainbow Passage and public speaking: average of initial twenty seconds of speech; Extraversion n = 26 and introversion n = 25: a total of three participants were not included in the analyses due to partial recording errors

A 2x6 two-way mixed ANOVA assessed the effect of phase (baseline speech, rest, anticipation, stressed speech ("we were away"), recovery, recovery speech) and personality (extraversion, introversion) on submental SEMG activation. The assumption of normality was met. The assumption of homogeneity of covariance matrices of the dependent variable across groups was not met [*Box's M* = 91.022, $F(21,9535.832) = 3.784$, $p < .001$] nor was the assumption of sphericity met for phase [Mauchly's $W = .220$, $\chi^2(14) = 74.448$, $p < .001$]. Therefore, the Huynh-Feldt adjustment was used for all analyses. There was a significant main effect of phase [$F(3.718,189.604) = 45.924$, $p < .001$, partial $\eta^2 = .474$]. Pairwise comparisons using Bonferroni adjustments revealed that there was no effect of phase across the speech phases (baseline speech, stressed speech, recovery speech) nor was there a phase effect across non-speech phases (rest, anticipation, recovery). The main effect of phase stemmed from the significant difference between speech and non-speech phases ($p < .001$) with higher SEMG activity during speech phases. The main effect for personality was not significant [$F(1,51) =$

.225, p = .637, partial η= .004], nor was the interaction of phase and personality [F(3.718,189.604) = 1.199, p = .313, partial η^2= .023]. The data are illustrated in Figure 4-3.

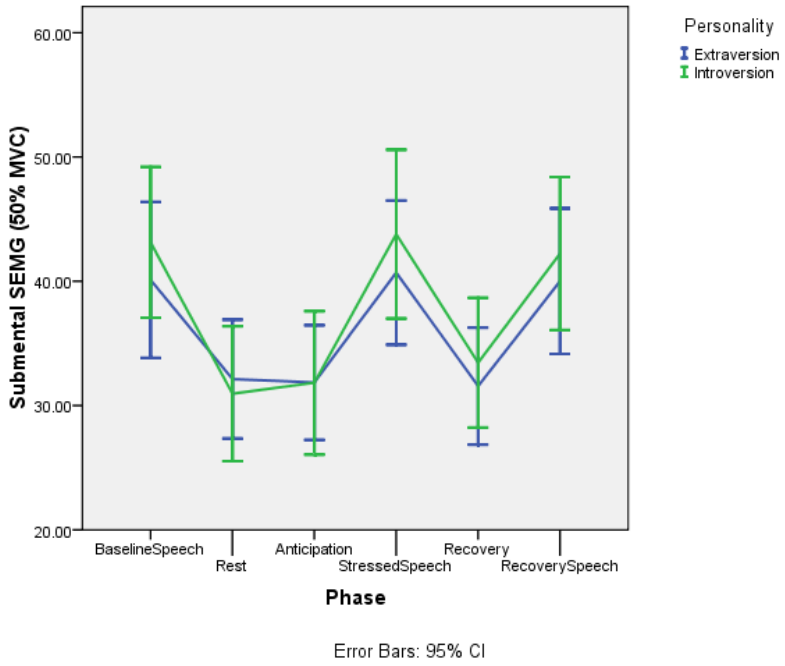


Figure 4-3. Submental SEMG (50% MVC) based on standard speech sample "we were away"

A second 2x6 two-way mixed ANOVA assessed the effect of phase (*Rainbow Passage 1*, rest, anticipation, *public speaking*, recovery, *Rainbow Passage 2*) and personality (extraversion, introversion) on submental SEMG activation. The assumption of normality was met except for the public speaking phase and for the introversion group only. The assumption of homogeneity of covariance matrices of the dependent variable across groups was not met [*Box's M* = 100.066, F(21,8800.239) = 4.135, p < .001] nor was the assumption of sphericity met for phase [Mauchly's W = .203, χ^2(14) = 75.127, p < .001]. Therefore, the Huynh-Feldt adjustment was used for all analyses. There was a significant main effect of phase averaged across personality [F(3.563,174.587) = 79.866, p < .001, partial η^2 = .620]. Pairwise comparisons using Bonferroni

adjustments revealed that there was no effect of phase across the non-speech phases (rest, anticipation, recovery), but there was one significant effect across speech phases (Rainbow Passage 1, public speaking, Rainbow Passage 2). The repeated baseline speech during recovery was significantly lower than the initial baseline ($p = .046$). Further, the main effect of phase also stemmed from the significant difference between speech and non-speech phases ($p < .001$) with higher SEMG activity during speech phases. The main effect for personality averaged across phase was not significant [$F(1,49) < .001$, $p = .996$, partial $\eta^2 < .001$], and neither was the interaction effect [$F(3.563,174.587) = .715$, $p = .567$, partial $\eta^2 = .014$]. These results are illustrated in Figure 4-4.

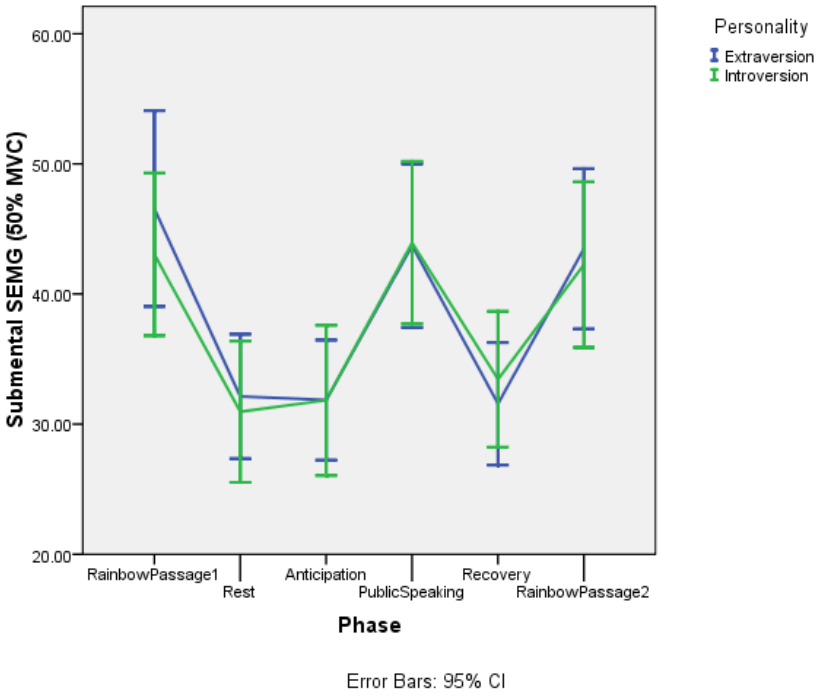


Figure 4-4. Submental SEMG (50% MVC) based on public speaking and reading of Rainbow Passage

Infrahyoid SEMG

Relative to infrahyoid SEMG data, again first a summary of the descriptive data will be provided in Table 4-6 followed by a summary of the statistical results.

Table 4-6. Descriptive statistics for infrahyoid SEMG (50% MVC) (a) based on standard speech sample "we were away;" (b) based on reading of Rainbow Passage and public speaking (speech phases highlighted)

(a)

	Extraversion (n = 26)			Introversion (n = 27)		
Phase	*M* (%)	*SD*	Range	*M* (%)	*SD*	range
Baseline Speech	38.22	18.96	9.12-97.27	54.24	39.02	21.78-221.31
Rest	28.86	10.71	7.62-50.45	33.18	12.61	15.00-71.72
Anticipation	28.89	10.10	9.40-47.60	32.81	14.66	2.66-77.15
Stressed Speech	37.74	16.87	8.43-85.39	53.26	44.82	18.64-243.17
Recovery	29.26	10.49	8.21-46.86	33.25	12.58	15.10-71.72
Recovery Speech	35.56	15.56	10.05-81.47	51.19	32.49	19.80-162.57

Note. All speech data were based on the phrase "we were away"
Extraversion n = 26: one participant was excluded due to partial recording errors

(b)

	Extraversion (n = 26)			Introversion (n = 25)		
Phase	*M* (%)	*SD*	range	*M* (%)	*SD*	range
Rainbow Passage 1	45.61	21.44	7.40-91.36	54.87	35.50	6.82-188.30
Rest	28.86	10.71	7.62-50.45	33.63	12.80	15.00-71.72
Anticipation	28.89	10.10	9.40-47.60	33.23	15.01	2.66-77.15
Public Speaking	45.02	19.55	13.25-93.59	76.43	71.16	24.04-339.48
Recovery	29.26	10.49	8.21-46.86	33.74	12.72	15.10-71.72
Rainbow Passage 2	42.73	21.02	6.47-93.83	54.58	32.65	6.62-165.14

Note. Rainbow Passage and public speaking: average of initial twenty seconds of speech; Extraversion $n = 26$ and introversion $n = 25$: a total of three participants were not included in the analyses due to partial recording errors

A 2x6 two-way mixed ANOVA was performed to assess the influence of phase (baseline speech, rest, anticipation, stressed speech, recovery, recovery speech) and personality (extraversion, introversion) on infrahyoid SEMG data. The assumption of normality was not met for the baseline speech phase (both groups) and for the introversion group for the stressor and recovery speech phases, but skewness was in the same direction for those phases and groups. The assumption of homogeneity of covariance matrices of the dependent variable across groups was not met [*Box's M* = 84.625, $F(21,9535.832) = 3.518$, $p < .001$] nor was the assumption of sphericity for phase [Mauchly's $W < .001$, $\chi^2(14) = 463.626$, $p < .001$]. Therefore, the Huynh-Feldt adjustment was used for all analyses. Results from statistical analyses revealed a significant main effect of phase [$F(1.303,66.462) = 13.940$, $p < .001$, partial $\eta^2 = .215$]. Pairwise comparisons using Bonferroni adjustments revealed that there was no effect of phase within the speech phases (baseline speech, stressor, recovery speech) nor within the non-speech phases (rest, anticipation, recovery). As for submental SEMG data, the main effect of phase stemmed from a significant difference between speech and non-speech phases (ranging from $p=.001$ to $p=.018$), with higher SEMG activity seen during speech phases. The main effect of personality was not significant [$F(1,51) = 3.761$, $p = .058$, partial $\eta^2 = .069$] as was the interaction of phase and personality [$F(1.303,66.462) = 2.381$, $p = .120$, partial $\eta^2 = .045$]. Results are illustrated in Figure 4-5.

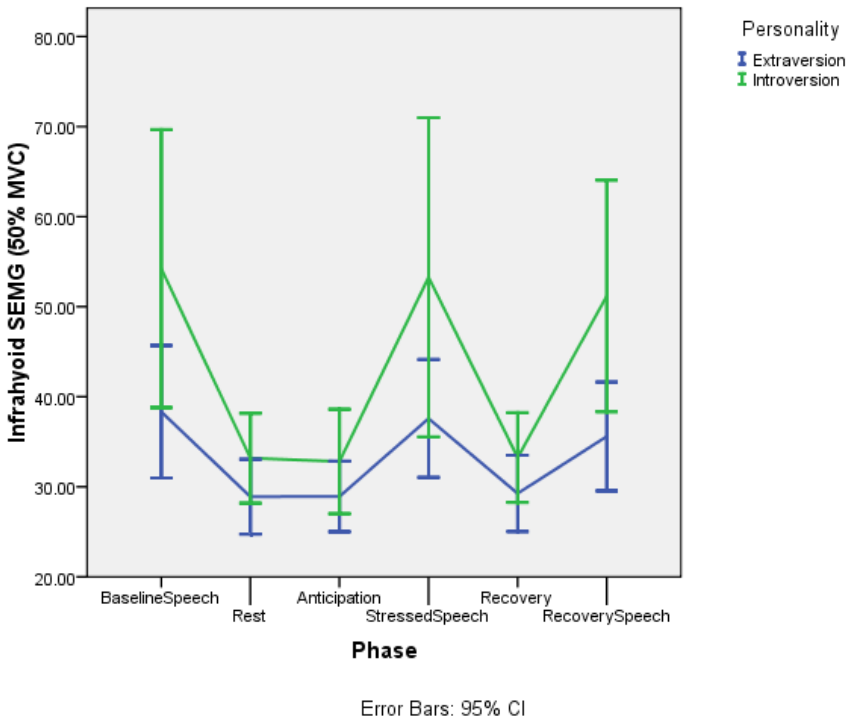


Figure 4-5. Infrahyoid SEMG (50% MVC) based on the standard speech sample "we were away"

Another 2x6 two-way mixed ANOVA was performed to assess the influence of phase (*Rainbow Passage 1*, rest, anticipation, *public speaking*, recovery, *Rainbow Passage 2*) and personality (extraversion, introversion) on infrahyoid SEMG data. The assumption of normality was not met for the introversion group for the baseline speech, public speaking, and recovery speech phases. The assumption of homogeneity of covariance matrices of the dependent variable across groups was not met [*Box's M* = 77.969, $F(21,8800.239) = 3.222$, $p < .001$] nor was the assumption of sphericity for phase [Mauchly's $W < .001$, $\chi^2(14) = 522.073$, $p < .001$]. Therefore, the Huynh-Feldt adjustment was used for all analyses. Results from statistical analyses revealed a significant main effect of phase [$F(2.127,104.203) = 14.839$, $p < .001$, partial $\eta^2 = .232$] and of personality [$F(1,49) = 4.798$, $p = .033$, partial $\eta^2 = .089$], but the interaction of phase and

personality was not significant [$F(2.127,104.203) = 2.508$, $p = .083$, partial $\eta^2 = .049$]. Pairwise comparisons using Bonferroni adjustments revealed that there was no effect of phase within the speech phases (baseline speech, public speaking, recovery speech) nor within the non-speech phases (rest, anticipation, recovery). The main effect of phase stemmed from significant differences between the following speech and non-speech phases (ranging from $p < .001$ to $p = .004$), with higher SEMG activity seen during speech phases: Rainbow Passage 1 as compared to rest, anticipation, and recovery and Rainbow Passage 2 as compared with anticipation. Results are illustrated in Figure 4-6.

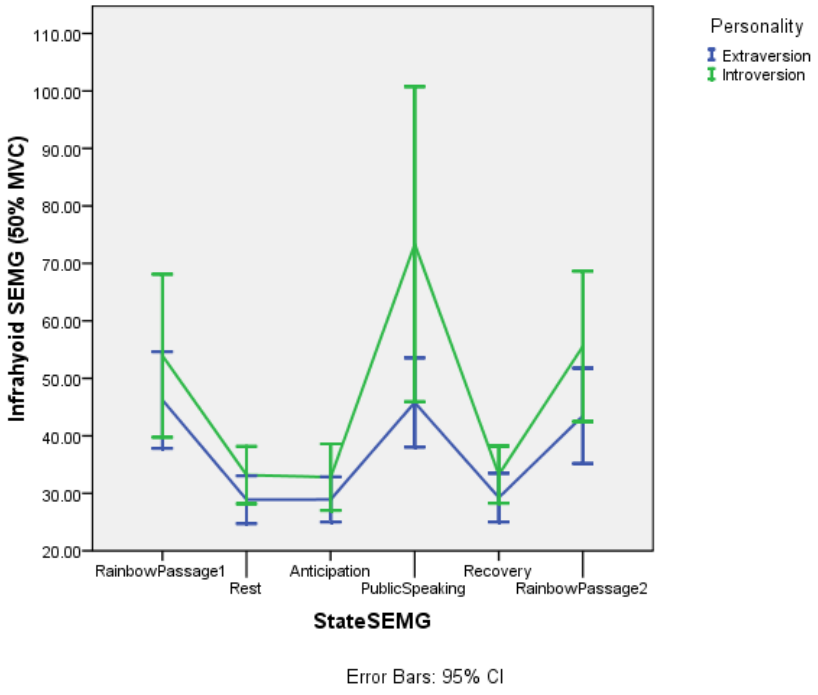


Figure 4-6. Infrahyoid SEMG (50% MVC) based on public speaking and reading of Rainbow Passage

Anterior tibialis

Lastly relative to the leg data (anterior tibialis muscle), a summary of the descriptive data will be provided in Table 4-7 followed by a summary of the statistical results.

Table 4-7. Descriptive statistics for anterior tibialis SEMG (50% MVC) (a) based on standard speech sample "we were away;" (b) based on reading of Rainbow Passage and public speaking (speech phases highlighted)

(a)

	Extraversion (n = 26)			Introversion (n = 27)		
Phase	*M* (%)	*SD*	Range	*M* (%)	*SD*	range
Baseline Speech	32.05	10.79	8.17-52.66	32.24	10.66	13.63-54.42
Rest	31.86	12.24	6.50-49.57	30.95	13.71	5.46-58.02
Anticipation	31.49	11.74	6.48-50.29	31.84	14.59	4.39-57.00
Stressed Speech	31.49	7.30	19.99-46.79	32.90	9.19	18.29-51.53
Recovery	31.57	11.65	6.54-49.56	33.45	13.19	6.80-57.00
Recovery Speech	32.93	7.93	17.45-52.84	32.14	9.14	16.07-54.47

Note. All speech data were based on the phrase "we were away"
Extraversion n = 26: one participant was not included in the analyses due to partial recording errors

(b)

Table 4-8. Descriptive data for anterior tibialis SEMG (50% MVC)

	Extraversion (n = 26)			Introversion (n = 25)		
Phase	*M* (%)	*SD*	range	*M* (%)	*SD*	range
Rainbow Passage 1	33.50	7.81	18.72-47.46	33.01	9.44	20.50-52.95
Rest	32.50	7.14	18.38-45.00	32.33	9.09	18.01-49.28
Anticipation	32.48	7.25	19.33-45.15	32.58	9.43	17.50-50.80
Public Speaking	32.43	7.51	19.36-45.86	37.97	27.14	17.08-161.80
Recovery	32.48	6.97	20.00-44.93	32.87	9.49	15.90-52.05
Rainbow Passage 2	32.23	6.88	18.44-44.96	32.13	9.08	16.21-50.46

Note. Rainbow Passage and public speaking: average of initial twenty seconds of speech;

Introversion $n = 25$: two participants were not included in the analyses due to partial recording errors

A 2x6 two-way mixed ANOVA was performed to assess the influence of phase (baseline speech, rest, anticipation, stressed speech, recovery, recovery speech) and personality (extraversion, introversion) on anterior tibialis SEMG data. The assumption of normality was not met for the introverted group during the stressor phase, but skewness was in the same direction for that phase and both groups. The assumption of homogeneity of covariance matrices of the dependent variable across groups was not met [*Box's M* = 91.288, $F(21,9535.832) = 3.795$, $p < .001$] nor was the assumption of sphericity for phase [Mauchly's $W = .020$, $\chi^2(14) = 192.835$, $p < .001$]. Therefore, the Huynh-Feldt adjustment was used for all analyses. Results from statistical analyses revealed no significant main effects of phase [$F(1.913,97.572) = .140$, $p = .861$, partial $\eta^2 = .003$] and of personality [$F(1,51) = .027$, $p = .869$, partial $\eta^2 = .001$ nor an interaction of phase and personality [$F(1.913,97.572) = .216$, $p = .797$, partial $\eta^2 = .004$]. The results are illustrated in Figure 4-7.

