Vocal Indices of Stress: A Review

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Summary: Objective. Identification of stress patterns in the voice has multiple potential applications. The objective was to review literature pertaining to the effects of various forms of stress upon the healthy voice.

Study Design. Literature review, discussion of results, and direction for further study.

Methods. This review article offers a model of stress and a review of the historical and recent research into the effects of stress on the voice. Electronic databases were searched using the key words. No studies were excluded on the basis of design; however, an attempt was made to include in the discussion studies which primarily address physiological and acoustic vocal parameters. The results of greater than 50 studies examining the effect of stressors ranging from lie and guilt to high altitude and space flight upon the voice were included in the review.

Results. Increase in fundamental frequency is the most commonly reported effect of stress in well-controlled trials. The trend, however, is not universal. A reduction in noise as reflected by the diminished vocal jitter is reported, but less frequently.

Conclusions. Stress types, gender, and individual differences in baseline autonomic tone may explain the primarily equivocal findings of effects of stressor exposure or perceived stress on voice; and as such, the article concludes with a discussion of directions for future study.

Key Words: Voice–Stress–Emotion–Gender–Sympathetic nervous system–Deception–Guilt–Detection–Tremor–Performance–Beta-adrenergic blockade–Neurohumoral–Cold pressor–Fundamental frequency–Jitter–Shimmer–Propranolol–Menstrual cycle–Pilot–Altitude.

INTRODUCTION

Our voices reveal a plethora of information. The average speaker's voice may reveal his or her age within a 10-year-span,[1–4] state of general health,[5,6] body size,[7,8] race,[9,10] and gender.[4,10] Does our voice also betray our emotional state? Does it reveal our stress levels? There have been numerous attempts undertaken by investigators representing a wide spectrum of disciplines to answer both questions for a variety of reasons. For example, interest exists in identifying stress in the voices of pilots and astronauts in attempt to avert in-flight catastrophe and aid in the development of voice recognition systems that function despite stress-related alterations in the acoustic signal.[11] In light of recent events, there has been an interest in identification of stress in the voices of potential terrorists before they strike and in the voices of criminals for the purpose of lie and guilt detection. In addition, many professional singers and speakers report vocally detrimental effects of stress-induced performance anxiety and seek treatment to prevent the phenomenon.[12–14] Despite the interest in identifying stress and negative emotions in the voice, there remains a paucity of objective data and few consistent findings as to the vocal effects of stress.

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Defining stress

A somewhat amorphous term, "stress" is used in many disciplines from engineering and physics to biology. When used in biology, it generally refers to an aversive phenomenon that can lead to adverse physiological, emotional, cognitive, or behavioral consequences. Organisms strive to maintain an internal state of balance or baseline (homeostasis) and stress tends to disrupt this balance. A stressor, be it physical or emotional, can disrupt baseline leading to physiological adaptations designed to deal with the situation. When an environmental demand (ie, stressor) surpasses the ability of the organism to maintain homeostasis, a physiological cascade of events is initiated to attempt to restore homeostasis. Furthermore, what constitutes a stressor (environment or event that may induce a stress response) may differ from organism to organism or person to person. That is, a stress response is initiated when personal resources, differing substantially by individual, are deficient for managing a given situation. In the short term, stress is adaptive and self-corrective in that the organism's physiological state autocorrects after removal of the stressor, returning the organism to baseline. Human beings, however, tend to have an array of stressors which are chronic and which do not allow for self-correction. When an organism is subjected to a sustained adaptive physiological response, a number of long-term system-wide accommodations may develop. Those accommodations may include among others, suppression of the immune system, dysregulation of inflammation, disturbances in lipid and glucose metabolism, and increases in other cardiovascular risk factors.[15]

Traditional views of stress in the social and life sciences focus on the concept of homeostasis, the aforementioned state of internal balance, developed by Cannon[16] and subsequently elaborated upon by Selye.[17] These models refer to homeostasis

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as a suite of physiological mechanisms designed to return bodily systems to baseline or initial conditions, when they are disrupted.[16] The two major interconnected systems responsible for the stress response are the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS). The ANS is further subdivided into parasympathetic and sympathetic branches.