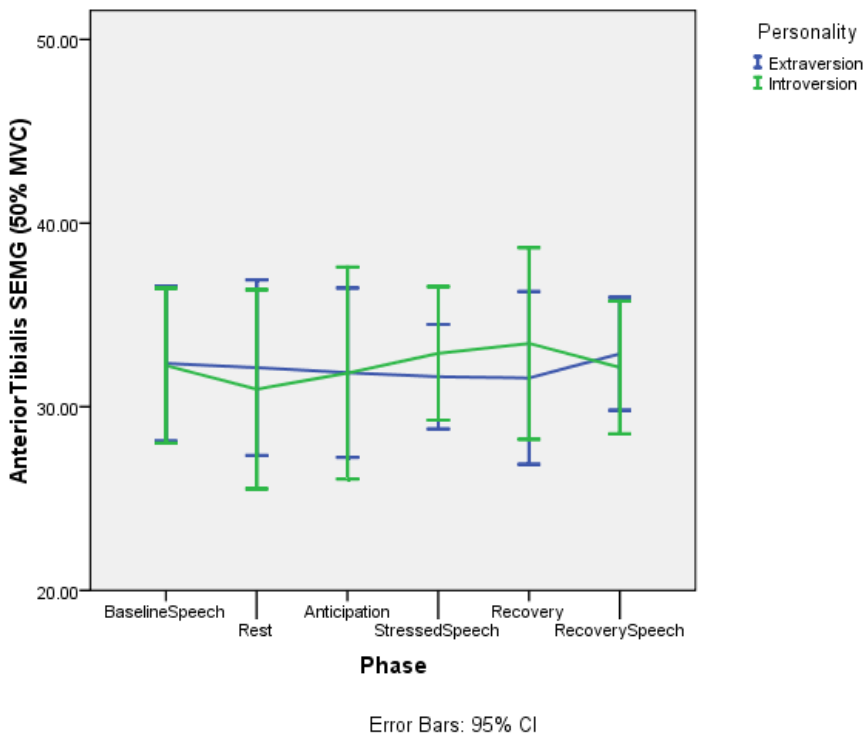


Figure 4-7. Anterior tibialis SEMG (50% MVC) based on the standard speech sample "we were away"

Another 2x6 two-way mixed ANOVA was performed to assess the influence of phase (*Rainbow Passage 1*, rest, anticipation, *public speaking*, recovery, *Rainbow Passage 2*) and personality (extraversion, introversion) on anterior tibialis SEMG data. The assumption of normality was not met for the introverted group during the baseline speech, rest, anticipation and stressor phases. The assumption of homogeneity of covariance matrices of the dependent variable across groups was not met [*Box's M* = 108.307, $F(21,8800.239) = 4.476$, $p < .001$] nor was the assumption of sphericity for phase [Mauchly's $W < .001$, $\chi^2(14) = 517.797$, $p < .001$]. Therefore, the Huynh-Feldt adjustment was used for all analyses. Results from statistical analyses revealed no significant main effects of phase [$F(1.141,55.910) = 1.366$, $p = .252$, partial $\eta^2 = .027$] and of personality [$F(1,49) = .119$, $p = .732$, partial $\eta^2 = .002$] nor an interaction of

phase and personality [$F(1.141,55.910) = 1.452$, $p = .237$, partial $\eta^2 = .029$]. The results are illustrated in Figure 4-8.

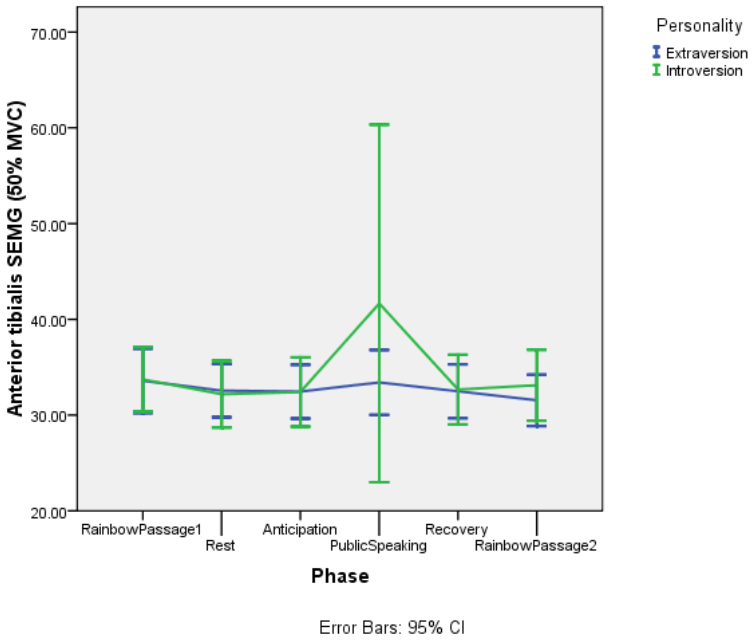


Figure 4-8. Anterior tibialis SEMG (50% MVC) based on public speaking and reading of the Rainbow Passage

4.1.3 Secondary outcomes (II)

Table 4-9 provides a snapshot of the experimental questions under investigation in this section.

Table 4-9. Dependent and independent variables for statistical equations pertaining to Specific Aim 2

Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
Vocal effort	Phase	Personality (extraversion/introversion)	All
F_0	Phase	Personality (extraversion/introversion)	All
Intensity	Phase	Personality (extraversion/introversion)	All

Vocal effort

First, a summary of the descriptive data will be provided in Table 4-10 followed by a summary of the statistical results.

Table 4-10. Descriptive statistics for Direct Magnitude Estimation (DME) of vocal effort

	Extraversion (n = 26)			Introversion (n = 27)		
Phase	*M*	*SD*	range	*M*	*SD*	range
Baseline Speech	102.50	9.92	100-150	120.00	33.97	100-200
Public Speaking	139.23	64.18	100-300	180.37	122.00	100-500
Recovery Speech	108.08	21.78	100-200	113.52	28.72	100-200

Note. A DME of 100 represented comfortable amount of vocal effort

A 2x3 two-way mixed ANOVA was performed on the level of perceived vocal effort as a function of phase (baseline speech, public speaking, recovery speech) and personality (extraversion, introversion). The assumption of normality was not met for any phase regardless of group, but skewness was in the same direction for all cells. The assumption of homogeneity of covariance matrices of the dependent variable across groups was not met [*Box's M* = 40.879, $F(6,18767.603) = 6.377$, $p < .001$], and neither was the assumption of sphericity for phase [Mauchly's $W = .198$, $\chi^2(2) = 81.015$, $p < .001$]. Therefore, the Huynh-Feldt adjustment was used for phase data. A multitude of outliers was present for each group and for each phase. Data from one extreme outlier in the extraversion group was removed, for a subject who scored more than three *SD* above the group average during the public speaking phase. There was a significant main effect of phase [$F(1.139, 58.082) = 13.173$, $p < .001$, partial $\eta^2 = .205$]. Pairwise comparisons using Bonferroni adjustments showed that the public speaking scores were significantly greater than both the baseline speech ($p = .001$) and recovery speech scores ($p = .003$), but the non-

stressed speech scores did not differ significantly from each other (baseline and recovery speech). Also the main effect for personality was significant [$F(1,51) = 4.014$, $p = .050$, partial $\eta^2 = .073$], but the interaction effect was not [$F(1.139, 58.082) = 1.369$, $p = .251$, partial $\eta^2 = .026$]. Figure 4-9 illustrates the results for perceived vocal effort.

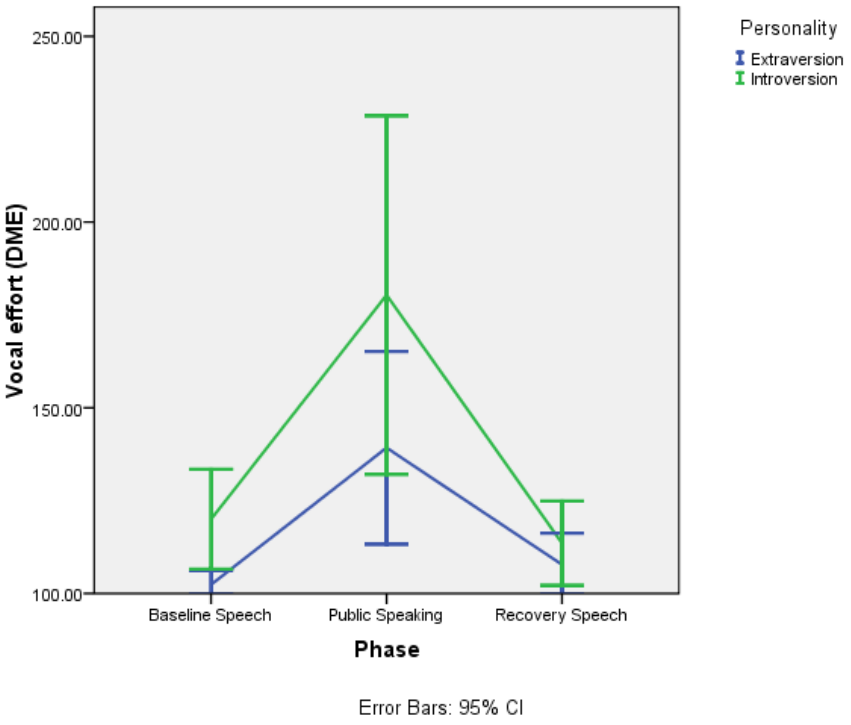


Figure 4-9. The distribution of perceived vocal effort (Direct Magnitude Estimation) per phase and between groups

Vocal fundamental frequency

First, a summary of the descriptive data will be provided in Table 4-11 followed by a summary of the statistical results.

Table 4-11. Descriptive statistics for voice fundamental frequency (F_0)

	Extraversion (n = 27)			Introversion (n = 27)		
Phase	*M* (ST)	*SD*	range	*M* (ST)	*SD*	range
Baseline Speech	221.62 (A)	17.23	185.73-249.69	218.12 (A)	19.72	172.16-256.83
Stressed Speech	210.94 (G#)	21.57	167.93-255.34	209.46 (G#)	20.90	157.60-246.86
Recovery Speech	219.79 (A)	22.06	189.45-267.54	219.83 (A)	24.79	163.37-273.44

Note. All speech data were based on the phrase "we were away"
ST = semitones

A 2x3 two-way mixed ANOVA was performed on the level of vocal fundamental frequency (F_0) as a function of phase (baseline speech, stressed speech, recovery speech) and personality (extraversion, introversion). The assumption of normality was met for all phases and groups. Both, the assumption of homogeneity of covariance matrices of the dependent variable across groups [*Box's M* = 2.867, $F(6,19591.245) = .448$, $p = .847$] and the assumption of sphericity for phase were met [Mauchly's $W = .919$, $\chi^2(2) = 4.286$, $p = .117$]. There was a significant main effect of phase [$F(2,104) = 9.551$, $p < .001$, partial $\eta^2 = .155$]. Pairwise comparisons using Bonferroni adjustments showed that values for stressed speech were significantly lower than both the baseline speech ($p = .003$) and recovery speech values ($p = .002$), but the non-stressed speech values did not differ significantly from each other (baseline and recovery speech). The main effect for personality was not significant [$F(1,52) = .111$, $p = .741$, partial $\eta^2 = .002$], and neither was the interaction effect [$F(2,104) = .244$, $p = .784$, partial $\eta^2 = .005$]. Figure 4-10 illustrates the results for vocal F_0.

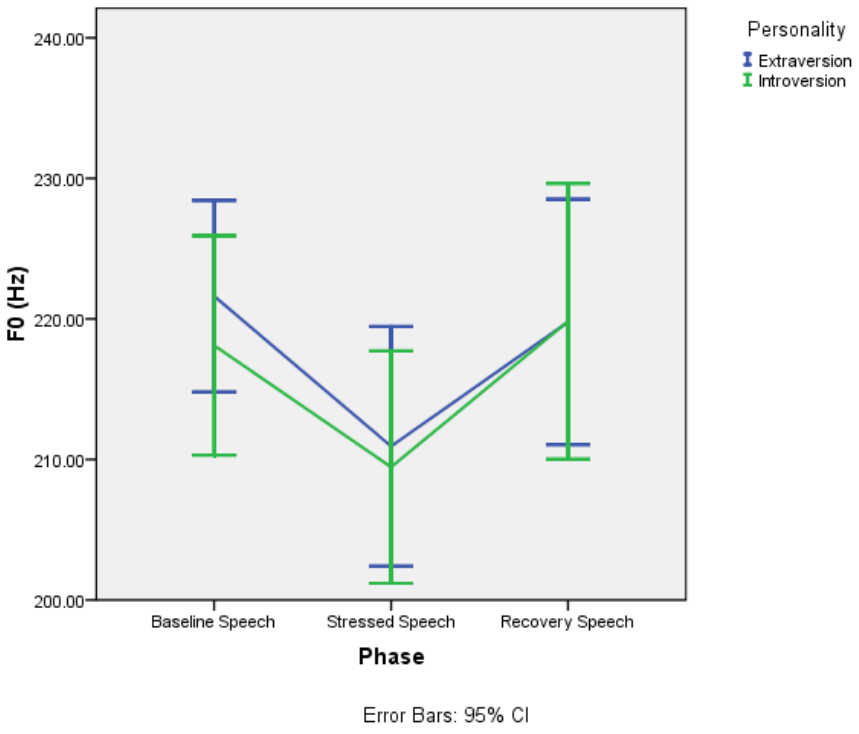


Figure 4-10. The distribution of vocal fundamental frequency (F_0 in Hz) per phase and between groups

Voice intensity

First, a summary of the descriptive data will be provided in Table 4-12 followed by a summary of the statistical results.

Table 4-12. Descriptive statistics for voice intensity (dB)

	Extraversion (n = 27)			Introversion (n = 26)		
Phase	*M*	*SD*	range	*M*	*SD*	range
Baseline Speech	65.28	2.91	59.22-70.67	64.78	3.70	58.31-77.92
Stressed Speech	61.95	3.34	57.25-71.30	62.01	3.87	55.46-74.90
Recovery Speech	65.02	2.96	60.25-72.03	65.06	3.83	60.10-78.15

Note. All speech data were based on the phrase "we were away"

A 2x3 two-way mixed ANOVA was performed on the level of voice intensity in dB as a function of phase (baseline speech, stressed speech, recovery speech) and personality (extraversion, introversion). The assumption of normality was not met for the introversion group for any of the three phases. The assumption of homogeneity of covariance matrices of the dependent variable across groups was met [*Box's M* = 7.208, $F(6,18767.603) = 1.124$, $p = .345$] as was the assumption of sphericity for phase [Mauchly's $W = .948$, $\chi^2(2) = 2.696$, $p = .260$]. Data from one outlier in the group with introversion was removed, for a subject whose data were more than three *SD* above the group average. There was a significant main effect of phase [$F(2,102) = 100.328$, $p < .001$, partial $\eta^2 = .663$]. Pairwise comparisons using Bonferroni adjustment revealed that vocal intensity was significantly lower during stressor exposure as compared to either baseline speech or recovery speech (both $p < .001$). The main effect for personality was not significant [$F(1,51) = .699$, $p = .407$, partial $\eta^2 = .014$], and nor was the interaction effect [$F(2,102) = .866$, $p = .424$, partial $\eta^2 = .017$]. Figure 4-11 illustrates the results for intensity.

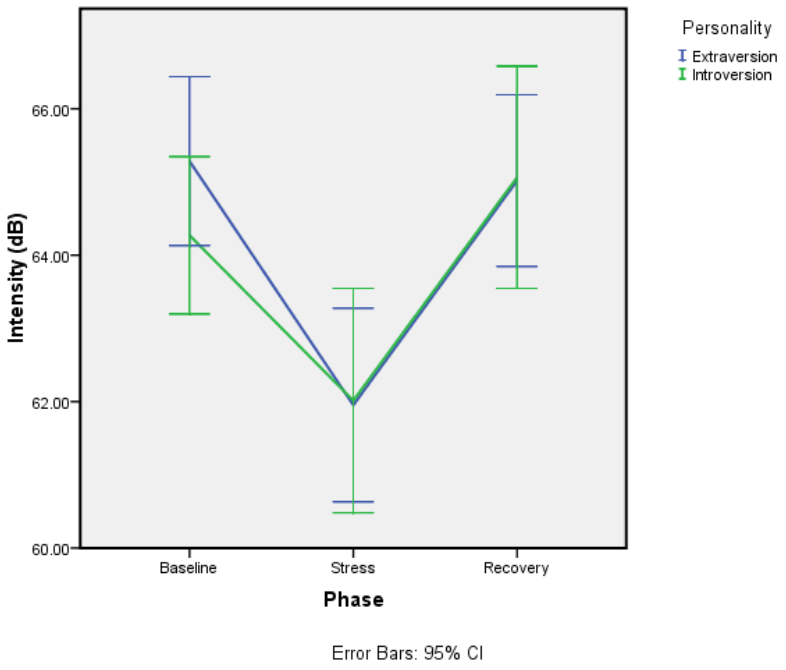


Figure 4-11. The distribution of voice intensity (dB) per phase and between groups

4.1.4 Exploratory outcomes (III)

Systolic blood pressure

Table 4-13 provides a snapshot of the experimental questions followed by a summary of the descriptive data for systolic blood pressure (SBP) in Table 4-14.

Table 4-13. Dependent and independent variables for the statistical equation pertaining to Specific Aim 3

Dependent Variable	Independent Variable 1	Independent Variable 2	Interactions
SBP	Phase	Personality (extraversion/introversion)	All

Table 4-14. Descriptive statistics for systolic blood pressure (SBP mmHg)

	Extraversion (n = 26)			Introversion (n = 27)		
Phase	*M*	*SD*	range	*M*	*SD*	range
Baseline Speech	114.12	7.15	98.50-134.00	113.20	11.81	92.00-139.50
Rest	107.66	5.55	95.60-122.40	106.36	8.88	87.60-127.4
Anticipation	116.44	8.11	101.50-134.00	118.33	12.69	95.50-145.50
Public Speaking	125.30	10.12	106.00-148.75	125.52	12.86	102.25-148.50
Recovery	112.16	5.53	99.60-123.20	112.39	8.56	94.60-132.80
Recovery Speech	113.31	6.84	95.50-128.00	114.83	10.94	92.50-137.50

Note. Extraversion n = 26: one participant was excluded due to recording errors.

A 2x6 two-way mixed ANOVA was performed on the level of SBP as a function of phase (baseline speech, rest, anticipation, public speaking, recovery, recovery speech) and personality (extraversion, introversion). There were no baseline differences during rest between groups, $t(51) = .637$, $p = .527$. The assumption of normality was met for all data for all phases and both groups. The assumption of homogeneity of covariance matrices of the dependent variable across groups was met [*Box's M* = 32.833, $F(21,9535.83) = 1.365$, $p = .123$]. However, the assumption of sphericity for phase was not met [Mauchly's $W = .369$, $\chi^2(14) = 48.940$, $p < .001$]. Therefore, the Huynh-Feldt adjustment was used for phase data. There was a significant main effect of phase [$F(3.865, 197.114) = 77.080$, $p < .001$, partial $\eta^2 = .602$]. Pairwise comparisons using Bonferroni adjustments showed that almost all phase combinations were significantly different from each other (ranging from $p < .001$ to $p = .007$) except for the comparison of the non-stressed speech samples (baseline and recovery) and of note, the comparison of recovery with either baseline or recovery speech. The main effect for personality

was non-significant [$F(1,51) = .015$, $p = .904$, partial $\eta^2 < .001$] as was the interaction effect [$F(3.865, 197.114) = .828$, $p = .506$, partial $\eta^2 = .016$]. Figure 4-12 illustrates the results for SBP, especially the direction of differences among phases. In addition, Figure 4-13 provides an in-depth look at the participants' time course of SBP throughout the protocol for each measurement point, in particular for the recovery phase.