The sympathetic branch or sympathetic nervous system (SNS) is typically associated with the fight-or-flight reaction to both physical and socioemotional stressors with typical indices including increased heart rate, skin conductance, and blood pressure.[18] Sympathetic arousal results in both peripheral (ie, redirection of energy from the viscera to the periphery) and central (ie, appropriate aggression and arousal) responses.[19]

The HPA axis is preferentially activated by social stressors, particularly those that are unpredictable and is associated with adrenocortical release of the neurohormone cortisol.[20,21] Cortisol is implicated in enhanced mental activity and improved cognitive problem solving necessary for responding to unpredictable challenges.[22–24] Chronic cortisol secretion, however, resulting from a chronic stressor is associated with neural atrophy[15] and is thought to be responsible for the link between chronic stress and emotional dysregulation such as mood and anxiety disorders.[25,26] Though these two systems (HPA axis and ANS) are anatomically and physiologically distinct, they are closely intertwined with extensive circuit integration and cross-regulation[19] and may be considered components of a single stress response system as they work in conjunction to influence the body's response to stressors.[22] Resulting changes from the interactions of these two systems can take the form of significant downstream effects in the physiological systems affected by the stressor, including vocal changes.

As presented above, stress occurs in the short term, seconds to minutes to hours, and in the long term, over weeks, months, and years. Addressing the effects of stress on the voice on either temporal domain is a rather daunting task. Given this, the initial focus should be on the short-term effects of stress on the voice to address baseline responses. Thus, the major focus of this work is on acute rather than chronic responses to stress, as those are most relevant in the search for vocal markers of stress.

Hypothesized stress-related voice alterations

Although vocal effects secondary to HPA axis activation may be difficult to predict, changes in voice secondary to sympathetic agonization might be hypothesized. For example, it might be expected that increases in fundamental frequency (F_0), subglottal pressure, jitter, shimmer, maximum airflow declination rate, voice onset time (VOT), vocal intensity, and speaking rate would result from sympathetically modulated increases in heart rate and bronchodilation. Orlikoff and Baken[27] found that heart rate contributed 0.5–20% to the total vocal F_0 perturbation measure or jitter, whereas Orlikoff[28] demonstrated that the heart rate contributed 11.8% to the total vocal intensity perturbation measure or shimmer. This modulation could result in part from heart rate influence on the subglottal pressure,[28] which is theorized to influence the F_0 in a linear fashion. F_0 of the voice is reported to increase by 3–6 Hz per unit measure (cmH_2O) increase in subglottal pressure.[29,30] It would be expected that increased lung pressure would also contribute to increased vocal intensity and F_0.[31] Bronchodilation secondary to sympathetic arousal would be expected to result in voice production at higher lung volumes and ergo increased VOT.[32] Therefore, VOT might be hypothesized to increase during sympathetically stimulated bronchodilation.

METHODS

Regular literature review has been conducted since 2003 using the following databases: MEDLINE, PsychINFO, and Science Direct. Review was conducted using the following search terms: voice, stress, emotion, anxiety, workload, gender, SNS, lie, deception, guilt, detection, tremor, performance, beta-adrenergic blockade, neurohumoral, cold pressor, F_0, jitter, shimmer, propranolol, menstrual cycle, pilot, and altitude.

Inclusionary criteria

Citations were not excluded due to publication year, but attempt was made to include only citations reporting acoustic or aerodynamic voice data and/or other physiological data. Attempt was made to sample a large cross-section of stressor subtypes.

Exclusionary criteria

Studies employing perceptual voice analysis, only, were not the focus of this review.

Greater than 50 experimental citations were selected for inclusion in this review. The results should be interpreted with caution since there was no correction for inflated alpha. An attempt was made to sample a large cross-section of stressor subtypes, both physical and psychoemotional. The SNS fight or flight response can be induced through innumerous physical mechanisms. For example, investigators have attempted to capture the voice under various conditions, including exercise[33] and extreme altitude.[34] In addition, stress responses under the jurisdiction of both the SNS and the HPA axis have been attempted through electric shock and shock threat,[35–37] mental loading or workload,[38,39] mock attempts at deception and guilt,[35,40] vocal portrayal of various emotions[41,42] and neurohumorally through SNS and HPA cold pressor activation and pharmacologic modulation of the effects through the use of beta-receptor blockade.[43]

RESULTS

Study descriptions

The methods of voice analysis are as varied as are the methods of stress induction that have been used in attempt to identify stress in the human voice. Most of the methods used to identify guilt or falsehood could be described as nebulous, perhaps even surreptitious. Almost without exception, study replication has been rendered impossible. Other issues include the difficulty consistently and validly capturing the effects of psychoemotional distress in the laboratory.[44] Even naturalistic setting behavior can be altered by the presence of voice recording

instrumentation. The literature contains few naturally collected examples of voice produced under emotional duress.[45,46] Attempts to produce voice while portraying emotions are purely that—portrayals. As such, the validity of outcomes is, at best, questionable[47] because overemphasis of emotion might be expected.[44]

Although difficult to design, researchers from a broad array of disciplines including but not limited to vocology, otolaryngology, neurology, aviation and space medicine, voice science/speech pathology, psychology, neuroscience, gravitational physiology, speech communication, and clinical autonomics have attempted to validly measure the effects of various stressors upon the voice. The following is a brief review of some of the findings followed by a discussion of the outcomes and suggestions for further study.

Tremor modulation and lie detection

Gunn and Gudjonsson[46] examined recorded conversations between a terrorist and police before and after a hostage killing. The researchers analyzed the recordings using the psychological stress evaluator (PSE) that has been marketed by Dektor since 1976 for the purpose of lie detection. It was claimed that a tremor-like modulation of the vocal amplitude following the killing as contrasted with a lack of this modulation before the killing was identified by the PSE. The authors explained that the lack of tremor immediately before the killings indicated a high-stress situation as indicated by the voice in contrast to the presence of tremor after the killings, which indicated alleviation of stress. The ability to identify stress in the voice is the premise underlying the marketing of the so-called voice "lie detector tests" such as the PSE. These devices have been marketed under such names as: The Truster (Makh-Shevet, Israel), Ex-Sense Pro (Nemesysco's Voice Analysis Technologies, Israel), Computer Voice Stress Analyzer (CVSA) (National Institute for Truth Verification, West Palm Beach, FL), Liarcard (Tel-Tech Systems, Toms River, NJ); X13-VSA (X-13 VSA Ltd.), DeFIBulator (Spion, Romania), TrusterPro (Nemesysco Ltd., Israel), and the Vericator (Trustech Ltd., Israel).

The manufacturers promote the ability of their device to detect the presence of an 8–14 Hz microtremor under the jurisdiction of the central nervous system in the unstressed voice, which is absent from the vocal signal during times of stress.[46,48,49] The acoustic microtremors are purportedly caused by the action of laryngeal muscle tremors upon the valved airstream. Furedy[50] described the term, "deception," as the human equivalent of the hiding response and as such a variant of the flight or fight response. Deception is hypothesized to induce stress when individuals behave in a manner (ie, lie) that violates their convictions.[51] Sociopaths, devoid of convictions, would not be expected to demonstrate a change in the otherwise predictable patterns. The existence of a microtremor in the voice during times of stress was presumed to be based on the observation of physiological tremor (8–14 Hz) in the long muscles of the extremities that is well documented to increase in amplitude during times of stress.[52,53] The apparent assumption underlying the use of the PSE is that a similar physiological tremor in the range of 4–6 Hz is manifested in laryngeal muscle contractions.[54,55] The premise that the tremor is absent during stress, however, appears to contradict the well-documented, enhanced physiological tremor during stress that is relieved by beta-adrenergic blocking agents.[52,53,56]