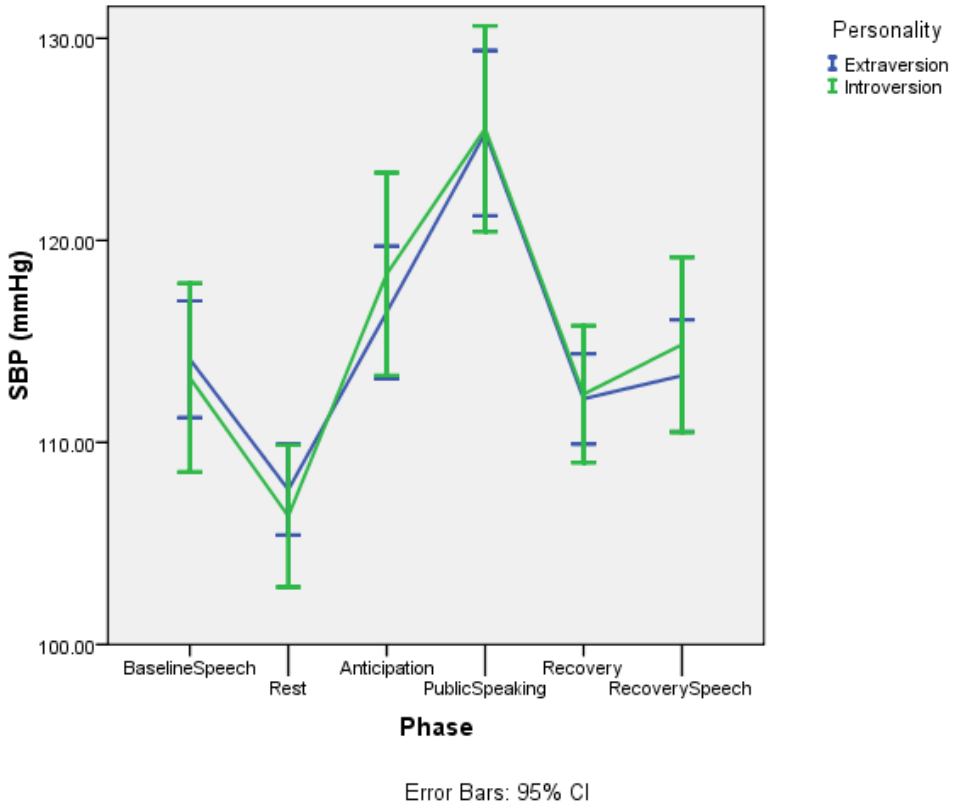
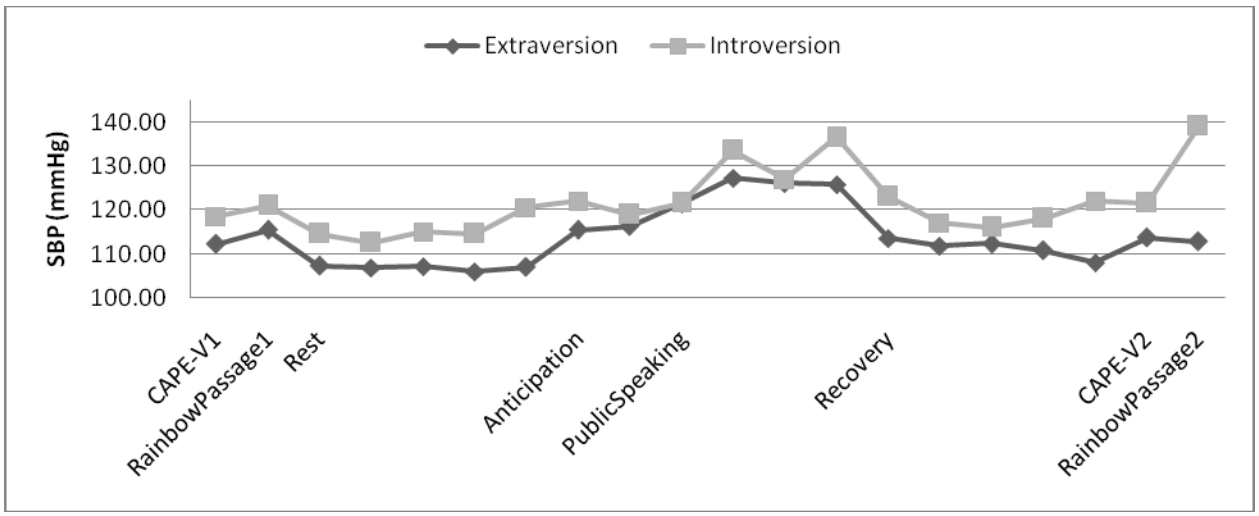


Figure 4-12. The distribution of systolic blood pressure (SBP) averages per phase and between groups



Note. CAPE-V = Consensus Auditory Perceptual Evaluation of Voice, i.e. sustained vowels and sentences; CAPE-V + Rainbow Passage = baseline/recovery speech; measurements were taken every 90 seconds.

Figure 4-13. The distribution of each systolic blood pressure (SBP) measurement point across phases and between groups

Negative emotional state

Table 4-15 provides a snapshot of the experimental question followed by a summary of the descriptive data for negative emotional state in Table 4-16.

Table 4-15. Dependent and independent variables for the statistical equation pertaining to Specific Aim 4

Dependent Variable	**Independent Variable 1**	**Independent Variable 2**	**Interactions**
Negative emotions	Phase	Personality (extraversion/introversion)	All

Table 4-16. Descriptive statistics for negative emotional state (*PANAS-X*)

	Extraversion (n = 27)			Introversion (n = 27)		
Phase	*M*	*SD*	range	*M*	*SD*	range
Rest	10.67	1.11	10-14	11.11	2.15	10-18
Public Speaking	18.19	5.92	10-28	20.89	6.26	11-35
Recovery	12.41	3.33	10-24	14.11	4.85	10-26

Note. Maximum score = 50
PANAS-X = Positive and Negative Affect Scales-X

A 2x3 two-way mixed ANOVA was performed on the level of negative emotions as a function of phase (rest, public speaking, recovery) and personality (extraversion, introversion). The assumption of normality was violated for the baseline and recovery phase and for both groups and for the public speaking phase, but only for the extraverted group. All data were, however, skewed in the same direction. The assumption of homogeneity of covariance matrices of the dependent variable across groups was met [*Box's M* = 18.586, $F(6,19591) = 2.903$, $p = .008$]. However, the assumption of sphericity for phase was not met [Mauchly's $W = .608$, $\chi^2(2) = 25.412$, $p < .001$]. Therefore, the Huynh-Feldt adjustment was used for phase data. There was a significant main effect of phase [$F(2,1.494) = 96.573$, $p < .001$, partial $\eta^2 = .650$]. Pairwise comparisons using Bonferroni adjustments revealed that negative emotion scores were significantly different for all phase combinations, of note including the comparison between rest and recovery (all $p < .001$). The main effect for personality was non-significant [$F(1,52) = 3.028$, $p = .088$, partial $\eta^2 = .055$], as was the interaction effect [$F(2,1.494) = 1.550$, $p = .221$, partial $\eta^2 = .029$]. Figure 4-14 illustrates the results for negative emotional state.

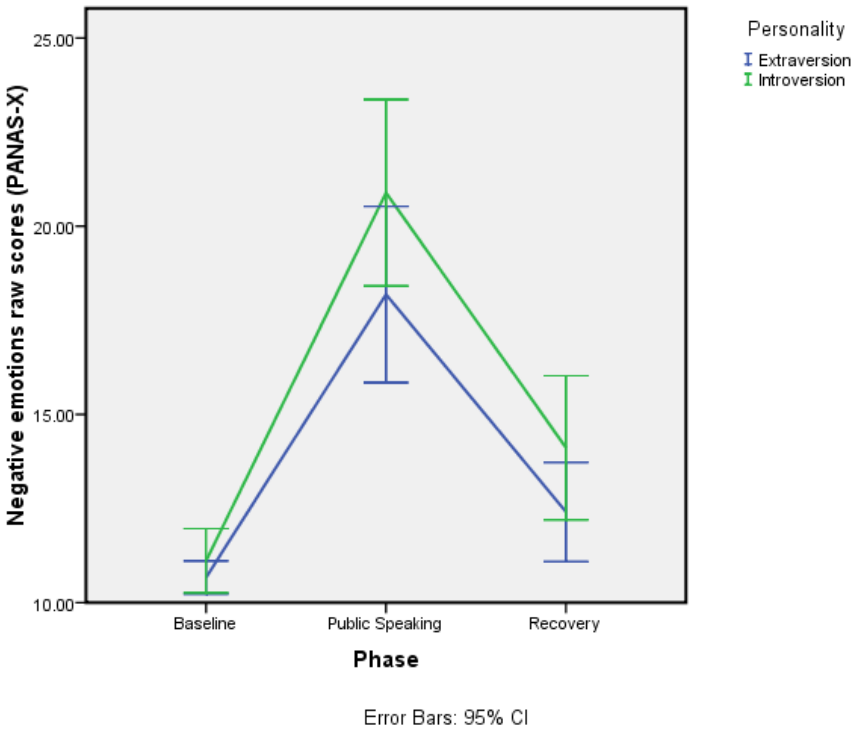


Figure 4-14. The distribution of state negative affect across experimental phases between groups

Fear of public speaking

Table 4-17 provides a snapshot of the experimental question followed by a summary of the descriptive data for fear of public speaking during the experimental public speaking stressor in Table 4-18.

Table 4-17. Dependent and independent variables for the statistical equations pertaining to Specific Aim 5

Dependent Variable	**Independent Variable 1**
Fear of public speaking	Personality (extraversion/introversion)

Table 4-18. Descriptive statistics for fear of public speaking (*PRCS*)

	Extraversion (n = 27)			Introversion (n = 27)		
Variable	*M*	*SD*	range	*M*	*SD*	range
PRCS	10.96	7.36	1-28	18.33	6.98	4-29

Note. Maximum score = 30
PRCS = Personal Report of Confidence as a Speaker

A one-way between-subjects ANOVA was performed on data reflecting fear of public speaking, relative to the public speaking task, as a function of personality (extraversion, introversion). The assumption of normality was met and no outliers were present. The assumption of homogeneity of variance was met [Brown-Forsythe $F(1,52) = .101$, $p = .752$]. There was a significant difference on fear of public speaking between personality groups [$F(1,52) = 14.25$, $p < .001$, $\eta^2 = .215$]. The introversion group rated their fear of public speaking for the speaking task in this study significantly higher than the extraversion group. Figure 4-15 illustrates the results for fear of public speaking.

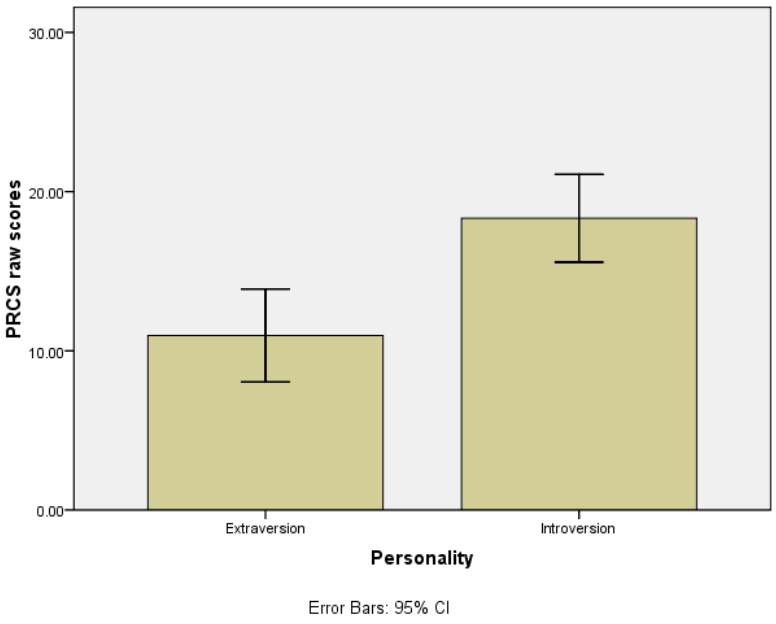


Figure 4-15. The distribution of fear of public speaking between groups

Rumination

Table 4-19 provides a snapshot of the experimental question followed by a summary of the descriptive data for rumination in Table 4-20.

Table 4-19. Dependent and independent variables for the statistical equation pertaining to Specific Aim 6

Dependent Variable	**Independent Variable 1**
Rumination	Personality (extraversion/introversion)

Table 4-20. Descriptive statistics for rumination

	Extraversion (n = 27)			Introversion (n = 27)		
Variable	*M*	*SD*	range	*M*	*SD*	Range
Rumination	18.63	6.73	7-30	20.67	5.27	9-30

Note. Maximum score = 35

A one-way between-subjects ANOVA was performed on rumination during the recovery period as a function of personality (extraversion, introversion). The assumption of normality was met and no outliers were present. The assumption of homogeneity of variance was met [Brown-Forsythe $F(1,52) = 3.80$, $p = .057$]. There was no significant difference in rumination between personality groups [$F(1,52) = 1.54$, $p = .221$, $\eta^2 = .029$]. Figure 4-16 illustrates the results for rumination.

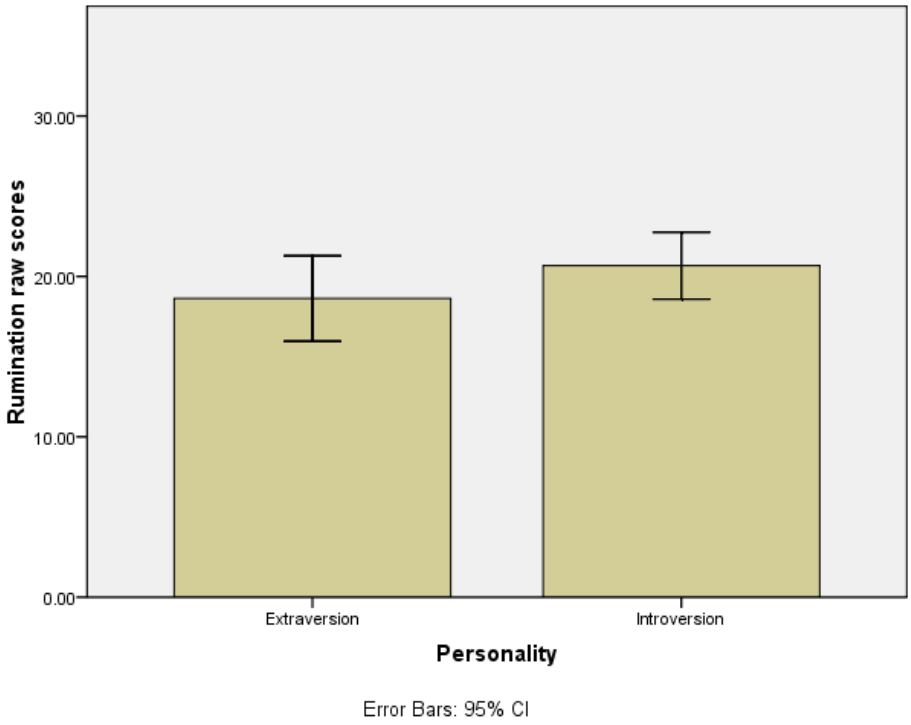


Figure 4-16. The distribution of rumination between groups

Summary and correlations

Table 4-21 summarizes the statistical results. This table includes additional statistical analyses performed on subgroups of the total sample guided by the pilot study and research questions,

which partly suggested that a differentiation by neuroticism will be important. The total sample was divided to obtain two strongly opposing groups: *non-neurotic* extraverts (n = 19) versus *neurotic* introverts (n = 19). The stress reactivity scale of the *MPQ-BF* was used with a cut-off set at the norm score. Additional results were included in the table if the p-value of a statistical equation improved when controlling for neuroticism in the total sample.

Table 4-21. Summary of statistical results

Variable	Main effect personality [a]	Main effect phase	Interaction
Primary outcomes[b]:			
Submental SEMG	p = .996 (.368)[c]	p < .001	p = .567
Infrahyoid SEMG	p = .033	p < .001	p = .083 (.076)
Anterior tibialis SEMG	p = .732 (.301)	p = .252	p = .237
Secondary outcomes:			
Vocal Effort	p = .050 (.037)	p < .001	p = .251 (.149)
F_0	p = .741 (.137)	p < .001	p = .784 (.283)
intensity	p = .407	p < .001	p = .424
Tertiary outcomes:			
SBP	p = .904 (.427)	p < .001	p = .506
Negative emotional state	p = .088 (.038)	p < .001	p = .221
Rumination	p = .221 (0.69)	-	-
Fear of Public Speaking	p < .001	-	-

Note. [a] All main effects for personality implied higher values for introverts as compared to extraverts
[b]SEMG data based on public speaking and reading of Rainbow Passage
c All values in parentheses represent additional statistics performed with subgroups controlled for neuroticism: non-neurotic extraverts (n = 19) versus neurotic introverts (n = 19)

The following table is a correlation matrix for the pooled study sample (Table 4-22). The correlation matrix focuses on the stressor (public speaking) phase. The goal was to detect how the main primary and secondary outcome measures correlated with personality and potential mediators such as SBP, negative emotional state, and fear of public speaking. Notably submental

SEMG did not significantly correlate with any other measure whereas infrahyoid SEMG did significantly and negatively correlate with extraversion and positively with the *VHI* and vocal effort. However, no significant or moderate correlations were found for any of the potential mediators with infrahyoid SEMG activity.

Table 4-22. Correlation matrix of the pooled study sample (n = 54)

Variable	E	SP	N	SR	VHI	PRCS	Neg.[a] emot.	SBP[a]	Voc. eff.[a]	SM[a b]	IH[a b]
E	-	.723**	-.311*	-.464**	-.485**	-.466**	-.218	-.079	-.213	-.029 (-.029)	-.258 (-.275*)
SP		-	-.309*	-.407**	-.457**	-.542**	-.293*	-.086	-.289*	-.131 (-.014)	-.163 (-.217)
N			-	.827**	.436**	.442**	.345*	.091	.337*	.154 (.168)	.027 (.021)
SR				-	.529**	.356**	.259	.177	.332*	.255 (.240)	.222 (.184)
VHI					-	.440**	.386**	.248	.355**	.180 (.129)	.377** (.427**)
PRCS						-	.735**	.077	.148	-.035 (-.132)	-.028 (.031)
Neg. emot.							-	.141	.201	-.073 (-.103)	.121 (.161)
SBP								-	.232	-.008 (.103)	.161 (.056)
Voc. eff.									-	.133 (.147)	.329* (.306*)
SM										-	.072 (-.115)
IH											-

Note. ** correlation is significant at the .01 level (2-tailed)
*correlation is significant at the .05 level (2-tailed)
E = extraversion; SP = social potency, N = neuroticism, SR = stress reactivity; VHI = Voice Handicap Index; PRCS = Personal Report of Confidence as a Speaker; Neg. emot. = negative emotional state; SBP = systolic blood pressure; Voc. eff. = vocal effort; SM = submental SEMG (50% max); IH = infrahyoid SEMG (50% max).
[a] correlations are based on data for the stressor phase
[b] first correlation based on activity during the standard phrase ("we were away"); correlation in parentheses based on activity for the first twenty seconds of public speaking

5.0 DISCUSSION

"In Britain, about 1 million people are believed to suffer from spider phobia; indeed, it is the second most common British phobia, after public speaking. That would make spider phobia the most common unreasonable fear in Britain. Public speaking, after all, entails at least some genuine social and career risks, but British spiders are usually harmless." (Beauregard & O'Leary, 2007, p.137)

The presented study aimed to assess the theory of the dispositional bases of vocal nodules and functional dysphonia (Roy & Bless, 2000a). This trait theory of voice disorders is rooted in psychobiological theories of personality (Eysenck, 1967; Gray, 1985). It claims that dispositions to react with behavioral activation versus behavioral inhibition, for example triggered by trait-specific sensitivities toward reward versus threat respectively, act as mechanisms that predispose extraverts to develop vocal fold lesions and introverts to develop muscle tension dysphonia (MTD). In the presented study, the focus was placed on examining especially those aspects of the theory that are related to behavioral inhibition and by extension possibly processes related to MTD. A stress reactivity task paradigm was chosen (1) to assess the widely presumed role of psychological stress on vocal behavior and (2) and to ensure the best possible match between personality and stressor sensitivities, i.e. stressor sensitivities known for introverted individuals such as sensitivity to threat. Stressor-induced increases in negative emotional state were used as an indicator of "perceived stress."

The specific aims and experimental questions were asked in reference to the causal model under investigation, shown graphically in Figure 2-6. According to this model, personal factors, specifically personality (introversion vs. extraversion) interact with situation (stressor) to influence emotional and cognitive reactions, which in turn drive autonomic nervous system (ANS) response, which further influence somatic responses such as laryngeal motor responses. Not shown in the figure, but implicit in the model are feedback loops such that ultimately, the model becomes non-linear. However, at this stage of inquiry the focus was the model's multiple linear tiers. The short-term goal was to provide preliminary information that will be useful towards the ultimate goal of complex model building including reverberative interactions.

The discussion of the results is organized in accordance with the model. To reiterate, the completed study assessed the assumption that person by situation interactions *do* influence laryngeal behavior (primary outcome, I) as well as corollary voice output parameters (secondary outcomes, II). At a tertiary and exploratory level (III), the study sought to obtain preliminary data around possible processes that may mediate effects anticipated from preliminary data, in terms of possible person by situation influences on (IIIa) emotional and cognitive reactions and (IIIb) ANS reactions, (IIIc) emotion and cognition's possible effects on laryngeal responses, and (IIId) associations between ANS functions and laryngeal responses. The discussion addresses these issues, in turn. Moreover, miscellaneous findings of relevance are discussed and conclusions are formed within the greater theoretical scheme. The chapter concludes with a discussion of limitations and future directions.