Identification of deception-associated vocal microtremor alteration was purported to have been used for insurance fraud identification, new employee screening, police interrogations, and intelligence interrogations at the Pentagon, Guantanamo Bay, and in Iraq when questioning potential criminals and terrorists. Covert use through telephone connection was common.[57] Despite the reports of several investigators highlighting a lack of construct validity and inability to identify stress and/or truth from lie using the systems,[58–60] the devices remained in use by the US Department of Defense, employers, and police officials for several decades and until the bulk of objective study outcomes revealed that such methods could not identify truth from falsehood or stress, validly.[40,58–61]

Shipp and Izdebski[60] looked for evidence of the purported laryngeal muscle tremor. Hook-wire electrodes were inserted into the laryngeal muscles and no periodic component of muscle contraction below 20 Hz was identified. Hollien and Harnsberger,[62] as well, examined the spectrographs of subjects receiving electric shock and those of controls. No energy bands beneath the F_0 of the voice were identified. The ability of a vocal lie detector to identify stress in the voice was tested again by Hollien et al.[58] Two groups of normal male and female subjects were evaluated under the separate conditions of electric shock and first-time public speaking. Voices were recorded on audiotape and evaluated by the PSE. Correct identification of stressed versus nonstressed conditions by individuals fully trained in the use of the PSE system was no greater than 52% or chance value.[58] Hollien et al criticized the systems for lacking both a methodological theoretical basis and cited research support. Several supporters of the systems, as well, have criticized the manufacturers for the lack of objective and consistent standards for the use of the methodology.[37,57,63]

Manufacturers of the TrusterPro claim that their device can distinguish lie from truth, and more specifically, cognitive stress from the emotional stress associated with guilt and/or deceit using spectral analysis and identification of microtremor. Gamer et al[47] examined the ability of the TrusterPro to identify guilty knowledge by offering financial reward to participants who could successfully fool the TrusterPro during a mock crime using the Guilty Knowledge Test (GKT). The TrusterPro examiner was blind to the guilt or innocence of the study participants. Advantages to the study design were the inclusion of galvanic skin response (GSR) and cardiovascular and respiratory responses. Findings included a significant reduction in respiratory flow and a significant increase in GSR in the guilty condition. Blood pressure was not significantly altered; however, phasic heart rate significantly increased and then diminished in the guilty condition. In contrast, the TrusterPro could not correctly identify innocence from guilty knowledge. The authors concluded that although the GKT appeared to adequately induce stress as demonstrated by significant GSR, cardiovascular, and respiratory changes, the TrusterPro was unable to identify stress in the participants' voices.

Harnsberger et al[64] tested the ability of the layered voice analysis (LVA) system to identify truth from deception in a double-blind experiment. Seventy-eight participants participated. The participants were instructed to make an argument on such topics as gun control, religion, and sexual orientation with some of the statements consistent (ie, truth) and some statements inconsistent (ie, lie) with the participants' beliefs. The investigators designed the task with six varying degrees of stress. For example, the "high stress lie" included a "dual stressor" whereby participants were warned that they would receive electric shock if they failed to display adequate signs of lying. Likewise, the "high stress truth" task included electric shock as participants read statements they believed to be true. Advantages of the study included the addition of galvanic skin response (GSR); pulse rate, and an anxiety/stress scale. In addition, the researchers included in the final analysis the data from only those participants whose stress condition GSR, pulse rate, and anxiety measures were more than double that of the baseline levels. An additional advantage of the study was the inclusion of two sets of examiners for the LVA analysis: two LVA company examiners and two of the investigators/authors. The investigator examiners were informed that the LVA performs an "automatic" analysis; however, the manufacturer's examiners were observed to use various protocols inconsistently throughout the study. Results of the analyses of both teams were below or no better than chance levels. The authors concluded that although they had included only those participants who demonstrated stress as indicated by both autonomic and emotional indices, the LVA system was unable to identify stress with any degree of accuracy.

The use of voice stress analysis continues. Most recently, there was news of attempts to identify stress in the voice of a Florida man accused of a vigilante killing for which he countered that the shooting was in self-defense. Although it was determined that the evidence was inadmissible in court, it remains that an attempt was made to include in the legal proceedings the outcome of a CVSA analysis of the defendant's voice post-shooting.[65,66] Summation of the previous section, however, would appear to indicate that tremor modulation of the voice is not a reliable measure of lie, guilt, or stress. This does not preclude that the concept may be a valid one but rather that significantly more information is needed before validity of the methodology may be assumed.

Performance anxiety, voice, and beta-adrenergic blockade

The emotional and physiological responses in stage fright or performance anxiety are manifestations of the SNS fright, flight, or fight response. Tachycardia, elevated blood pressure, diaphoresis, altered salivary secretion, and vocal tremors have been reported to accompany stage fright and each symptom can be troubling.[67–69] In addition, speakers and performers often report feelings of breathlessness, flushing, dizziness, and nausea,[12,13,67,68] which can have a direct or indirect deleterious effect upon speaking and/or singing performance. Acute performance stress also reliably activates the HPA axis, particularly if the task is perceived as uncontrollable and/or characterized by social-evaluative threat in which performance has the potential to be negatively judged by others.[20] As the HPA axis is important in regulating the mobilization of energy resources and directing attention and memory, moderate HPA activation can actually improve performance. However, because of the integration of the HPA and autonomic nervous system, many of the effects noted above can negatively impact voice.[15]

Dietrich and Abbott[70] induced stress through a public speaking task in a group of 54 healthy females and measured extralaryngeal electromyography (EMG), F_0, and vocal intensity. Both F_0 and vocal intensity demonstrated significant reduction from baseline during public speaking for the participants as a whole. Heightened EMG signals during the stressor phase were observed in the infrahyoids in a subgroup demonstrating introversion as compared with a subgroup demonstrating personality characteristics more consistent with extroversion, perhaps indicating that some personalities are more susceptible to some aspects of the stressor of public speaking.

Singers and public speakers have used alcohol,[71] hypnosis,[72] and prescription medications[13,71] to reduce or eliminate the symptoms of performance anxiety. Among the prescribed and unprescribed treatments used to combat stage fright symptoms, beta-adrenergic blocking agents are frequently reported.[12–14] Beta-adrenergic blockade works by antagonizing the binding of norepinephrine and epinephrine to beta-adrenergic receptors, thereby attenuating the beta-mediated SNS stimulatory effects on the heart and other organs. Although many speakers and performers taking beta-blockers report improved performance following use, it remains unclear whether the perception of enhanced performance is due to placebo effect or a valid improvement in vocal performance. Additionally, a valid performance improvement could be attributable to either a centrally mediated effect of certain beta-blockers such as propranolol (eg, anxiety reduction) or to peripheral pharmacologic effects upon the cardiac and respiratory/phonatory systems.[73]

The outcome measure in most studies of beta-blockade during vocal performance has been the perception of vocal quality.[12,13,67,69,71,73,74] Investigators reported that performance quality improved,[67,69,74] improved or declined according to dosage[12] or to pre-performance levels of anxiety,[73] or remained unchanged[13,71] after beta-adrenergic blockade. The findings of these studies are equivocal, but consistent with the largely conflicting outcomes of studies designed to identify the effect of stress on the voice.

Acute anxiety, workload, and voice

What are the effects of acute anxiety on voice? An analysis of the audiotaped telephone conversations of two Consolidated Edison employees during the 1977 New York blackout revealed that although the F_0 and intensity of one employee's voice elevated with presumed levels of stress, no such changes were evident in the voice of the other employee.[45] In addition, although the speaking rate elevated for one employee, it remained relatively uniform for the other.