5.1 PRIMARY OUTCOMES

The primary question was whether people generated extralaryngeal reaction to perceived stress in a speaking task and if so, if the reaction depended on personality, on average. Besides discussing the phase x personality interaction, which was of central interest, it is important to discuss the main effect of personality as well. Of further interest was whether observed reactions were task-specific, i.e. speech related [extralaryngeal surface electromyography (SEMG): submental and infrahyoid] or general to the whole soma (leg SEMG: anterior tibialis). Relative to the first question – does perceived stress during a speaking task produce an extralaryngeal reaction – results showed that extralaryngeal SEMG for both submental and infrahyoid sites was lowest during silence (rest, anticipation, recovery) and significantly greater during speech production (baseline speech, public speaking, recovery speech). However, SEMG values for the sites were not significantly different within non-speech or speech phases. In other words, averaged across personality, stressor exposure during speech did not significantly increase extralaryngeal SEMG in comparison to SEMG values during speech without the stressor.

Relative to the role of personality in laryngeal SEMG activity, results indicated that personality predicted selected SEMG responses in *general*, i.e. across tasks. Specifically, infrahyoid muscle activity was significantly higher in introverts than extraverts as extracted from and substantiated with three different types of speech samples under both stressor and non-stressor conditions: reading of an all-voiced sentence, reading of the Rainbow Passage, and free speech during public speaking. Personality did not, however, predict submental SEMG activity. This disproportionately higher recruitment of infrahyoid musculature in introverts versus extraverts during speech, compared to submental muscle activity, is a key finding. This finding

transcends stress-related behavior and moreover hints at a general, potentially counterproductive, speech pattern in introverts. In addition, infrahyoid muscle activity arguably reflects more closely than the submental data phonation as opposed to articulation. In comparison, extraverts' extralaryngeal muscle activity, whether submental or infrahyoid, was remarkably stable across speech tasks both statistically and numerically, and seemingly balanced in that neither muscle group seemed to dominate over the other.

Against predictions, the phase x personality interaction turned out non-significant at the $p = 0.05$ level. However, descriptively, stress reactivity was observed in the introverted group during public speaking by way of a spike in activity in the infrahyoid musculature in comparison to baseline and recovery speech conditions, which was not seen in the same way in the extraverted group. In fact, this interaction was significant at the $p = .01$ level and further supported by the fact that partly but not only neurotic introverts, highly stress reactive individuals, were driving the results. In addition to the verbal public speaking stressor, the silent stressor anticipation phase functioned as a non-speech stressor. The SEMG data were also examined for signs of stress reactivity during anticipation, however no significant phase nor group difference emerged. Numerically, a slight trend in increased SEMG activity relative to rest was noticeable in submental activity only and only in introverts. Moreover, this muscle activity did not fully return to baseline during the recovery phase post public speaking and even stayed above the level of the activity seen during the anticipation phase. It is conceivable that this heightened activity during anticipation and recovery was linked to factors such as articulatory tension (e.g., tongue tension, teeth clenching), i.e. behaviors and movements that keep the submental muscle group engaged and the larynx elevated. Thus, while increased infrahyoid muscle tension was a key finding during speech production in introverts, marginally elevated

submental muscle activity was an interesting finding during phases of perceived non-speech stress.

As mentioned at the outset of this section, muscle activity was also concurrently recorded from the anterior tibialis muscle throughout the protocol. No significant findings emerged relative to phase, group, or phase x group differences. However, descriptively there was a noticeable spike in leg muscle activity during public speaking in the introverted group only. This spike paralleled the spike seen in infrahyoid muscle activity during public speaking for introverts. Therefore, a cautious interpretation is that the sequelae of exposure to a *speech* stressor are not only task-specific but to some degree general to the whole soma, at least in some individuals.

A general word of caution is indicated for the SEMG findings. SEMG data collections are fraught with challenges. A conscientious effort was made to normalize raw SEMG data in order to allow for a fair comparison of data across individuals. As a result, despite the challenges and a potentially remaining margin of error for the normalization process it is remarkable that *vocally normal* introverts significantly differed from extraverts in the magnitude of infrahyoid behavior during speech production. Infrahyoid activity was, in percent, markedly greater than submental activity which presumably created an imbalance of elevators and depressors. In contrast, the extraverts' extralaryngeal muscle activity, i.e. the activation of laryngeal elevators and depressors, was almost balanced in SEMG magnitude, relative to maximum force. The exact quantification of the combined muscular forces and their effect on laryngeal elevation versus depression is difficult to pinpoint. Unfortunately, the ratio of submental and infrahyoid activity can hardly be precisely quantified due to the challenges in obtaining absolutely accurate reference points for muscle activity, especially in laryngeal muscles. Theoretically, greater

infrahyoid activity over submental activity could tether the extralaryngeal framework and obstruct free laryngeal movement and it is not excluded that the same could be true for the opposite play of forces, i.e. greater submental over infrahyoid activity.

In summary, the significant personality difference in the magnitude of speech-related extralaryngeal muscle activity in introverts versus extraverts, and numerically the difference in muscle activation pattern, is *tentatively* interpreted as a sign of behavioral inhibition in introverts. Coactivation of extralaryngeal muscle groups may inhibit, or in other words, impede effortless speech production, which thrives on flexible laryngeal movements. The theoretically important discussion of behavioral inhibition will be picked up again in the conclusion section. Of further concern was the circumstance that such, even slightly, increased muscle activation was potentially carried through emotionally charged nonverbal phases such as anticipation and recovery and may contribute to chronic tension states.

5.2 SECONDARY OUTCOMES

The secondary questions asked whether there were changes in voice characteristics (perceived vocal effort; voice F_0; voice intensity) to perceived stress and if so, if the reaction depended on personality, on average. Again, before discussing the phase x personality interaction, which was of central interest, it is important to discuss the main effect of personality as well.

5.2.1 Perceived vocal effort

Reports of increased vocal effort and vocal fatigue with speaking are hallmark symptoms of voice disorders of many origins, although the full set of mechanisms behind such complaints remains unspecified (Hillman, et al., 1989; Lowell, Barkmeier-Kraemer, Hoit, & Story, 2008; Roy, Merrill, Thibeault, Gray, & Smith, 2004; Sapir, Keidar, & Mathers-Schmidt, 1993; E. Smith, Gray, Dove, Kirchner, & Heras, 1997; Solomon, 2007). Almost every patient with a voice disorder has these complaints, regardless of the specific pathology involved (Verdolini, 2000b). In the presented study, average vocal effort scores significantly increased across groups during the public speaking stressor. Moreover, vocal effort values throughout the protocol were significantly greater for introverts than for extraverts indicating a main effect for personality. However, again, the phase x personality interaction fell short of significance, although participants with introversion had descriptively greater vocal effort scores during public speaking than participants with extraversion. A common interpretative challenge that was well-represented here was that measures of vocal effort rely on a person's self-report and self-report is known to be influenced by a person's degree of neuroticism. At this point it should again be highlighted that the majority of introverted participants tended to be neurotic while the majority of extraverted participants tended to be non-neurotic. Indeed, moderate significant positive correlations were found in the total sample between vocal effort scores and neuroticism (Table 4-22). Thus, the results may have been, at least partly, driven by a participant's level of stress reactivity. The vocal effort scores were also significantly moderately negatively correlated with the social potency aspect of extraversion. This brief discussion highlights that the reconciliation

of so-called “subjective” and “objective measures” of vocal and laryngeal function gains in importance, as discussed shortly.

Remarkably, introverts also reported slightly elevated degrees of vocal effort during baseline readings as compared to extraverts. This tendency could be in line with notions that the introverts’ laryngeal system may be more vulnerable and more easily challenged than occurs for extraverts. For example, the baseline recordings required the continuous reading of the Rainbow Passage, which could have elicited a mild degree of vocal fatigue and in addition could have triggered an automatic switch to a “reading mode” that required more energy than normal reading (i.e. clear speech, projection). On the other hand and as discussed shortly, perceived increases in vocal effort could have been also the result of perceived mild performance stress at the beginning of the study. Lower baseline effort scores in this group for the repeated baseline speech during the recovery phase as opposed to the initial baseline speech, at least in part, support this notion.

Obviously, the stressor-induced increase in perceived vocal effort does not immediately reveal anything about the plethora of possible sources. For example, it is not readily clear whether perceived vocal effort stemmed from peripheral neuromuscular and metabolic processes (intra- or extralaryngeal, articulatory, respiratory, head and neck musculature, posture), biomechanical tissue fatigue, or central/mental effort (McCabe & Titze, 2002; Solomon, 2007). Most likely vocal effort reports were related to the cumulative toll of public speaking from various speech and voice production demands ranging from peripheral to central aspects (including perceptual ones). Only a fraction of sources have been investigated in this study to evaluate whether the subjective increase in vocal effort could be matched with any objective

changes. The main factors under investigation were personality, acoustic changes, and extralaryngeal muscle tension.

Vast individual differences have to be expected in the resistance and vulnerability to vocal effort and vocal fatigue (Solomon, 2007). High-degrees of within-group variability sometimes preclude the observation of significant group differences as seen in a study by Lowell et al. (2008), which compared teachers with and without self-reported voice complaints across various speech tasks including a simulated teaching task. Yet, the presented study demonstrated that if, for instance, personality is controlled for, group differences can be detected. The personality trait neuroticism is notorious for affecting a person's vulnerability to negative affect, to notice symptoms, and to feel compelled to report them (Suls & Martin, 2005). This tendency could also be seen in the study sample and as a matter of fact with any self-report measure used in this study as further outlined in this chapter. Still, there is a possibility that there were also physical responses as a function of personality that actually did increase physical effort to produce voice. A main goal of this study was to tease out these influences. As an aside, the current results are also in line with previously reported results that increased levels of vocal effort were found in vocally normal individuals high on social anxiety as compared to control subjects in response to mental imagery of valence and communication scripts (van Mersbergen, et al., 2008). This parallel is not surprising as neurotic introverts have a tendency to score higher on social anxiety (Dietrich & Verdolini Abbott, in preparation).

A couple measurement issues should be briefly addressed. There are various approaches to the assessment of vocal effort, which encompass direct magnitude estimation as used in this study (Wright & Colton, 1972), visual analogue scales (Lowell, et al., 2008), and an adopted Borg CR-10 scale for vocal effort ratings (van Mersbergen, et al., 2008). Basic challenges across

measurement instruments are that vocally normal individuals may not immediately understand the term vocal effort, that their mental representation of vocal effort may also encompass speech and cognitive effort, and that everyone's inner scale may differ. Of note, further direction beyond initial instructions was only provided if a participant asked for clarification at which point the participant was redirected to focus on "vocal" effort, the effort to produce voice. Ultimately, some vocal effort scores in any study sample may seem random, but more importantly the question remains to what extent vocal effort was physiologically valid. The next two sections focus on collected objective measures related to vocal and laryngeal function.

As an aside, data on contributions to perceived vocal effort are rare. The presented data on perceived vocal effort using direct magnitude estimation complement existing data by Colton on the relation between subglottic pressure and vocal effort (Colton, 1972a,b; Colton, unpublished).

5.2.2 Acoustic changes

Voice fundamental frequency (F_0) and voice intensity (dB) were tracked throughout the speech phases of the protocol. The all-voiced sentence "we were away a year ago" was used for analyses in an effort to obtain phonemically controlled content across participants and phases. Specifically, the first part of the sentence "we were away" was analyzed as many participants made a speech pause after "away," which in turn interrupted voicing. Results indicated that both personality groups reacted remarkably in parallel and thus, no group differences emerged. Voice F_0 and intensity significantly decreased during stressor exposure. On average, a drop in one semitone was noted. Descriptively, introverts already started out with a lower F_0 at baseline and

with a quieter voice. During the recovery speech at the end of the protocol, however, the groups performed almost identically as the introverts' F_0 and voice intensity increased resulting in a close match with the extraverts. The initial differences possibly were an indication, again, that the first baseline speech task already represented some level of psychological stress for the introverts from which they recovered at the very end of the protocol. A similar trend was seen in the vocal effort ratings. Hence, it was wise to have included both a baseline and a repeated baseline speech task in addition to the stressed speech.

The results are, however, somewhat at odds with the general notion in the literature that a hallmark of stress is increased F_0 and loudness (Juslin & Scherer, 2005). At the same time the literature on vocal expression of emotion paints a differentiated picture that allows for a multitude of acoustic results as described by Scherer (1986) and the hypotheses for this project anticipated this divergence from the general trend. According to the "component patterning" model of vocal affect expression (Scherer, 1986), fear and terror are linked to increases in F_0 by tapping an *activation* dimension in the model. However, if participants felt inhibited, taken aback, and not activated as the result of stress, the prediction of an increase in F_0 would probably vanish. This brief discussion illustrates that it is difficult to generalize vocal affect expression across situations and individuals and consequently the presented results would be tightly linked to the public speaking x personality situation.

Moreover, an intriguing thought was that the acoustic results could reflect some sort of role playing on the part of the participants who consciously or unconsciously tried to produce more "lawyer-ly" speech. This possibility cannot be excluded as the effects were seen for all participants. In a similar vein, this phenomenon could also be related to a general "public speaking mode" that may be similar across participants and includes a drop in voice fundamental

frequency. The broader literature on speech production should be further consulted for possible interpretations.

Physiologically and behaviorally (motivationally), the parallel drop in F_0 and voice intensity could be interpreted as a sign of a "holding back" and less drive of the laryngeal system during the social-evaluative stressor. It may be representative of the circumstance that the participants approached the task gingerly and cautiously. Many participants also exhibited glottal fry according to informal observations and they may have reverted to this speech pattern to a greater extent during perceived stress. A related notion was that, with the acoustic changes and the assumption of vocal fry, a decrease in vocal fold tension and a decrease in subglottic pressure may have occurred. This potential constellation of vocal fold muscular changes and aerodynamic changes would be inconsistent with either adducted or non-adducted laryngeal hyperfunction as described by Hillman et al. (1989), because either one would be expected to result in an increase in F_0. The interpretation of less laryngeal drive under the stressor condition appears most plausible and is supported by findings on respiratory function during various speech tasks in teachers with and without voice problems by Lowell et al. (2008). The authors found less driving pressure in teachers with voice problems during simulated teaching tasks as compared to structured speaking tasks. The simulated teaching tasks even had a built-in mini-social-evaluative component to increase ecological validity. In that study, the decreased driving pressure also resulted in a drop in loudness as compared to the control group.

5.2.3 The relation between changes in voice characteristics and SEMG

At this point of the discussion, the central question is whether the perceived increase in vocal effort for the introverted participants or the combined drop in voice F_0 and intensity for all participants were in any way related to changes in extralaryngeal muscle activity. Less drive in the laryngeal system as hypothesized based on the acoustic results could impede effortless voice production *over time* if, for example, respiratory support for voice production was lacking. Still, group differences in vocal effort cannot be sufficiently explained with the acoustic results, which did *not* indicate group differences. Also, only extralaryngeal processes were measured while intralaryngeal processes may have had an impact on acoustic changes as well, e.g. a decrease in vocal fold tension, i.e. *contractile* tension in the thyroarytenoid (TA) muscle. Further, relatively increased infrahyoid muscle activity may have pulled the larynx downward and aided in a drop in F_0, but the extralaryngeal pattern was different by group and yet the acoustic pattern was similar between the groups. This constellation is suggestive of the possibility that a tentative lack in phonatory drive during stressor exposure may have been accompanied by greater extralaryngeal tension in introverts than in extraverts. Arguably, further associated data on intralaryngeal and aerodynamic properties will be crucial to fill in the missing links.

The extralaryngeal SEMG results were pivotal for the interpretation of the study. Unique and crucial evidence was provided that links perceptual increases in vocal effort to objective increases in laryngeal muscular tension that could at least account for one *physiological source* of vocal effort (beside additional effects of mental effort and neuroticism). A significantly greater amount of infrahyoid muscle activity was found in introverts as opposed to extraverts throughout the protocol. As mentioned in the section on SEMG, such imbalanced muscular

activity in the anterior neck may likely have contributed to perceptions of vocal effort and may have been potentiated under conditions of stress. Indeed, infrahyoid muscle activity during stressed speech was significantly moderately positively correlated with vocal effort ratings (Table 4-22). In other words, the perception of increased vocal effort in introverts had at least for some individuals muscular underpinnings.

5.3 EXPLORATORY OUTCOMES

The last section will be devoted to the discussion of potential processes that may have instigated the participants' laryngeal and vocal behavior throughout the protocol. The following variables of this stress reactivity protocol were of interest: cardiovascular reactivity, emotional reactivity (negative emotional state), fear of public speaking, and rumination. First there was the question of whether there was a significant reaction to perceived stress in either variable -- systolic blood pressure (SBP), negative emotional state, or fear of public speaking -- and further, if the reaction depended on personality, on average. Second was the question whether SBP, negative emotional state, or fear of public speaking mediated relations between personality and phase on SEMG activity.

5.3.1 Cardiovascular reactivity

Autonomic blood pressure activity was measured every 90 seconds throughout the protocol. The comparison of group averages revealed no significant differences, but there was a main effect of

phase. As expected, SBP was lowest during the rest phase, increased significantly during non-stressed baseline speech production, further increased significantly during the *silent* stressor anticipation, and made another significant jump and peaked during the public speaking stressor. After the stressor, SBP significantly dropped yet remained significantly higher during the recovery phase as compared to the rest phase. With regard to personality differences, it was apparent that the introverts' SBP hovered over that of the extraverts for every single measurement, mostly in parallel with the general trend described above. However, actual group differences were minimal and not significant. It has been previously found that it is difficult to find statistical group differences during public speaking, for example in a study that compared public speaking under social-evaluative conditions versus public speaking with social-evaluative conditions removed. Only marginally higher SBP and heart rate were found for the group who performed the speech under social-evaluative conditions in that study (Gruenewald, et al., 2004).

Interestingly, group differences became most apparent during periods of anticipation, which in the wider sense not only refers to the stressor anticipation phase proper, but also to the last minutes of the rest phase as well as even arguably parts of the recovery phase (anticipation of future phases). Of note, participants were not informed about the various experimental phases beforehand. Everyone started out with reading tasks, which was consistent with the study advertisement. Thereafter, they were only instructed about the immediate phase ahead but not beyond. Thus, anticipation brewed for both groups towards the end of the rest phase, but was more pronounced for introverts than for extraverts. Suspense for the introversion group increased during the last three minutes of the rest phase and remained at this increased level throughout the anticipation phase while suspense gradually increased for extraverts during the last minute of the rest phase and peaked at the end of the anticipation phase. *Both* groups performed public

speaking under high levels of autonomic cardiovascular reactivity beyond what was seen during non-stressed reading. It is important to note that cardiovascular reactivity dropped for the last measurement of the stressor phase. There are two likely explanations. First, it was a difficult task to speak for the entire duration of five minutes and it was not uncommon for the amount of effective speaking time to drop towards the last minute. Secondly, a certain degree of habituation was likely taking place. Both groups probably became somewhat used to the situation and they knew that the five dreaded minutes would be over any time.

From a different angle, the last two minutes of the speaking stressor basically represented a transition towards recovery. Extraverts showed a gradual downward trend towards, but not quite reaching, baseline SBP during the recovery phase. At first, introverts showed a gradual protracted trend towards recovery as well, but the last two measurements during the recovery phase revealed a divergent path. The introverts' SBP started increasing again, most likely in anticipation of the subsequent – unknown - experimental phase.

Overall, autonomic cardiovascular reactivity, as measured by SBP throughout the experiment, painted a picture of a general underlying level of potentially increased sympathetic arousal for introverts. As a caveat, SBP is only a crude measure of sympathetic nervous system (SNS) activity and more precise measures of SNS activity are available, such as heart rate variability or heart pre-ejection period (Rushmer, 1989), which should be considered in future studies. For now, we know that plain speech production engages the cardiovascular system to some extent (Kleinow & Smith, 2006) as seen in this study, but furthermore, heightened cardiovascular activity probably represented a state of constant vigilance and unease throughout the protocol on the part of the introverts. Introverts probably viewed the *entire* experiment as a performance situation although care was taken to allow for acclimatization and adequately long

rest and recovery phases. Physiological arousal was especially apparent during any states of *anticipation*. The cardiovascular findings were in parallel with the course of negative emotional state throughout the protocol as described in the following section.