Mendoza and Carballo[39] attempted to analyze vocal acoustics collected during stress induction through cognitive workload (ie, task demands under the pressure of time). Eighty-two

undergraduate students, both male and female, were studied. Vocal F_0, pitch perturbation quotient, amplitude perturbation quotient, and noise-to-harmonic ratio (NHR) were collected and analyzed. Stress induction methods included reciting the alphabet in direct and inverse orders and reading a tongue twister with and without delayed auditory feedback. All conditions required the vowel /a/ be prolonged after the illumination of a red light midway through the task. Subjects were informed that their semester grades were dependent upon the successful performance. Findings included a significant increase in F_0 and a reduction in jitter as compared with baseline for all conditions. Shimmer effects were task dependent with a reduction exhibited in the tongue twister condition. Consistent with the reduction in vocal jitter, NHR diminished over all conditions. The authors concluded that F_0 increases and vocal perturbation (eg, jitter and shimmer) diminishes during experimentally induced stress.

A trial using a modified Stroop Test for stress induction and collection of the acoustic voice signal revealed changes in F_0, vocal jitter, and speaking rate that was dependent upon study phase or minute.[75] The authors reported significant increases in F_0 and speaking rate and a significant reduction in jitter during the second minute of the task. F_0 remained elevated from baseline but diminished somewhat during the remaining three minutes of the Stroop task. Likewise, speaking rate remained elevated and jitter remained diminished during the duration of the task.

Similar research performed on 54 female students appeared to indicate a reduction in F_0 in the stress condition.[76] The students were required to prepare and read their first public speech before 70 fellow students. The nonstress condition consisted of a repetition of the speech before no audience in a subsequent semester. The signals from the two conditions were analyzed and compared. Comparison revealed that with the exception of a lowering of the F_0 in the stress condition, few variables demonstrated significant alteration. Advantages to the study design were the inclusion of both perceptual judgments of vocal quality and a state-anxiety inventory. However, a limitation of the design may have been the instructions: (1) to apply speech pathology optimum voicing principles (ie, easy vocal onset, appropriate breath support, loudness, pitch and prosody, good posture, balanced resonance, and normal speaking rate) and (2) to practice the speech before the experiment. Students reported less anxiety during the stress task, possibly consistent with practice. It could be hypothesized that both the instructions and the opportunity to practice may have artificially altered voice production in the stress condition. In addition, the delay between collection of the stress condition and the nonstress samples could be hypothesized to have invalidated the findings. Curiously, judgments of vocal quality ran contrary to both acoustic findings and the results of the state-anxiety inventory with judges having reported a deteriorated vocal quality under stress in contrast to the nonstress condition. No mention was made to the blindedness or lack thereof, of the judges.

Emotional status and voice

There are those who would argue that the human response to stress is best measured psychologically through the inclusion of indices of emotional status[77,78] in combination with or in lieu of physiological measures (eg, heart rate and GSR). The interaction of voice, gender, emotional or mood status, and heart rate variability in 75 healthy, young, male and female university students was studied by Park et al.[79] Subjects completed the 65-item Profile of Mood States (POMS) that purports to capture rapidly alternating mood states including confusion-bewilderment, anger-hostility, depression-dejection, tension-anxiety, vigor-activity, and fatigue-inertia. Resting heart rate and the vocal acoustic signal were collected and compared with POMS scores. The standard deviation of the F_0 corresponded significantly with "high tension" for the female participants; however, only vocal shimmer was found to differentiate "high tension" from "low tension" in the male participants, the variable being significantly reduced in those reporting a state of "high tension".