5.3.2 Negative emotional state and rumination

As expected for a stress reactivity protocol, negative emotional state significantly increased for both groups during the stressor as compared to baseline and validated the effectiveness of the experimental manipulation. However, there were no significant group differences. Descriptively however, there was a general trend toward greater increases in negative affect in response to public speaking for introverts, but within-group variability, most likely related to the trait neuroticism for either group made it more difficult to find a statistical difference.

In addition to stress reactivity, recovery characteristics were also a corollary interest in this study. Of relevance, both groups did not fully recover from negative affect during the recovery phase, especially introverts. Negative affect during the recovery phase was even significantly higher than during baseline. Relatedly, rumination was high during the recovery phase, but did not differ significantly between groups. It was hypothesized that the introversion group would score significantly higher on negative emotional state and rumination than the extraversion group based on experience from the pilot study. Numerically speaking, introverts did score higher, but only with a marginal difference, because both groups ended up scoring fairly high. In particular, the extraversion group scored higher than expected on both negative emotional state and rumination. In parallel with the results for state negative affect in response to the stressor and for the recovery phase, the main reason for the lack in difference probably lies

with the fact that both groups contained a mix of participants with and without neuroticism, which made the group difference dwindle. A crucial factor was that extraverts who were also neurotic were not excluded, which was not the case for the pilot study. Further, the significantly higher degree of public speaking anxiety in the introversion group interestingly did not translate into significant differences on negative affect and rumination for the groups. Instead trait neuroticism was a better predictor for the degree of negative emotional state and rumination.

A related explanation may be that the participants, especially *neurotic* extraverts, were more susceptible to cueing of rumination via the wording of the questions in the rumination questionnaire and as a function of their degree of trait and state negative affect. Rumination encompasses emotional and cognitive aspects and is clearly linked to one's degree of neuroticism (Siegle, et al., 2004). It has been previously shown that group differences in rumination across populations were mediated by the level of dysphoria even in nondepressed groups (Siegle, et al., 2004).

5.3.3 Fear of public speaking

As alluded to earlier, participants in the introversion group reported significantly more fear of public speaking in reference to the impromptu speech than their counterparts in the extraversion group. Relative to available normative data (Phillips, Jones, Rieger, & Snell, 1997), the introverted group scored .43 *SD* (63rd percentile) above and the extraverted group scored .45 *SD* (39th percentile) below the norm on the *PRCS*. Furthermore, the introverted participants -- as a group -- met the cut-off that was recommended by Paul (1966) for intervention for public speaking anxiety, which in essence consists of a desensitization program. The group data clearly

indicated that on average introverts had a higher comorbidity of fear of public speaking than extraverts. In fact, Beatty, McCroskey, and Heisel (1998) have proposed the "communibiological paradigm" that draws on Eysenck's and Gray's personality theories to explain communication apprehension. In this paradigm communication apprehension is conceptualized as a biological predisposition, which affects neurotic introverts disproportionally.

All the while, public speaking anxiety is known to be a strikingly common fear in the population. Prevalence estimates range from 24% to 34% (Cho, Smits, & Telch, 2004; Furmark, et al., 1999; Lawm, Schwartz, Houlihan, & Cassisi, 1994). Further, public speaking anxiety can be a circumscribed fear (Furmark, et al., 1999; Barry J. Ries, et al., 1998), but often is part of a social anxiety disorder. Social anxiety disorder, in fact, is among the most common mental disorders with a lifetime prevalence of 13.3% (Kessler et al., 1994 in Fresco, et al., 2001). Further, a social anxiety disorder may be primarily linked to fear and avoidance of *performance* situations, but in severe cases may also encompass fear and avoidance of *social interaction* situations in general (Mennin, et al., 2002). Due to apparent effects on quality of life it is not surprising that depression is a common comorbidity (Fresco, et al., 2001).

Therefore, it is not surprising that a comparison of strong introverts with strong extraverts revealed greater fear of public speaking in introverts than in extraverts. However, not every introvert or extravert has to fit this bill by default. A small number of cross-overs in either group illustrated that there were also introverts who did not experience much fear of public speaking and extraverts who did. A participant's degree of fear of public speaking was critical for the study, because the stressor was in fact public speaking.

5.3.4 Correlations with primary and secondary measures

A corollary question in this study was how the selected psychobiological measures correlated with each other, especially the variables that were of interest as potential mediators for significant differences in extralaryngeal SEMG, i.e. increased infrahyoid activity in introverts: SBP, negative emotional state, state fear of public speaking. Within-group individual variability was evident in either group and high- and low reactors were present in either group to the extent that some participants could be considered cross-overs in that they reacted more like a member of the opposite group. Consequently, it was reasonable to examine correlations in the pooled study sample and to screen the data for future eligibility for structural equation modeling as described in section 5.7.6. The correlation matrix focused on data on individual traits and data from the experimental stressor phase only (Table 4-22). First of all, all self-report measures for the entire sample were significantly correlated with the trait of neuroticism. This correlation was not surprising, but needed to be spelled out. There were significant moderate positive correlations among trait neuroticism and negative emotional stress reactivity, fear of public speaking, vocal handicap, and perceived vocal effort during the stressor phase. These correlations should not be underestimated as about 70% of the introverts in the current sample were neurotic introverts and about 30% of the extraverts were neurotic extraverts (Table 4-2). Extraversion, i.e. the independent variable personality, was significantly *negatively* correlated with fear of public speaking (strong) and negative emotional state (moderate), which was in line with notions that extraverts are socially and communicatively potent and high on positive emotionality (Gross, et al., 1998). In terms of extralaryngeal muscle activity, infrahyoid muscle activity was significantly negatively correlated with extraversion (moderate). No correlation for

submental muscle activity turned out to be significant, but the highest correlation that could be observed was a weak positive correlation between submental activity and stress reactivity. No significant correlations were found between the primary dependent variable SEMG and either negative emotional state, SBP, or state fear of public speaking (weak correlations only). However, in a separate correlation matrix for the introversion sample, SBP was significantly positively correlated with neuroticism. Moreover, especially state negative affect and fear of public speaking were not suitable candidates for covariates, because they were moderately to strongly correlated with the independent variable personality. As a potential mediator, SBP was least correlated with the independent variable personality, but did not significantly predict infrahyoid muscle activity.

In summary, extraversion was equivalent to less fear of public speaking and less negative emotional state during the public speaking stressor. Infrahyoid muscle activity was primarily linked to lower scores on extraversion (i.e. introversion). Thus, the degree of extraversion and relatedly fear of public speaking appeared to have an impact on a participant's infrahyoid muscle activity. Notably, a significant group difference could only be found for fear of public speaking, but not for state negative affect nor SBP. The most intriguing part of the correlation matrix was that infrahyoid muscle activity was significantly positively correlated with vocal effort. This finding provided crucial support for the fact that perceptions of vocal effort did in fact have some physiological basis and appeared to be rooted in personality differences. In conclusion of this section, it appeared that, at least with the current available data, autonomic cardiovascular stress reactivity did not play a major predictive role how individuals will use their laryngeal system under conditions of psychological stress. Although, both introverts and extraverts felt physiologically activated and negatively affected to some extent by the experimental stressor, the

extralaryngeal behavioral pattern differed as a function of personality. A cautious conclusion is that stressor-induced increased infrahyoid muscle activity in introverts was mostly driven by a trait-specific disposition to react with behavioral inhibition via activation of the somatic sensorimotor system, here interpreted as increased contractile tension working against voice production under conditions of social-evaluative stress, such as public speaking.

5.4 COROLLARY OUTCOMES

An important corollary finding of the study emerged during the screening process. Although only vocally normal participants were recruited for the study, introverted participants scored significantly higher on the *Voice Handicap Index* (*VHI*) than extraverted participants. Inclusion in the study was determined based on a participant's denial of a voice disorder in combination with a final clearance by the Principal Investigator based on cumulative evidence that no voice pathology was present which, however, did not include a laryngovideostroboscopic exam of the vocal folds. This difference in *VHI* scores as a function of personality was robust and was replicated in the total sample of individuals screened for a pilot study as well as the presented study (Dietrich, 2008).

The fact that introverts scored higher on the *VHI* at baseline is noteworthy, because of its potential for revealing how personality may be linked with a person's vocal status. The *VHI* was developed to quantify a patient's self-perceived vocal handicap and vocal disability in light of daily functioning and voice-related quality of life (Jacobson et al., 1997). There are three subscales that attempt to tap into functional, physical, and emotional consequences specifically.

The groups' means for the study reported here fell well in the normal range (introversion *M* = 16.89, *SD* = 11.28) given a ballpark mean of 33.69 (*SD* = 5.60) for a mild dysphonia as reported by the group who developed the questionnaire. However, introverted participants clearly scored closer to the mildly dysphonic range than extraverted participants (extraversion *M* = 7.48, *SD* = 6.42). Further, in relation to normative data available from individuals without a voice disorder, which indicated a mean of 8.75 (*SD* = 14.97) and median of 4 (Rosen et al., 2004), the extraverted group scored at the lower end of the normal range (median 6) while the introverted group definitely scored at the higher end of the normal range (median 16).

A closer look at the groups' response pattern was warranted. The top three questions that received the highest scores within each of the three subscales functional, physical, and emotional were identical across groups. Group differences were only evident in the *frequency* of the respective complaints within each subscale, which was consistently lower for the extraverted group. Summarizing the complaints, the most common "positive responses" were: (1) functional: low volume, lack of projection; (2) physical: change in voice quality; (3) emotional: emotional reactions to requests to repeat oneself or emotional discomfort with own voice. The complaint with the greatest clinical relevance had to do with a lack of vocal loudness or power. The complaints about changes in voice quality seemed less clinically relevant because participants often informally referred to their "morning voice" and the fact that they would change their pitch or voice depending on the communication partner (e.g., friend or teacher). The influence of personality on scores on the *VHI* became most apparent with regard to the emotional subscale. The questions about whether one feels annoyed/embarrassed when asked to repeat are not worded in relation to a voice disorder and thus, are highly unspecific. Instead, personality may strongly influence a person's response to these questions irrespective of voice problems. Further,

the personality trait neuroticism likely increases symptom reporting in any individual (Suls & Martin, 2005). This was evident in the distribution of scores while the main differentiation between higher scores on the *VHI* for introversion and lower scores on the *VHI* for extraversion remained in place. In other words, neurotic introverts scored higher than non-neurotic introverts, but non-neurotic introverts still scored higher than any extravert (Dietrich, 2008).

Correlations confirmed that scores on the *VHI* in the entire study sample were significantly strongly positively correlated with neuroticism, but also equally strongly negatively correlated with extraversion (Table 4-22). Hence, the fact that introverts scored higher on the *VHI* cannot be merely attributed to their propensity to worry, but may indeed be related to minor vocal complaints linked with the expression of introversion in general. Moreover, in light of the experimental stressor situation moderate positive correlations between the *VHI* scores and both vocal effort scores and infrahyoid muscle activity became apparent. This combination of correlations underscored the introverts' apparent vulnerability to experience vocal effort and laryngeal muscle tension.

In summary, on average, the responses on *VHI* questionnaires from introverted individuals -- more than extraverted ones -- may have hinted at vocal weaknesses and subclinical vocal complaints such as quiet voice and lack of vocal strength especially if challenged (e.g., when required to project or to talk over noise). Such a baseline difference in vocal constitution/physique or vocal behavior is not trivial. The questionnaire data suggested that introverts may be at a vocal disadvantage in comparison to extraverts who are traditionally thought to be naturally loud and outspoken, characteristics which were informally confirmed auditory-perceptually and formally by their specific *VHI* answers. The presence of a vocal disadvantage in introverts seemed to materialize in greater perceptions of vocal effort during

plain reading and speech tasks including the public speaking stressor meant to challenge the fine control of the laryngeal system in the face of an emotional stressor. It is disconcerting that for some introverts the step up to a situation in which frank voice problems emerge via vocal effort and vocal fatigue may not be far. Such problems may put them at risk in the long term depending on the social and professional demands placed on the individual.

5.5 CONCLUSION

The presented study aimed to address gaps in the literature around the relation between personality and voice disorders by systematically studying the personality-emotion-voice nexus with a psychobiological stress reactivity paradigm. Specifically, this was the second study after van Mersbergen et al.'s study (2008) to test the theory of the dispositional bases of voice disorders that was introduced by Roy and Bless (2000a). However, while van Mersbergen's study focused on emotion- and communication-charged mental imagery, the completed study was unique insofar it explicitly aimed to examine the relation between personality-dependent *stress* reactivity and vocal behavior.

Public speaking proved to be an effective stressor and although not a naturalistic one, it possessed high ecological validity as a vocal challenge. This study was the first study to provide evidence that (1) individuals used their extralaryngeal system differently as a function of personality with generally greater infrahyoid extralaryngeal muscle (re)activity in introverts than extraverts, (2) exposure to a public speaking stressor increased perceptions of vocal effort for most participants in the entire sample but significantly more for introverts, and (3) that the

person-by-situation mismatch (introverts-by-public speaking) played a central role in driving differential extralaryngeal somatic behavior that was interpreted as behavioral inhibition more than autonomic cardiovascular reactivity. The study also made the point that subjectively heightened vocal effort had at least partially an objective physiological basis in the form of increased infrahyoid muscle activity in introverts.

The study focused on introverts' behaviors more than on extraverts' behaviors, because the stress reactivity protocol aimed to trigger behavioral inhibition more than behavioral activation. In other words, the induction of threat and fear (and a sense of perceived *punishment*) was paramount and the study probably rarely, if at all, provided extraverts with a true sense of *reward*, which would have been necessary to investigate the effects of reward on behavioral *activation*. Indeed, by way of their personality composition extraverts were less affected by the prospect of public speaking, Nonetheless, many extraverts clearly felt an increase in negative emotions to the experimental protocol. Stated differently, extraverts appeared affected by the stressor induction as well, but they were characterized by greater resilience to the effects of stressor exposure. Due to the study design that was geared towards exploring the links between personality and MTD, a discussion of the results pertaining to extraversion and phonotrauma was largely forfeited at this point in time.

Overall, the "introversion-behavioral inhibition-MTD" limb of the trait theory of voice disorders (Roy & Bless, 2000a) could be confirmed to the extent that a significant group difference of greater infrahyoid muscle activity in introverts than extraverts was interpreted as a sign of behavioral inhibition. Behavioral inhibition has been described in the psychology literature as slowing, halting, or abrogation of ongoing behavior as the result of heightened conflict monitoring (Amodio, Master, Yee, & Taylor, 2008). The exposure of participants to

impromptu public speaking should and could have indeed triggered conflict monitoring activity in participating introverts. The neurocognitive process of conflict monitoring has been linked to heightened anterior cingulate activity and is thought to represent motivation for behavioral inhibition (Amodio, et al., 2008). Unfortunately, how the neural correlates of behavioral inhibition specifically network with the neural control of the larynx is still entirely new territory (see future directions). Arguably, one way to halt ongoing laryngeal behavior would be to impede it by obstructing laryngeal movement. A somatic (and not autonomic) response such as increased laryngeal depression during voice production may count as such a *behavior*.

Further, the initially proposed psychobiological framework for studying psychological stress and its relation to voice disorders (Dietrich & Verdolini Abbott, 2008) would have to be modified based on the current results to clarify that psychological stress primarily acts on laryngeal behavior through self-regulatory processes that affect behavioral motivations such as behavioral inhibition in introverts. The direct role of increased sympathetic nervous system activation on laryngeal behavior under conditions of stressor exposure will be tempered at this point although increased autonomic blood pressure activity during perceived stress was certainly a common corollary characteristic. However, personality-dependent, emotional, neurocognitive processes feeding somatic responses may play a far more prominent role for determining laryngeal behavior (Figure 5-1).

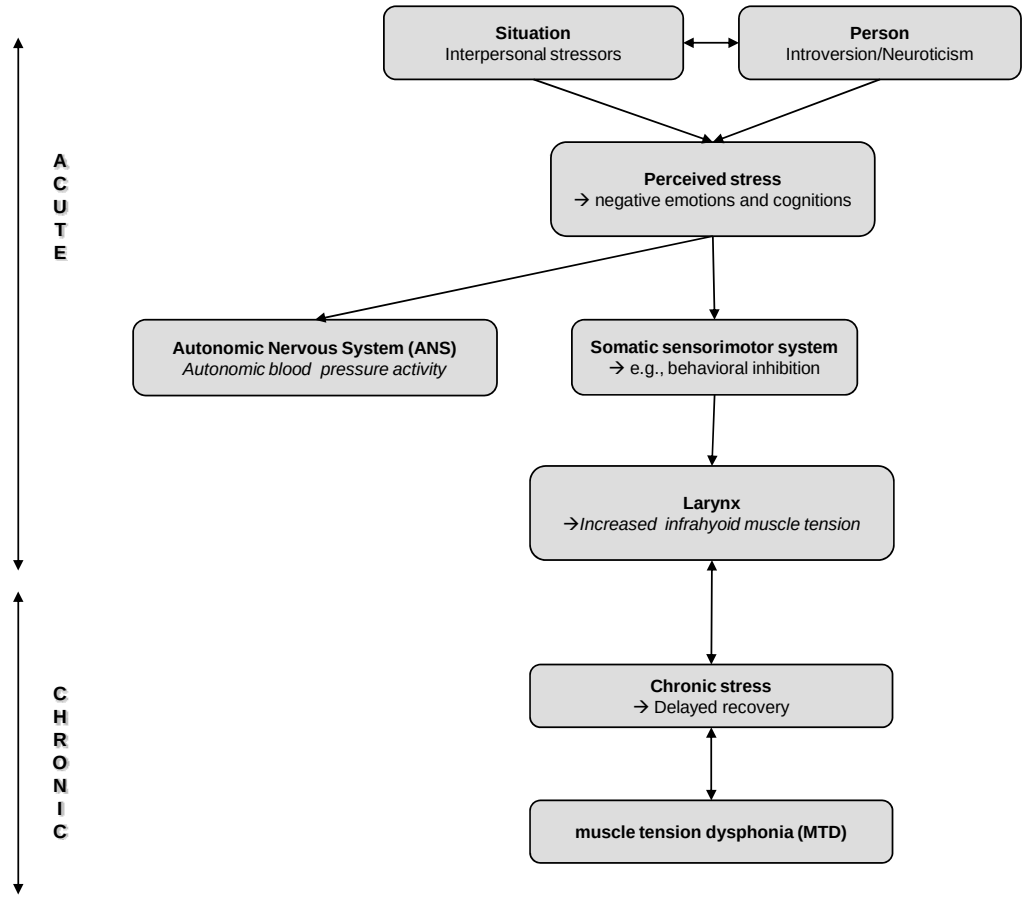


Figure 5-1. A psychobiological framework for studying stress and its relation to voice disorders. Adapted from Dietrich, M. & Verdolini Abbott, K. (2008). Psychobiological framework for stress and voice: A psychobiological framework for studying psychological stress and its relation to voice disorders. In: K. Izdebski (Ed.), *Emotions in the Human Voice* (Vol. II, Clinical Evidence, pp. 159-178).

A cumulative and meaningful finding of the study was that introverts appeared to be more vocally vulnerable in daily life as well as during stressor exposure as compared to extraverts. Introverts seemed less comfortable at being vocally and communicatively outgoing. Their laryngeal systems seemed to be easier to disturb under stressor exposure than those of extraverts. Everything comes down to the consideration that voicing becomes less efficient and more cumbersome under conditions of perceived stress especially for susceptible individuals such as highly neurotic introverts. Altogether, introverts may be at *risk* for future muscle tension-

related voice problems. However, the gestalt matters, i.e. the expression of introversion. A combination of markers as examined in this study may circumscribe an introvert's risk for future voice problems. The markers believed to be relevant are higher scores on the functional and physical subscales of the *VHI*, especially baseline complaints about a quiet voice and lack of power in one's voice; pronounced introversion in combination with pronounced stress reactivity (> 1 *SD*); and complaints of vocal effort during a vocal challenge test. In particular, the added factor of trait neuroticism was notorious. The destructive force of neuroticism was that it can heighten one's stress reactivity and contribute to a constant state of vigilance and worrying, which proved problematic if a person does not relax laryngeally during any nonverbal periods. Thus anyone lacking quick and full recovery may reinforce a vicious cycle and may be automatically at a higher risk for MTD.

The results are particularly promising, because group differences could be found despite a considerable amount of variability within each group and despite the fact that participants were vocally normal, healthy, and young individuals who scored psychosocially in the normal range. By extension, the results are probably only the tip of the iceberg and otherwise would be more marked in a psychosocially more severe sample (compare the section on limitations). The lower state of vocal functioning of introverts as a group – as described in this study - is of concern, because they may equally be of risk for a voice disorder as vocally hyperactive extraverts only the type of voice disorder may differ (MTD versus phonotrauma respectively). Introverts have not been traditionally targeted for voice education and so to speak form a neglected group. A key question in this context is to what extent an introvert's limited vocal constitution (quiet voice) is an anatomical or physiological given or the result of trait-specific vocal *behavior*, which in turn would be amenable to behavioral modification. The stance taken here is that a penchant to

laryngeal behavioral inhibition could be modified with resonant voice therapy, which trains effortless resonant voice that carries easily (Verdolini, 2000a). However, a person's vulnerability to stress reactivity, which likely affects laryngeal functioning, may not be solely addressed by voice therapy (see future directions).

At large, the research program has started to plot a continuum of psychobiological effects of exposure to a stressor on laryngeal functioning with supporting data from this study and a pilot study that recruited participants closer to the extremes. Risk and vulnerability to muscle tension-like vocal symptoms seem to lie on a continuum with introverts scoring psychosocially closer to the extreme being more prone to be affected.

Discussing the potential links between personality and MTD has a long tradition in the field of voice disorders. Unfortunately, such discussions have not been backed with solid experimental psychobiological research by the researchers interested in this area on the one hand, and have been neglected by those turning towards bench science in the field of voice disorders on the other hand (as an example of the extremes). Yet, such research is long overdue and the presented study represents a critical step forward. In addition, this study should encourage other researchers in the field to pursue multimodal (psychobiological) research. The combination of subjective and objective measures is timely and warranted in voice disorders research and will significantly broaden our understanding of human vocal behavior in a variety of situations. The completed research will be extended in a programmatic fashion as outlined in the future directions.

5.6 LIMITATIONS

The presented study has to be viewed in light of its limitations, which should be addressed in future studies. The main limitations center around the variability in the data linked to the study's inclusion criteria and the study's ecological and clinical validity. The selection of participants based on personality differences for the pilot study and subsequently for the main study was a learning process in itself. It involved decisions to be made around the (1) testing instrument, (2) the personality scales to be used, and (3) the cut-off scores in light of the research questions.

The *Multidimensional Personality Questionnaire – Brief Form* (*MPQ-BF*) (Patrick, et al., 2002) and the *Eysenck Personality Questionnaire – Revised* (*EPQ-R*) (Eysenck & Eysenck, 1994) were short-listed for this project. Both questionnaires have been previously used with patients with voice disorders (Roy, et al., 2000a; van Mersbergen, et al., 2008). The *MPQ-BF*, which is a tool for the investigation of the genetic, neurobiological, and psychological substrates of personality, was favored for the pilot study. The *MPQ-BF* is in its three-factor structure similar to Eysenck's personality model (Patrick, et al., 2002). However, while the *EPQ-R* provides scores only on the broad traits such as extraversion, neuroticism, and psychoticism, the *MPQ-BF* allows for a comprehensive analysis of personality using a range of broad as well as discrete traits: (1) Positive Emotionality (PEM): Well-Being, Social Potency, Achievement, Social Closeness; (2) Negative Emotionality (NEM): Stress Reaction, Alienation, Aggression; and (3) Constraint (CON): Control, Harmavoidance, Traditionalism. Yet, challenging was that no trait is specifically labeled "extraversion." The general notion is that positive emotionality is correlated with extraversion while negative emotionality is related to neuroticism (Church, 1994; LaRowe, et al., 2006; Larsen & Ketelaar, 1991). The core of PEM is considered Well-Being

(Patrick, et al., 2002), but in the pure interest of interpersonal aspects of PEM related to extraversion, the average of scores on Social Potency (PEM-agentic) and Social Closeness (PEM-communal) have been found to be the closest match in a comparison of various personality questionnaires (Church, 1994; Patrick, et al., 2002). The core of NEM is Stress Reactivity, which coincides well with neuroticism as measured in other personality questionnaires (Church, 1994; Patrick, et al., 2002).

In summary, the initial review of personality questionnaires resulted in a preference for the *MPQ-BF*, because of its highly differentiated structure and focus on emotional dispositions, which have been highlighted as a crucial factor in patients with voice disorders. Further, it was possible to map scales from the *MPQ-BF* onto the personality constructs extraversion and neuroticism. Lastly, a choice had to be made where to set the limits for extraversion and introversion in order to achieve an optimal differentiation between the groups under investigation. The main goal was to recruit participants who were stable extraverts and introverts, i.e. scoring reliably above versus below the norm on extraversion. The cut-off was set to an experimental 0.5 *SD* above or below the norm on extraversion. Test-retest data from the pilot study have retrospectively shown that the created buffer zone was effective in avoiding overlap between groups as participants' scores may slightly vary over time (Dietrich & Verdolini Abbott, in preparation).

However, limitations became apparent when a different personality questionnaire, the *EPQ-R*, was used to screen for extraversion for the main study and when neuroticism was not controlled as was the case for the pilot study. The effect was a partial loss of differentiation between groups and increased variability within the groups. The *EPQ-R* replaced the *MPQ-BF* as screening instrument for the main study, because it is widely accepted in the psychology

literature for assessing extraversion whereas the *MPQ-BF* assesses extraversion indirectly as part of positive emotionality. Corresponding data on the *MPQ-BF* were intentionally retained in order to correlate extraversion with other emotional subscales and to learn more about adequate selection criteria for future studies. Corresponding scores on both questionnaires revealed that the normal curve was slightly shifted depending on the questionnaire. For example, a -.5 *SD* for extraversion on the *EPQ-R* may result in a score above the norm on "extraversion" on the *MPQ-BF* and vice versa. In short, using the .5 *SD* criterium for the *EPQ-R* bears a risk for cross-overs between groups. In other words, the *MPQ-BF* is more sensitive at recruiting stable extraverts and introverts with a .5 *SD* buffer zone, whereas it would be advisable to increase the buffer zone to at least .7 *SD* when using the *EPQ-R*.

On the topic of neuroticism, two maximally opposing groups were formed for the pilot study, which were *neurotic* introverts versus *non-neurotic* extraverts. For the main study on the other hand, the study design was streamlined and participants were selected based on their score on extraversion only. As expected and hoped for, the trend in the data was that most introverts were also neurotic and most extraverts were non-neurotic. However, on the flip side about one-third of the participants in either group did not fit this pattern (based on stress reactivity on the *MPQ-BF*) and to complicate matters the personality instruments differed in their norm curve for neuroticism. As illustrated in Table 4-1, the *EPQ-R* underestimated the degree of neuroticism, which was most evident for introverts. It could be argued that the *MPQ-BF* overestimated the degree of stress reactivity in the groups, but a conservative standpoint is chosen for this discussion. The fact that about one third of the extraverts could be considered neurotic probably increased reactivity in the extraversion group to such a degree that some of the anticipated group

differences dwindled. Such an effect is reasonable as neuroticism, state negative affect, and rumination are tightly linked (Suls & Martin, 2005).

In summary, there was a small portion of heterogeneity present within each group, which could have been better controlled with stricter personality inclusion criteria. Moreover, statistical power was based on the pilot study, which did control for neuroticism. Hence, the presented study was insofar limited as the power to detect interactions was not optimal with neuroticism not being controlled for and inclusion criteria for extraversion being more lax. The summary table at the very end of the Result Chapter presented *p*-values from additional statistical analyses that split the sample into non-neurotic extraverts and neurotic introverts. In fact, controlling for neuroticism highlighted the already existing trends, mostly significant main effects of personality throughout the study. Clearly, the role of extraversion in combination with neuroticism should be investigated with a carefully powered study that clearly differentiates between neurotic and non-neurotic introverted and extraverted individuals respectively. In fact, it is known that personality studies in general require large samples beyond what is unfortunately commonly seen in studies in Communication Sciences and Disorders.

From a clinical point of view, several limitations have to be addressed. This study claims to be relevant for patients with MTD, but several factors limit its generalizability to voice disordered populations. The completed research has been executed with vocally normal participants who were mostly college students. It has not been shown that the presented results can be generalized to patients with MTD, which includes the unresolved issue if disproportionate activation of laryngeal depressors as was seen in this study is equivalent to the laryngeal pattern seen in patients with primary MTD (compare section 1.1.3 on muscle tension patterns in MTD).

An acute stress response in vocally healthy individuals may present differently than chronic laryngeal muscle tension.

The study would possess greater ecological and clinical validity had a middle-aged female cohort been recruited, because patients with MTD are typically middle-aged women. However, besides the age factor, middle-aged women in general and patients with MTD in particular, would have a higher chance of less than perfect health and a variety of comorbid conditions. For example, participants may not pass the hearing screening, be obese, have untreated or treated hypertension, smoke, take antidepressive or anxiolytic medications, or have other medical conditions such as rheumatoid arthritis only to name some criteria. A central issue is that many patients with MTD would not even have qualified for the current study, because of treated or untreated clinical depression. Furthermore, if participants high on neuroticism will be sought for future studies, a greater degree of depression in the study population will likely be seen. However, excluding such patients or vocally healthy women would be a distortion of reality. Instead, besides screening for depression, another level of clinical screening would have to be ensured, which could differentiate between clinical depression versus high levels of worrying. To offset the effects of depression on symptom reporting, multimodal experiments, which include both self-report and objective measures, will be crucial.

A miscellaneous caution in the interpretation of the data has to do with the speech tasks throughout the protocol. The rationale for the speech tasks was outlined in the section on methods. However, it is true that repeated reading of a sample (e.g., Rainbow Passage) in itself may alter responses. Care will have to be taken in future studies to consider the cost and benefit ratio of selecting speech tasks and the sequence of them.

5.7 FUTURE DIRECTIONS

The here presented study "the effects of stress reactivity on extralaryngeal muscle tension in vocally normal participants as a function of personality" is embedded in a research program that aims to systematically investigate the relation between personality and voice disorders. Within that research program, a particular focus is placed on the role of perceived stress on health and disease, in this case vocal pathologies. Progressive research goals have been proposed at the outset of this study. The results of this experiment constitute novel contributions to the complex questions at hand and provide a springboard for future research. Extensions of the presented research aligned with the research program will be discussed in the following sections.

5.7.1 Investigate *effects* of exposure to an acute stressor on laryngeal function

Only a fraction of the effects of exposure to an acute stressor on laryngeal function has been investigated in this study. The main outcome measure was *extra*laryngeal SEMG of the anterior neck, which means that no immediate insight can be gleaned about concurrent stressor-induced *intra*laryngeal changes. Yet it will be important to investigate in future research, which intralaryngeal *patterns* emerge as a function of personality and stressor. Laryngeal hyperfunction is frequently referred to as if it is a single phenomenon. However, at least two broad muscle activation patterns, *nonadducted* hyperfunction and *adducted* hyperfunction, have been proposed that are thought to differentiate patients with MTD from patients with phonotrauma respectively (Hillman, et al., 1989). Roy and Bless (2000a) further postulated that interindividual differences

in laryngeal patterns have their roots in personality differences driven by inclinations toward behavioral inhibition (introversion) and behavioral activation (extraversion) respectively. Unfortunately, it cannot be claimed without doubt if the observed SEMG activity in this study correlated on the one hand with stressor-induced increased muscular activation in the vocal folds and on the other hand with different intralaryngeal patterns as a function of personality or perceived stress.

A variety of direct and indirect measures of laryngeal function are available. The following discussion is not exhaustive. In terms of *direct* approaches, laryngeal intramuscular needle EMG in humans, for example of the thyroarytenoid muscle, would help to clarify the changes in muscular activation with respect to perceived stress, but would fail to provide information about related vocal fold configurations and biomechanical changes. Nonetheless, another valuable contribution of needle EMG would be to investigate if stress induction leads to processes that mimic vocal fold muscle fatigue (e.g., spectral compression) (Solomon, 2007). However, the feasibility of a stress reactivity protocol with a speech portion and concurrent needle EMG is unclear to date as needle EMG in itself is a stressor. Without doubt, more research in the area of laryngeal neurophysiology will be necessary to move our understanding of phonatory processes in light of stressor exposure forward.

Direct approaches in humans are limited and the study of the role of stress on laryngeal muscle biology may require animal models. Some biological changes of interest in the intrinsic laryngeal muscles are (1) neuromuscular changes such as sympathoneural activity, (2) metabolic changes such as fluctuations in blood flow, and (3) neuroendocrine changes such as shifts in hormones, all of which may have an impact on laryngeal functioning. For instance, various theories in the physical therapy literature on muscle fatigue cite depletion of energy stores,

accumulation of metabolic waste products, and dehydration as critical factors in muscle fatigue (McCardle, Katch, & Katch, 2007; Waters, 2004). Within voice research, individual differences in oxygen consumption during various voice and speech tasks have started to receive attention so to explore one source of vocal fatigue (Nanjundeswaran, Verdolini, Van Swearingen, & Gartner-Schmidt, 2007). In addition, psychoneuroimmunology (Kemeny, 2003; McEwen, 1998) would probably be relevant for the voice domain especially with respect to phonotraumatic changes and laryngeal wound healing.

Indirect measures can be employed to infer intrinsic laryngeal processes (Hillman, et al., 1989; Solomon, 2007). For example, aerodynamic function assessment and electroglottography (EGG) provide information about subglottic pressure, transglottal airflow, and vocal fold contact patterns (Hillman, et al., 1989; Lowell, et al., 2008). Hillman et al. (1989) illustrated how configurations of subglottic pressure and airflow characteristics mapped onto nonadducted and adducted laryngeal hyperfunction. Further, phonatory threshold pressure, the least amount of subglottic pressure to initiate phonation (Verdolini, et al., 1994), is a valuable measure well related to the perception of phonatory effort (Chang & Karnell, 2004; Solomon, 2007). With regard to vocal fold contact patterns, recently EGG data have been for the first time successfully collected from *continuous* speech by Lowell et al. (2008).

A more direct approach to the investigation of vocal fold vibration and closure patterns during phonation is laryngeal endoscopy. A videolaryngostroboscopic exam is currently the gold standard in voice centers (Colton, et al., 2006). However, vocal folds vibrate around 200 Hz per second in the average female and stroboscopy gives the examiner only an illusion of vibration and vocal fold contact by averaging vocal fold motion from various points during the vibratory cycle. Fortunately, as a valuable source of information high speed imaging of vocal fold motion

at a rate up to 4000 frames per second has come within reach for research purposes. The jump in temporal resolution will likely greatly refine our understanding of vocal fold vibratory function in humans with and without voice disorders.

Only sparse high-speech imaging data on patients with voice disorders are available at this point in time. Relevant for the presented research program at hand, a study sample of eleven patients with non-organic MTD revealed that a common characteristic was *adducted* hyperfunction with unique vibratory modes (Patel, 2006). The patients with MTD presented with an increased closed phase and pressed voice quality. Those findings are in contrast to Hillman et al. (Hillman, et al., 1989) who reported that *non-adducted* hyperfunction was a hallmark of MTD assuming a true non-organic origin and course of the vocal pathology. Initial high speed imaging data do not support this model. It may be that stroboscopy tricks the eye and falsly suggests non-adduction or that those patients with MTD did not include anyone with "true" non-adducted hyperfunction. Further, it is possible that both patients with phonotrauma and MTD present with adducted hyperfunction. However, the distinguishing feature may be the degree of collision forces acting on the vocal folds, which then would expected to be higher in patients with phonotrauma than in patients with MTD. High speed imaging will significantly add to the knowledge base in voice science. It will allow for a more accurate representation of vocal fold vibratory behavior and moreover it will be possible to correlate the images, for example, with data from EMG, EGG (electroglottography), or intralaryngeal pressure sensors to measure collision forces. Unfortunately, limitations exist. High speed imaging is costly and requires large storage capacities. Typically, not more than an eight-second portion is recorded. However, it will be a valuable tool to examine selected patients with voice disorders.

Another relevant measure in future psychobiological studies would be respiratory function. The *interaction* between phonation and respiration has not been pursued in the presented study and even rarely in voice disorders research in general (Lowell, et al., 2008). The respiratory and laryngeal subsystems are inextricably linked during speech production (Kent & Read, 2002). Further, the muscle tension in MTD may involve widespread dyscoordination of not only phonatory, but also respiratory, resonatory, and articulatory gestures (e.g., tension in mandible and tongue, shallow breathing) as well as tension in the upper body (head, neck, and shoulders) (Kinzl, et al., 1988; Morrison, et al., 1986; Morrison & Rammage, 1993). Thus, a dysregulation of breathing and voicing, such as speaking while breathholding as an extreme example, could conceivably increase phonatory effort.

One recent study on respiratory and laryngeal function during spontaneous speaking in teachers with and without voice complaints aimed to investigate sources of vocal fatigue and illustrated how inefficient use of lung volume may be linked to perceptions of vocal effort (Lowell, et al., 2008). The authors found evidence that individual differences in speech breathing exist, which may contribute to vocal fatigue. The teachers with voice complaints used smaller lung volumes at the beginning of breath groups and completed the breath groups with smaller lung volumes as compared to teachers without voice problems. Besides structured phonation and speech tasks, experimental tasks included a conversation and simulated teaching sample as well. Interestingly, the simulated teaching sample was actually performed under social-evaluative stress conditions to increase the ecological validity of the experiment, but the study was not conceptualized as a psychobiological study. Regardless of the exact reason for the apparent group differences, the clinical significance of speaking at smaller lung volumes has to do with the increased cost for muscles linked to the need for greater expiratory muscular pressure (Forner

& Hixon, 1977 in Lowell, et al., 2008). The study is an important contribution, because it supported the notion that perceived symptoms of vocal effort have in fact a physiological basis. In addition, support for a respiratory-laryngeal interaction was found numerically to the extent that contact phase asymmetry (contact-closing versus contact-opening phase) in teachers with voice problems may exacerbate vocal complaints by increasing vocal fold tension at the end of breath groups (Lowell, et al., 2008).

The last point to be made in this section should be devoted to the distinction between acute stress and chronic stress. It is important to learn what stressors are capable of doing to the laryngeal system acutely, but eventually voice psychobiological research will also have to turn towards investigating the effects of *chronic* stress on the larynx and vocal functioning. Changes may persist and even may have cumulative effects over time that need to be explored in the context of a a person's voice disorder.

5.7.2 Identify psychobiological *mechanisms* that underlie changes in laryngeal function subsequent to exposure to a stressor

The detailed investigation of psychobiological mechanisms that underlie changes in laryngeal functioning as a function of personality is a new frontier in voice disorders research. Such research should decidedly encompass the entire processing spectrum, i.e. from central mechanisms to peripheral mechanisms. In this section, first, potential peripheral mechanisms will be outlined followed by central mechanisms.

Research investigating the stressor-induced changes in intralaryngeal muscle physiology appears nonexistent. Mostly, voice researchers focus on the phenomenon of laryngeal muscle

"tension", because it is the proximal symptom of MTD and thought to be related to the omnipresent problem of vocal fatigue in a wide array of patients with voice disorders. However, increased muscular tension represents only one aspect of the multitude of changes potentially occurring during stressed speech. For example, vocal effort is likely the result of a combination of peripheral physiological events in the vocal folds (besides mental vocal effort). It has been established that the SNS has neural connections to the larynx (Hisa & Sato, 1991), but specific *functional* neuromuscular changes in the vocal folds in response to an experimental stressor have not been traced to the present knowledge. Further, a host of physiologic, metabolic, and hormonal changes could be expected such as changes in blood flow, changes in oxygen saturation, and changes in the viscosity of secretions. With regard to psychoneuroimmunology, knowledge about the effects of psychological stress on laryngeal wound healing could be another piece of the puzzle in the quest for the pathogenesis of and recovery slope from vocal fold lesions. Overall, all laryngeal changes combined could drive changes in laryngeal functioning such as vocal effort and vocal fatigue.