Laboratory-induced (neurohumoral) stress and voice

The findings of reduced noise in the vocal signal[39,75,79] and increased F_0 during stress[39,75,80–84] mirrored those of Giddens et al[43] in a double-blind trial in which acoustic and aerodynamic parameters of voice were collected during SNS agonization using cold pressor-induced pain. F_0, jitter, shimmer, speaking rate, maximum flow declination rate, subglottal pressure, and VOT were collected from 12 healthy young adults, both male and female, under four conditions: baseline, cold pressor stress (ie, pain), beta-adrenergic blockade with propranolol or placebo and beta-adrenergic blockade or placebo plus cold pressor. Participants were randomized to a treatment group receiving propranolol or placebo. Numerous trends were observed including increased F_0, mean airflow declination rate, and speaking rate during cold pressor-induced stress for all participants. The findings appeared consistent with significant increases in HPA[85] and SNS-modulated heart rate and blood pressure. However, significant findings were limited to an increase in jitter during beta-adrenergic blockade with propranolol and a significant increase in speaking rate during cold pressor stress in the placebo control group. Several gender differences were observed throughout each phase of the study and the researchers hypothesized that increased baseline sympathetic tone and failure to control for menstrual cycle phase[86–90] in the female participants may have contributed to the paucity of significant findings.

Monitoring of stress in voices of pilots

A means of stress identification in the voice signal as a method for controlling pilots' and astronauts' stress levels and voice identification during stress has been a priority in the field of aerospace medical research.[55,82–84] In their study of 17 male subjects performing a manual tracking task under varying mental workload levels, Brenner et al[57] reported significant increases in heart rate, F_0 and vocal intensity during a manual tracking plus counting task. Similarly, Griffin and Williams[80] reported significant increases in F_0, intensity, and speaking rate for 20 student naval aviators during a dichotic listening, plus visual tracking, plus counting task (ie, maximum

workload). Speaking rate was marginally increased, and the vocal perturbation indices, jitter and shimmer, demonstrated no significant change with increasing workload levels. Similarly, Kuroda et al,[82] Simonov and Frolov,[83] and Simonov et al[84] reported increases in F_0 and associated increases in speech formants with increasing levels of workload (stress) during their attempts to document stress in the voices of male aviators, paratroopers, and air traffic controllers.

Increase in the F_0, however, has not been universally documented as a response to either psychoemotional or physical stress associated with high altitude or space flight in male participants.[38,91] Johannes et al[91] found that high altitude, a physical stressor long thought to activate the SNS, resulted in no significant changes in F_0. In addition, two F_0 patterns were observed during a spacecraft docking task, characterized as a psychological stressor by the authors. One of the three male pilots demonstrated a decrease in F_0 during docking, whereas the remaining two pilots demonstrated increased F_0 while docking. The authors determined that, thereafter, any attempt to document a stress-associated change in F_0 would first require identification of the speaker's "autonomic outlet type." Specifically, they reported that individuals categorized to physiologically reactive subtypes who exhibited increased cardiovascular response to stress demonstrated little change in F_0 associated with stress as contrasted with those persons comprising the least physiologically reactive subtypes (ie, reduced tone and cardiovascular response to stress) and those who demonstrated significant changes in F_0 under stress conditions.[68]

DISCUSSION AND CONCLUSION

Although identification of a universal voice marker has eluded investigators, the myriad of variables involved may offer an explanation. The most obvious variable is stress type. For example, neurohumorally mediated stress (eg, cold pressor stress) might be assumed to bypass the limbic system and its inhibitor, the prefrontal cortex,[92] thus differentiating the effects of neurohumoral stress from those of psychoemotional stress. Advantageous in that it can be easily used in the laboratory, a disadvantage of the cold pressor test is that the pressor response may differ by gender[90] and baseline sympathetic tone, ergo the prognostic utility of cold pressor for prediction of future hypertension and the purpose for which the test is most often used.