Central nervous system (CNS) activation represents the top of the processing chain for voice production (Ludlow, 2005). The investigation of CNS control of phonation in humans away from research in animals is an ongoing research effort. Such research is also complex, because brain activation differs depending on the laryngeal task at hand including (1) vocal expression of emotions, (2) voice for speech, and (3) breathing, swallowing, and cough (Ludlow, 2005). The current model of vocal control derived from animal research describes two neural pathways, which are the (1) limbic cingulo-periaqueductal pathway (phylogenetically older vocalization system: readiness to vocalize, vocal expression of emotions) and the (2) motorcortical vocal control pathway (phylogenetically newer vocalization system: patterning of

vocal utterances, learned speech) (Jürgens). In addition, central pattern generators are active for reflexive movements such as breathing (Ludlow, 2005). The limbic vocal control pathway runs from the anterior cingulate cortex (ACC) to the periaqueductal grey (PAG) before entering the reticular formation that has direct connections with the phonatory motoneurons. In contrast, the motorcortical vocal control pathway runs from the laryngeal motor cortex directly to the phonatory motoneurons via the reticular formation. In addition, subcortical feedback loops involving the basal ganglia and cerebellum furnish preprocessed information to the motor cortex via the thalamus for voluntary fine motor control. Thus, it bypasses the ACC and PAG. Moreover, the reticular formation in the pons and medulla oblongata serves to integrate not only laryngeal but also respiratory and articulatory functions and hence is a critical relay station for speech.

At this point, detailed knowledge about the neural control of *various* vocal tasks as well as the precise *interactions* among emotional, volitional, and reflexive aspects of voice production in humans is largely missing (Jürgens; Ludlow, 2005). Of note, both the limbic and the neocortical vocal control pathways have been found to be activated during voluntary vocalization in humans (Schulz, Varga, Jeffires, Ludlow, & Braun, 2005). The precise interactions between the subcortical animal vocalization system and the exquisitely fine controlled neocortical system should be at the center of investigations around the relation among personality, stress reactivity, and neural laryngeal control. It has long been hypothesized that heightened negative emotional states would result in a strong interference of the limbic vocalization system with the fine control of the voluntary vocalization system (Aronson, 1990). Fortunately in this current day and age, researchers are empowered with the tools to observe potential stressor-induced shifts in the neural control of vocalization in humans.

Various neuroimaging techniques are available that differ in spatiotemporal resolution (Huettel, Song, & McCarthy, 2004). Of the available techniques, functional magnetic resonance imaging (fMRI) of vocal control in humans is a logical next step (Ludlow, 2005). fMRI to image neural activity in the brain in the context of human behavior is a young technology and comes with its own set of challenges. It is widely being used to investigate *non-vocal* cognitive paradigms (Joseph, et al., 2006) and it is increasingly being used with stress reactivity task paradigms mostly with interest in cardiovascular psychophysiology (Gianaros, et al., 2005; Gianaros, Jennings, Sheu, Derbyshire, & Matthews, 2007). However, using fMRI for voice research is yet another frontier within fMRI research, because voicing and even more so speaking can cause severe motion artifacts (Huang, Carr, & Cao, 2001). Currently, sophisticated task protocols are being designed and tested and different types of fMRI are used to tackle the challenges (Huang, et al., 2001; Özdemir, Norton, & Schlaug, 2006; Wang, et al., 2007). Undoubtedly, the use of fMRI in voice and voice disorders research bears great potential to advance our understanding of the functional neural networks subserving voice production under several conditions including exposure to a stressor.

Findings from fMRI studies that have investigated neural correlates of stress reactivity will be helpful to form hypotheses about neural networks subserving vocalization under conditions of perceived stress as a function of personality. For example, well fitting with the context of the presented research, neural correlates of speech *anticipatory* anxiety have been described in males with and without generalized social anxiety (Lorberbaum, et al., 2004). Group differences in brain activity were found. Subcortical and limbic regions responsible for emotional processing (pons, striatum, amygdala, uncus/anterior parahippcampus, insula, temporal pole) were far more involved in individuals high on social anxiety than cortical regions

responsible for cognitive processing (dorsal anterior cingulate, prefrontal cortex). This study is an excellent example how fMRI research can substantiate anecdotal knowledge, in this case that a person can become so anxious that he or she cannot think clearly and by extension – which would have to be investigated in future research -- cannot phonate clearly. At a more fundamental level, gender differences in neural response to psychological stress have been revealed (Wang, et al., 2007). During performance of a verbal mental arithmetic stressor, males showed increased cerebral blood flow in the right prefrontal cortex (negative emotion, vigilance, goal-directed behavior) and decreased blood blow in the left orbitofrontal cortex while females' blood flow primarily increased in the limbic system (ventral striatum, putamen, insula, cingulate cortex).

With regard to cardiovascular psychophysiology, the ACC (perigenual, mid-anterior, posterior (BP), dorsal (HR)) has been shown to be a neural correlate for heightened blood pressure and heart rate during a stressor, i.e. the ACC was involved in autonomic control during cardiovascular arousal (Critchley, et al., 2003; Gianaros, et al., 2005; Gianaros, et al., 2007). Other co-activated brain regions were the bilateral prefrontal cortex, insula, thalamus, PAG, and cerebellum (Gianaros, et al., 2005; Gianaros, et al., 2007). Overall, both cortical and subcortical brain regions were involved during stress reactivity (Critchley, et al., 2003).

All in all it should be apparent that there is overlap of neural networks recruited during times of stress reactivity and neural networks implicated during voice production. More importantly, the "stress networks" (Wang, et al., 2007) overlap with the limbic vocal control system, which suggests that vocal fine motor control may be lost under conditions of stress, because of dominating activity in the limbic system. However, it is unclear what the precise effects on vocal fold behavior would be and how stressor exposure and increases in laryngeal

SEMG and vocal effort would map onto cortical and subcortical brain function. The overarching future goal will be to investigate the functional brain network for vocal control under stress and the role of underlying personality differences. In parallel with goals expressed in the cardiovascular psychophysiology research, future work would test among other aims if heightened cardiovascular reactivity would represent a "neural phenotype by which psychological stress and other factors associated with exaggerated blood pressure reactivity may increase risk for" (Gianaros et al., 2005), e.g. voice disorders (Gianaros, et al., 2005). In conclusion, neuroimaging would allow for a closer look at the central mechanisms driving changes in vocal control.

In the future, the role of the ANS or the somatic sensorimotor system as mediators of laryngeal muscle tension has to be better differentiated. Therefore, more precise indicators of the parasympathetic and sympathetic nervous systems will have to be used alongside indicators of the somatic sensorimotor system, i.e. brain mapping of neural networks subserving laryngeal activation as described above.

5.7.3 Identify individuals at *risk* for psychobiologically mediated voice disorders

In short, the question being posed here is if some individuals are at risk for certain vocal pathologies "simply" because of their personality. Personality can be conceptualized in form of psychobiological tendencies (Eysenck & Eysenck, 1994; Gray, 1985; Patrick, et al., 2002). In turn, someone's vocal pathology could be considered psychobiologically mediated -- a psychosomatic manifestation so to speak – as long as no obvious contributing etiologies such as

of an iatrogenic or neurological nature prevail. Clinically speaking however, we as speech-language pathologists specialized in voice are not at a point to be able to confirm without doubt that this is the case and moreover, it would clearly require an interdisciplinary team effort. Still, laryngeal psychobiological research will gradually inform our understanding and may provide a set of biomarkers that will increase our confidence in the etiology of non-organic voice disorders.

Innate motivational and behavioral tendencies such as approach versus withdrawal or behavioral activation versus behavioral inhibition are thought to shape a person's social and communicative behavior. Trait-specific behavioral dispositions may open the door for uncontrolled (extraversion, overuse, phonotrauma) or constrained (introversion, neck bracing, muscle tension dysphonia) laryngeal behaviors. To complicate matters, most likely not one single personality trait will push a person over the edge toward a pathological state, but instead a combination of traits and the presence of those traits in a pronounced fashion (Eysenck & Eysenck, 1994; Gray, 1970).

For example obvious *neurotic introversion* has been underscored as psychosomatically detrimental for health and well-being in the psychology literature (Eysenck & Eysenck, 1994; Gray, 1970) as well as in the voice disorders literature for the case of MTD (Roy & Bless, 2000a). In other words, the degree and pattern of someone's personality plays a role. Among other factors, negative affect has been highlighted as one general key factor for adverse health outcomes (Cohen, et al., 1995). Still a considerable amount of interindividual variability may exist. For example, the individual's environment has to be accounted for as well. In an interactionist model (Bowers, 1973 in Segerstrom, 2003), if a personality is challenged some individuals may be in a vulnerable place more than others. Further, one's recovery characteristics

from stress exposure will be crucial and therefore, a neurotic introvert with slow recovery may be at a higher risk than a neurotic introvert with faster recovery. Last but not least, the individual threshold specifically for a voice disorder may subjectively and objectively vary. By way of example, the thresholds for the experience of vocal fatigue are known to vary widely and may be partly due to anatomical and biological baseline differences.

Of course, to fully pursue the role of personality as a risk factor for vocal pathologies, i.e. the ultimate question of cause and effect, longitudinal research is called for. For example, ideally the vocally normal participants from the presented study should be the subject of a series of follow-ups over time. However, logistics and resources will likely be problematic, because costly medical examinations by a laryngologist would be a must and college students frequently relocate. Nonetheless, longitudinal research will be quintessential in the future also with regard to the question if the stress-induced laryngeal tension-like behaviors from stress reactive vocally healthy participants in fact match the laryngeal profile of patients with MTD, which is to date unknown or if the results represent a separate acute MTD-like profile.

Apart from personality as individual difference with regard to adverse health outcomes including voice disorders, at a much more fundamental level gender differences should be considered. Gender differences have been shown in neural responses to psychological stress, specifically asymmetrical prefrontal activity in males and primarily limbic activation in females (Wang, et al., 2007). Thus, gender differences may predict vulnerability to psychosocial stress and importantly may provide evidence for a neurobiological basis for differences in health outcomes. A long list of mental and physical disorders, which occur more frequently in women already exists (e.g., anxiety, depression, chronic pain, autoimmune disease) (Holden, 2005; Kajantie & Phillips, 2006; Kudielkda & Kirschbaum, 2005; Lundberg, 2005 in Wang, et al.,

2007) and unfortunately, voice disorders can be added to this list as they are more frequent in women than in men (Thibeault et al., 2004). This discussion inevitably leads to the question of genetic differences and subsequent vulnerability to vocal pathologies. For example, the genetic underpinnings of personality in relation to the larynx will be fascinating. Genetic research in voice disorders has recently begun to receive attention, for example with regard to individual differences at the molecular level in susceptibility to phonotrauma (Lim, Bless, Munoz-Del-Rio, & Welham, 2008).

5.7.4 Generate and evaluate appropriate *education and prevention* programs

The majority of people take their voice for granted and do not think that they have any sort of control over it. As a result, the general awareness for voice disorders is low. At the same time voice disorders also do not receive the media attention that life threatening diseases, rightfully so, get. However, voice disorders can significantly affect one's quality of life and daily functioning (Krischke, et al., 2005; E. Yiu, 2002), and in the case of laryngeal cancer in fact can threaten one's life (Colton, et al., 2006). Consequently, voice disorders are a serious public health concern and public service announcements are important as a means of education.

At a less medical and more stylistic level, a clear and well-tempered voice conveys maturity and professionalism. For example, vocal fry, which is a habitual phonation at a low voice fundamental frequency, has been noted to be a widespread phenomenon among college students (Gottliebson, et al., 2007), and could be perceptually confirmed as part of the presented study. Vocal fry in isolation without other symptoms such as pressed voice does not constitute a

vocal pathology, but at the same time it is not considered a professional speaking pattern. For example, from personal experience some clinic supervisors in departments of Speech Language Pathology require their student speech language pathologists to eliminate vocal fry in the presence of patients in an effort to reinforce role model behavior.

Education and prevention should go hand in hand. Broad education about voice should be included in the curriculum where possible (e.g. guest lectures by speech language pathologists specialized in voice) and be repeated periodically throughout college. The goal of education programs should be to increase one's level of comfort with the own voice and to increase awareness of vocal behavior so to ensure that on the one hand a person will develop a sense of ownership over one's voice and on the other hand will be equipped to recognize a voice disorder early on. In addition, the effects of emotions and perceived stress on voice should be discussed as well.

The tenor of education efforts should be to increase vocal well-being for *everyone*. There is a lack of broad education programs while efforts are generally channeled into prevention programs for professional voice users. Research has impressively amassed evidence that occupational voice users, especially teachers, are at a higher risk for voice problems than other professions (Roy, et al., 2004; E. Smith, Kirchner, Taylor, Hoffman, & Lemke, 1998). Specific prevention programs for occupational voice users have been developed and are under investigation, yet true prevention programs should be implemented earlier on than often the case -- if they are planned at all -- and in a highly programmatic fashion, i.e. with student teachers as opposed to teachers and with a mechanism for refresher courses (Chynoweth, 2001; Roy, et al., 2003; Simberg, 2004). Further, the role of personality and stress reactivity with regard to laryngeal function should be incorporated in future programs.

Returning to the issue of the need for general voice education programs, the screening of vocally normal participants for the presented study and the participants' corresponding results on the *VHI* revealed that lack of power and intensity in one's voice was a common problem, which was further more pronounced in introverts than in extraverts (Dietrich, 2008). Relatedly, in order to ensure effective prevention, a valid point has been made that among occupational voice users individuals who report vocal effort and vocal fatigue should be singled out and targeted for specific prevention (Solomon, 2007). However, such a procedure could also be adopted for the population at large. For instance, the presented research has shown that introverts and neurotics are no strangers to perceptions of vocal effort and those individuals should be targeted. Current prevention efforts should be reevaluated. Many introverts may not end up as professional voice users by way of their personality characteristics and professional preferences and thus would be automatically neglected in prevention efforts, which focus primarily on preventing phonotrauma in professional voice users. Nonetheless, they may be at risk for voice disorders, specifically MTD related voice disorders. Introverts that have been screened for the presented study were characterized by a lack of control and confidence over one's voice, which could result in a higher risk for break-downs of the vocal system in times of challenge, e.g. giving a class presentation.

In summary, voice problems can be caused by many factors of which some are well-known, e.g., vocal demands of the teaching job, while others are less known. Consequently, (1) selected groups of individuals should be exposed to adequate voice education and prevention programs early on, (2) specific efforts should focus on targeting individuals with disposing and precipitating factors such as personality and stress reactivity, which previously have been largely neglected, and (3) voice education should be increased in the general (non-treatment seeking)

population of which a subset may experience mild (subclinical) voice complaints that prevent effective communication and vocal well-being.

5.7.5 Improve *clinical services and outcomes* for patients with existing muscle-tension related voice disorders

The immediate clinical question at hand is how the results of the presented study will inform clinical services for patients with voice disorders, specifically for introverted and extraverted patients with *stress-related* muscle tension voice disorders. Persistent negative emotionality and increased levels of life stress have often been cited as the culprit for poor treatment efficacy in patients with MTD (Roy, 2003). A long-term goal of this line of research will be to learn as much as possible about psychobiological mechanisms behind MTD and phonotrauma in order to treat them in the most effective way with an emphasis on relapse prevention. Intuitively, many speech-language pathologists already take a patient's personality and life situation into account and focus discussions on person-by-situation interactions that may be relevant in a patient's life and may contribute to the voice problems at hand, e.g. the urge to be continuously verbal and exalted at home or with a group of friends (phonotrauma) and the tendency to react with upper body tension to all sorts of life stress (MTD). However, what are lacking are evidence-based personalized voice therapy approaches that would adequately address personality and stress reactivity in voice patients.

Practitioners and patients alike become increasingly aware of complementary and alternative medicine (CAM) approaches to improve health and well-being (D'Antoni, Harvey, & Fried, 1995) and voice science should benefit from the rapid increase in systematic knowledge in

CAM. Findings are promising. It has been possible to *shift* affective dispositions in the desired direction with psychological interventions (Davidson, 2004) and stress reduction programs were shown to lower a person's degree of stress reactivity (Astin, Shapiro, & Schwartz, 2000). However, an intervention from the pool of CAM approaches for patients with voice disorders would have to be chosen systematically with regard to its potential to have positive effects on relevant psychological and biological mechanisms linked to, for example MTD. The presented study represents a first important step towards this goal.

Based on results from the foregoing study one prominent stress reduction approach appears promising for stress-prone patients with voice disorders, which is Mindfulness-Based Stress Reduction (MBSR; Kabat-Zinn, 1990). In the presented study, trait and state stress reactivity appeared to be the common denominator for the occurrence of increased muscle tension and perceptions of vocal effort in both individuals high on extraversion and high on introversion. At this point in time, MBSR is the most widely used structured stress reduction program in the world and the most researched one (Astin, et al., 2000). It is an 8-week program that requires daily practice and a high level of commitment. It is effective in improving health and well-being in anyone, but has been found to be especially effective for reducing physical symptoms such as pain (Kabat-Zinn, 1982) and improving mental well-being such as decreasing anxiety and increasing positive affect as shown by self-report and brain function studies (Davidson, et al., 2003; J. J. Miller, Fletcher, & Kabat-Zinn, 2003). Dispositional positive affect or a "resilient affective style" (Davidson, 2004, p. 1395) have been linked to left prefrontal activation and brain function studies have shown that MBSR can shift someone's tendency from negative affect to positive affect.

MBSR is a meditation approach where mindfulness stands for awareness (J. J. Miller, et al., 2003). The goal is to achieve a state of detachment from the stress reaction in order to explore and understand the mind-body connection. Self-regulatory cognitive processes are fostered in this approach. This approach seems attractive for use with stress reactive individuals prone to laryngeal muscle tension and perceptions of vocal effort, because it focuses on central processes in the mind-body chain such as perceptions and emotional reactivity. Although, muscular tension may be the end result of the stress response, it seems short-sighted to use, for example, biofeedback approaches to reduce muscle tension exclusively if cognitive and emotional processes are at the root of the problem. Although the first-line approach of MBSR is cognitive and awareness-based, meditation has been shown to exert its positive effects through a variety of mechanisms as follows including physiological ones (Astin, et al., 2000): (1) reduction in stress reactivity, (2) reduction in neuroendocrine and autonomic reactivity, (3) improved self-efficacy, and (4) improved relapse prevention. Thus, physiological stressor-induced processes such as seen in the presented study are regulated as well, but are not the sole focus of MBSR. In other words, MBSR addresses both ANS-mediated stress responses as well as voluntary-system-mediated stress responses.

In conclusion, the ultimate goal will be to identify and optimize efficacious psychologically- and biologically-motivated treatment approaches for MTD in order to enhance treatment compliance and long-term treatment efficacy (Dietrich & Verdolini Abbott, 2008). However, realistically one has to question what a certified Speech-Language Pathologist can do within the scope of his or her practice, which does *not* include psychological intervention, or what criteria for a psychological referral would be. The ultimate test would be to refer a patient with primary MTD and high levels of neurotic introversion to a course of MBSR without adjunct

voice therapy. Based on the foregoing results it is conceivable that a patient's voice would improve, because of self-regulatory processes that would *prevent* frequent or full-blown stress reactions. However, it is also conceivable that long-term treatment efficacy may only become optimal when stress reduction interventions are combined with voice therapy. In that case the question would be if individuals will need to develop a certain amount of understanding of the phonatory system to achieve a sense of self-efficacy to manage favorable or unfavorable stress-related laryngeal behaviors.

Clinically, it would be informative to perform some basic psychosocial testing with patients with MTD such as personality testing (under supervision of a psychologist) and chronic stress ratings (e.g. Perceived Stress Scale, Cohen & Williamson, 1988). A patient's grade of neuroticism will also be an important piece of information for the clinician, because neuroticism can increase symptom reporting, which may affect self-report measures such as the *VHI* used regularly in voice centers. Consequently, a patient's self-report measure should be viewed in the context of his or her personality and the clinician should watch out for response patterns. Further, based on the results from psychosocial testing, patients should be *educated* about their risks for, e.g. either MTD or phonotrauma and what the mechanisms for their vocal pathologies may be. At minimum SLPs should provide some basic education on the links between perceived stress and vocal function as well as perceived stress and therapy compliance. In fact, readiness for change according to the transtheoretical model of change (Prochaska, 2008), and by extension readiness for voice therapy, has recently received systematic attention in the voice disorders domain (van Leer, Hapner, & Connor, 2008) and it may be even recommended that a patient postpone voice therapy if chronic stress is overpowering. In such cases it may be advisable to recommend that a patient go through professional stress reduction first. Finally, the suggested

course of actions would be in accordance with predictive and personalized medicine as suggested by the National Institutes of Health (NIH).

5.7.6 Structural equation modeling

It has been acknowledged that finding adequate statistical analyses for psychobiological research can be challenging (Jennings & Gianaros, 2007). In particular, a challenge becomes apparent when a conceptual causal model is under investigation consisting of many parallel effects, interactions, and potential mediators. The psychobiological framework for studying stress and its relation to voice disorders, as discussed in Chapter 2, qualifies as such a model (Dietrich & Verdolini Abbott, 2008). Therefore, the *long-range goal* will be to test the model with a statistical procedure called Structural Equation Modeling (SEM) that has promise to investigate the accuracy of a proposed complex model in one equation. In the presented study, the application of SEM will not be possible due to a limitation in statistical power given the available data set of n = 54 –which was powered for the primary research question rather than for the proposed model as a whole. Still, a preliminary SEM will be possible in the future by combining data from the pilot study, main study, and by including participants who fell in the buffer zone designated to select participants based on personality. Moreover, it would seem there could be great value to introducing SEM to the field of Communication Science and Disorders even at a preliminary level, because it can provide a powerful tool towards understanding the complexity of behavioral science.

Although detailed mathematical background around SEM is beyond the scope of the presented document, the general idea is as follows[5]. SEM is a multivariate statistical method that is rapidly growing in popularity in the behavioral sciences, because it allows for the testing of complex theoretical models (Kim & Bentler, 2006). Reality, and by extension any psychobiological model that attempts to reflect it, is by nature complex and SEM provides the opportunity to examine the model as a whole instead of in a piecemeal fashion as in traditional ANOVA. In SEM, both direct (A → B) *and* indirect effects (A → ***C*** → B) can be investigated among a set of variables in a regression-like fashion. In particular, indirect effects are of interest in the context of this project, because they refer to potential mechanisms that may drive changes in the primary dependent variable of interest, laryngeal activation. For example, the indirect roles of both negative emotions and SBP for increasing SEMG activity under exposure to a stressor could be investigated, even in parallel. Unique to SEM is the fact that a variable can be both a dependent and independent variable at the same time. For instance, negative emotions could be both a dependent variable triggered by a stressor and in turn be a predictor variable for SEMG changes. The main research question in SEM would be to test the quality of the proposed psychobiological model for voice disorders in explaining the observed data. In other words, can the model reproduce and explain the relationships among the variables, to a satisfactory degree? Of note, strictly speaking SEM cann*ot* confirm or reject causality. Nonethelesss, SEM will ultimately be able to test the data for consistency with the model.

[5] SEM will be briefly described paraphrasing (with permission) an introductory chapter on this method written by the statistical consultant for this project, Kevin H. Kim, Department of Psychology in Education (Kim & Bentler, 2006).

5.7.7 Summary of future directions

The current trend in voice research can be summarized by the motto “from clinical symptoms to biological realities” (Thomas, 2007). Further, this time has been called the decade of “mechanism research,” where studies should carefully investigate any mechanisms by which, for example exercise results in functional change (Thomas, 2007). Without doubt, basic and clinical sciences need to be more intertwined than ever before in the form of translational science. A main goal of future research will be to establish a thorough line of *experimental* psychobiological research applied to clinical voice science, which has been a neglected area. Psychobiological methodologies bear great potential for the field of voice disorders, especially with regard to those voice disorders that have been traditionally viewed as ”psychological” such as MTD. A psychobiological approach could finally give MTD the credibility that it is currently missing. Such a statement implies that the presented results from vocally healthy participants cannot be readily generalized to patients with MTD and that the study will have to be replicated with patients. Voice researchers should not be discouraged by the sheer complexity of psychobiological research but instead use lessons learned in the psychophysiology literature. One major strength lies in the fact that psychobiological approaches allow for the simultaneous observation of perceptual and objective processes. In fact, the investigation of interactions of various levels of psychological and neurophysiological functioning is explicitly encouraged (Cacioppo, et al., 1998; Linden, et al., 2003) and will improve the power to describe the neurobiological basis of psychological stress (Wang, et al., 2007).

APPENDIX A

GENERAL PARTICIPANT CRITERIA

General Participant Criteria	Rationale
Inclusion criteria	
Females	*Voice:* Most literature indicates that more women than men are likely to experience voice problems, or at least to report them (Miller & Verdolini, 1995; Roy et al., 2004). *Objective measures:* A focus on one gender will reduce variability of SEMG measurements due to gender differences in laryngeal structures (Colton et al., 2006) and variability of cardiovascular measurements (Shapiro et al., 1996).
Ages 18-35 years	*General:* A coherent group, such as college students, is preferred in order to reduce extraneous variability in the data. *Larynx:* Age-related changes affect cartilages, connective tissue, blood supply, glandular secretions, and muscle (Colton et al., 2006). Changes to the larynx emerge after the 5th decade in females (Kahane, 1987). *Blood pressure:* Blood pressure increases with age due to change in kidney function (50s) and due to arteriosclerosis (60s) (Guyton & Hall, 2005).
Native speaker of English → self-report	Reduced English proficiency may be an additional stress factor during reading and public speaking.
Normal speech and language skills → as determined independently by the PI and a second certified speech-language pathologist based on conversational speech and reading (*CAPE-V*, Rainbow Passage) as well as a review of the participants' speech-language history	Deviations from normal speech and language skills may be an additional stress factor during reading and public speaking and may affect voice characteristics. Articulation within normal limits is required for the collection of adequate samples for the speech recognition part of the study.
Normal speaking voice → as determined independently by the PI and a second	Vocal fold pathology or any other voice disorders affect vocal functioning and auditory-perceptual changes in voice quality are frequently the result (Colton et al., 2006). The

certified speech-language pathologist based on auditory-perceptual ratings of the participants' conversational speech, reading, and voice production (*CAPE-V*, Rainbow Passage)	auditory-perceptual screening will help to exclude participants who have abnormal voice characteristics that are not self-perceived. However, the (common) presence of vocal fry is not considered an exclusion criterion as long as all other voice-related criteria are met (Gottliebson et al., 2007).
Exclusion criteria	
Current smoker	Smoking is known to irritate mucosa in the larynx and may affect laryngeal functioning and lead to laryngeal disease (Colton et al., 1996).
Acute or chronic upper respiratory infection at time of testing or cardiac, pulmonary, or neurological problems, specifically history or symptoms of systemic diseases known to affect the nervous or endocrine systems → by review of self-reported medical history and medications	Respiratory, cardiovascular, or neurologic disease may affect multiple speech subsystems (Darley, Aronson, & Brown, 1975; Lee, Loudon, Jacobson, & Stuebing, 1993) as well as normal cardiovascular and autonomic nervous system functioning (Freeman, 2006; Guyton & Hall, 2000). Upper respiratory infection may affect the respiratory and laryngeal subsystems (Boone & McFarlane, 2000; Verdolini et al., 2005).
Medications known to affect the nervous and endocrine systems in the week before the experimental session (contraceptives not included) → by self-report	Please see previous rationale.
Allergies, laryngopharyngeal reflux disease (LPR), and asthma, which both the participant and the P.I. will concur do affect the voice at the time of study participation → by review of self-reported medical history and medications	If uncontrolled, the listed conditions may affect laryngeal functioning in particular by irritating the laryngeal mucosa (Colton et al., 2006; Verdolini et al., 2005).
Controlled and uncontrolled hypertension (>140 mmHg systolic and/or 90 mmHg diastolic blood pressure; American Heart Association) → per blood pressure screening (in case of doubt 5 min. rest period and average of at least 3 measurements) (Shapiro et al., 1996)	Participants with controlled and uncontrolled hypertension will be excluded in order to reduce the variability of blood pressure responses among participants during the protocol (Shapiro et al., 1996) and to protect participants from extreme BP responses during the protocol.
Participants currently under the care of a psychiatrist, on medications for treatment of a psychiatric disorder, or with current moderate depression → by self-report, review of medical history and medications, and depression screening (*BDI*; score ≥16)	Acute psychiatric disease may affect participants' emotional and cognitive responses and general symptom reporting during the stress reactivity protocol. Antidepressive or anxiolytic medications may affect blood pressure.
Inadequate hearing → as per a pure-tone hearing screening at the frequencies of 1000, 2000, 3000, and 4000 Hz at 25dB HL (American Speech-Language-Hearing	Hearing loss may indirectly affect speech and vocal functioning, in particular loudness of voice (Colton et al., 2006).

Association)	
Body mass index (BMI) in the obese range → by self-report of height and weight and calculation of BMI and comparison with tables provided by the NIH Department of Health and Human Services (=/> 30); HTTP://WWW.NHLBISUPPORT.COM/BMI/BMICALC.HTM	Participants in the obese range will be excluded to reduce blood pressure variability in the study sample (Shapiro et al., 1996). Further, fatty tissue in the neck may preclude optimal SEMG recordings and introduce variability (Fridlund & Cacioppo, 1986) and simply complicate the optimal identification of laryngeal landmarks for the positioning of electrodes on the neck.
History of laryngeal trauma or surgery → by self report and review of medical history as well as visual inspection	Laryngeal trauma or surgery may affect laryngeal functioning. Neck scarring (e.g., post thyroid surgery) will prevent optimal SEMG recordings.
A current or lifetime history of a voice problem or voice disorder lasting 2 weeks or longer → self-report	The goal is to include participants who are not defining voice as a problem and to exclude participants who have a history of voice disorder. Voice disorder in this particular context will be defined as "condition of sufficient concern for the bearer to report it, register functional disruption because of it, and/or seek treatment because of it." (Verdolini & Ramig, 2001, p. 26).
General complaint of vocal effort and vocal fatigue while speaking → Self-report, Direct Magnitude Estimation (DME > 100), and *Vocal Handicap Index* (positive answers to questions #14 and #20 related to strain and effort during voice production)	Vocal effort and vocal fatigue are frequently associated with voice disorders, especially with vocal fold lesions or muscle tension dysphonia (Wright & Colton, 1972; Verdolini et al., 2005).
Previous voice therapy or professional singing, voice, or speech training → self-report	Past voice therapy is an indication of a voice disorder. Singing, voice, or speech training often includes laryngeal components and resonant voice training which may positively alter laryngeal functioning (Colton et al., 2006).

APPENDIX B

SPEECH BASELINE

B.1 *CONSENSUS AUDITORY PERCEPTUAL EVALUATION OF VOICE* (*CAPE-V*)

1. Please sustain the vowels /a/ and /i/ for **3-5** seconds each.
   /a/*
   /i/

2. Please read the following sentences:
   a. The blue spot is on the key again.
   b. How hard did he hit him?
   c. We were away a year ago.*
   d. We eat eggs every Easter.
   e. My mama makes lemon muffins.
   f. Peter will keep at the peak.

*Was elicited as reference sample before the onset of public speaking.

B.2 THE RAINBOW PASSAGE

When the sunlight strikes raindrops in the air, they act as a prism and form a rainbow. The rainbow is a division of white light into many beautiful colors. These take the shape of a long round arch, with its path high above, and its two ends apparently beyond the horizon. There is, according to legend, a boiling pot of gold at one end. People look, but no one ever finds it. When a man looks for something beyond his reach, his friends say he is looking for the pot of gold at the end of the rainbow.

Throughout the centuries people have explained the rainbow in various ways. Some have accepted it as a miracle without physical explanation. To the Hebrews it was a token that there would be no more universal floods. The Greeks used to imagine that it was a sign from the gods to foretell war or heavy rain. The norsemen considered the rainbow as a bridge over which the gods passed from earth to their home in the sky. Others have tried to explain the phenomenon physically. Aristotle thought that the rainbow was caused by reflection of the sun's rays by the rain.

Since then physicists have found that it is not reflection, but refraction by the raindrops which causes the rainbows. Many complicated ideas about the rainbow have been formed. The difference in the rainbow depends considerably upon the size of the drops, and the width of the colored band increases as the size of the drops increases. The actual primary rainbow observed is said to be the effect of super-imposition of a number of bows. If the red of the second bow falls upon the green of the first, the result is to give a bow with an abnormally wide yellow band, since red and green light, when mixed, form yellow. This is a very common type of bow, one showing mainly red and yellow, with little or no green or blue.

B.3 VOCAL EFFORT RATING

We would like you to rate your vocal effort now:

Let's say that 100 is a comfortable amount of effort in your voice. 200 would be twice as much effort as comfortable. 500 would be five times as much effort as comfortable. There is no upper end to the scale.

What number would you use to describe your vocal effort during the previous reading/speech?

APPENDIX C

EXPERIMENTAL STRESSORS

C.1 PUBLIC SPEAKING STRESSOR

Take stopwatch. Position video camera in front of participant. Turn on video camera. Position participant.

The task we are going to have you do now is a speech task.
Imagine you have applied for a job as a lawyer and you were invited to present yourself before a committee which will evaluate on the basis of your personal characteristics. Your task now is to convince the committee in a free speech that you are the best candidate for the vacant position. Following these instructions, you have two minutes to prepare for the speech. Please also note that you will be recorded by a video camera as well. We will record your speech for a subsequent voice frequency analysis to reveal any paraverbal signs of stress. The camera recording is used for later behavioral analysis. The members of the committee are trained in behavioral analysis and will take notes during your speech. Your speech is supposed to take five minutes. Do you have any questions?

[If question is asked regarding the type of lawyer, then respond: "That's up to you".]

Check posture.

Start recording. Say date, participant's #.
Please start your preparation period and please keep as still as possible.

Leave room.
After 2 minutes:

Please try to keep your head, arms and legs as still as possible.

First, please sustain /a/ for 3-5 seconds right now. [time duration]
Now, say the sentence: "We were away a year ago." [model the sentence with natural intonation and on one breath]

Please begin your speech and speak for the entire period of five minutes and maintain eye contact[6]. Start stopwatch.

[Instructions for experimenter who introduces the stress task: fake data protocol sheet on clipboard; take random notes; do not smile, do not laugh; maintain eye-contact.]

Only after a pause of more than 10 seconds are questions asked, e.g.,
You still have time, please continue…

[Keep follow-up questions to minimum]

Should it appear after another 10 seconds that the subject has nothing further to say, then the experimenter should ask questions until the end of the time period; the phrasing of these questions is left to the experimenter's discretion, e.g.

- *Why do you think that **you** are the best applicant for this position?*
- *What qualifies you in particular for this position?*
- *What other experiences have you had in this area?*
- *What about your studies identifies a special aptitude and motivation for this position?*
- *Where else did you apply? Why?*
- *What would you do, if your application here would not succeed?*

You can stop speaking now.
Please rate your vocal effort on this sheet.
Now we will have you rest quietly again for 7 minutes. Please try to keep your head, arms and legs as still as possible.

Take video camera and leave room.

[6] The instructions to speak for the entire period and to maintain eye contact have been added for the main study, because review of videos from the pilot study showed that some participants quickly ran out of things to say. In addition, follow-up questions were counterproductive, because they elicited only brief answers. Lastly, some participants avoided eye contact.

APPENDIX D

EXPERIMENTAL PROCEDURES

D.1 LAB PROTOCOL

Task	Verbal instructions	Duration/Measures	
Pre-arrival	Preparations Microphone loudness calibration		
Arrival	1. Complete intake form 2. Questionnaires 3. Offer bathroom	10 min.	
Instrumentation set up	SEMG (including photos for documentation) Microphone (headset mouth-to-mic distance 3 cm, 45^0 off-axis positioning) Blood pressure cuff, non-dominant arm	25 min.	
BP - Dinamap	*This cuff is automatic; you will know when it is recording your blood pressure because you will feel it inflate on your arm. This will happen at various times during the session. When you feel the cuff inflate, try not to move your arm. The cuff will feel tight, especially the first time it inflates.*		1x BP to be discarded R L arm Small cuff Large cuff
SEMG placement verification	Check for movement artifact Swallow Sustained high pitch: /i/ Sustained low pitch: /u/ Leg tension Keep light turned off during recordings.		
SEMG MVC	Visual feedback Motivation	12 min.	MAX

	Instruct for both tasks at beginning (1) mandible depression task against resistance; 100% and 50% (2) dorsiflexion foot against resistance; 100% and 50% x3 each for 5 seconds; 1-min. rest intervals		
Rest	*You will now rest for 2 minutes. After that we will continue with readings.*	2 min.	SEMG
Baseline speech recording	*We will now start with speech recordings. We will record your muscle movements, speech, and blood pressure. Please use your comfortable pitch and loudness. Please keep looking straight ahead. .* CAPE-V (monitor length of vowels, all-voiced sentence); turn on monitor, give signals for each reading; Rainbow passage- separate recording	3 min.	START ALL: SEMG + CLOCK +BP 0:00 0:00 1:30 Vocal effort rating
CS 1 reading	Change electrodes before and after Turn monitor on/off	10 min.	No BP Vocal effort
Baseline rest	Take clock. *We will now continue with a 10-minute, undisturbed, resting period. We will be taking measures. When you feel the cuff inflate, try not to move your arm. We will be also recording your muscle activity. Please keep looking straight ahead and do not cross your legs. Try to move as little as possible. Also, please do not sleep.* Start clock.	10 min.	Start at 3:30 … 3:30 5:00 6:30 8:00 9:30 10:00 STOP
PANAS-X	*Okay, now I would like you to take a few moments and indicate on this questionnaire how you were feeling over the past 10 minutes.*		

Public speaking	Notes:	2 min. anticipation /a/ + all-voiced sentence + 5 min. speech	START ALL: SEMG +CLOCK+BP 0:00 1:30 2:00 Stop RESET CLOCK START SEMG + CLOCK + BP 0:00 1:30 3:00 4:30 5:00 STOP
Vocal effort			Vocal effort
Recovery	*Now we will have you rest quietly again for 7 minutes. Please try to keep your head, arms and legs as still as possible.*	7 min.	START ALL: SEMG +CLOCK + BP 0:00 1:30 3:00 4:30 6:30 7:00 STOP
PANAS-X Rumination	*I would like you to complete this questionnaire one final time, focusing on how you were feeling (1) during the interview speech and (2) during the recovery period. I also have two other short questionnaires for you to fill out at this time. Please read the instructions carefully before filling them out.*		PANAS-X Speech and Recovery PRCS Rumination
Recovery speech	*Now, we will record your speech one final time.* CAPE-V Rainbow passage	5 min.	START ALL: SEMG +CLOCK + BP 0:00 0:00 1:30 Vocal effort rating
CS 2 mouthing	Change electrodes Turn on monitor Remove microphone Video camera focus on face	10 min.	

Remove instruments			
Debriefing			

D.2 DEBRIEFING

Thank you very much for your participation in our research study. Please note that we have disguised the full purpose of the study, in which you have participated. Not only were we interested in data that are helpful to build automatic speech recognition systems based on muscular responses in the face and neck, but we were also interested in running a psychophysiological study. In this study we examined whether and how psychological stress influences the functioning of muscles in the neck and subsequently voice production. A wealth of health psychology studies show that stress is associated with increased cardiovascular, autonomic nervous system, hormonal, immunological, and muscular responses. However, it remains unclear how stress exactly influences the functioning of the voice box (larynx) even to the extent of contributing to the development or maintenance of voice disorders. In particular, a voice disorder called Muscle Tension Dysphonia has been described in the literature as being related to persistent increased or dysregulated muscle tension in the voice box (larynx) due to unknown causes. Stress has been cited as one possible factor in this voice disorder. This study was designed to explore pathways and mechanisms that may account for the relationship between stress and voice production and stress and voice disorders including the possibility that personality influences the speech production pattern of responding to stress. For this purpose, we have collected data from individuals with and without voice disorders. In the future, the gathered knowledge will hopefully improve treatment programs for individuals with Muscle Tension Dysphonia or other voice disorders.

Again, thank you for your participation. If you have any further questions at this point or in the future, please do not hesitate to contact the Principal Investigator of this study, Maria Dietrich (phone 412-383-6709) or Dr. Katherine Verdolini (phone 412-383-6544). Further, you may contact the Human Participants Protection Advocate of the IRB Office, University of Pittsburgh (1-866-212-2668) or the Carnegie Mellon University Compliance Officer; Regulatory Compliance Administration; UTDC Building, Room 312; (412) 268-4727 to discuss problems, concerns, and questions.

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