In addition, neurohumorally induced stress could be criticized for artificiality. Vocalists and those who train vocalists would argue that perception of vocal quality under natural stress conditions and in natural settings is a better index of stress effects on the voice. Voice scientists, however, would offer the counter argument that we should attempt to identify physiological and/or acoustic parameters contributing to alterations in "vocal quality," and as such the voice must be captured for analysis. An obvious limitation is the difficulty in reproducing natural stress in the laboratory, and alternatively, measurement reliability issues when attempting to capture data in natural settings. Additionally, the response to natural (ie, psychoemotional) stress may vary greatly by individual. Scherer[44] ventures so far as to suggest that some individuals may voluntarily suppress the vocal response to stress.

The psychoemotional stress response may also differ by subtype. For example, social anxiety and fear, although both resulting in significantly increased cortisol production, have been found to differentially affect the central nervous system. Social anxiety elicits widespread cortical activation, but deactivation of the amygdale and hippocampus.[93,94] Conversely, fear is consistently associated with increased activity in the amygdala.[95,96] As postulated by Ganzel et al[97] allostasis, a stress model that emphasizes the dynamics of brain adaptation to chronic and acute stress, the nature of the stressor as well as the context (eg, social support system) in which the stress occurs must be taken under consideration. For example, increased social support has been found to mitigate the HPA axis stress response.[98]

The degree of physiological reaction to psychoemotionally induced stress, originating from the flight or fight response to a perceived threat, may also be dependent on the degree of development (ie, circuit complexity) of a given individual's prefrontal cortex and the subject's baseline autonomic tone, genetically influenced under acute stress and genetically and allostatic load influenced in cases of chronic stress.[97] Perhaps Johannes et al[99] and Johannes et al[91] are correct that autonomic outlet type (ie, baseline autonomic tone) will differentiate a given individual's reaction to a physical stressor such as high altitude. As such, they argued that changes in vocal F_0 must be correlated with electrocardiographic, respiratory, blood pressure, galvanic skin response (GSR), and electroencephalographic changes at sea level and then extreme altitude.[91,99]

Furthermore, baseline sympathetic tone may differ by gender and as such inclusion of both sexes may be a confounding factor in the search for the vocal characteristics of stress. Gender differences in cardiovascular response to performance anxiety and/or stress have been reported[86,100] with menstrual cycle phase[86–89,101] and increased vulnerability to stress-associated disorders, including those of voice, in the female[19,102–105] being offered as partial explanations. These findings may indicate a gender-determined autonomic variability in response to stress and the effects of stress on voice.

Review of the literature may indicate that future research should use designs that stratify by gender and baseline autonomic tone. However, the vocal response to stress may be as individual and unique as the voice itself. Perhaps complex constructs such as personality, influenced by birth order and other and innumerable factors, should be considered. As such, single-subject studies in which individual participants would serve as their own controls might be a preferable approach in the search for the vocal response to stress.

Alternatively, perhaps Porges[106] is correct in his assertion that those of us limiting ourselves to SNS and HPA indices of stress should look elsewhere, specifically at parasympathetic parameters because of the extensive parasympathetic innervation to the larynx, pharynx, face, and head. The commonly theorized reaction of an organism to a stressor is release of vasopressin and mobilization behavior including increased heart rate, pupillary dilation, and increased respiration to

facilitate fight or flight. Polyvagal theory,[106–109] however, allows for the possibility of a very different response to massive release of vasopressin, that of immobilization behavior and systems shutdown. Indices of this extreme response to a stressor would include bradycardia, miosis, and tracheal constriction through the dorsal vagal motor nucleus and vagal efferent nerves. The expected change in the voice from baseline would be quite different from that expected during mobilization behavior.

In summary, although findings are heterogeneous, review of well-controlled studies examining the effects of stress on the voice demonstrates a consistent if not universal trend of an increase in F_0.[36,39,43,75,80,81,83,84] An explanation for the increase in F_0 under duress may be stress-associated tensing of the musculature, specifically the cricothyroid muscle. Other explanations for the finding may include SNS-mediated increases in heart rate, blood pressure, and bronchodilation,[27–30] which in turn may increase F_0. In addition, reduced noise as reflected in jitter[39,75] and shimmer[79] and an associated increase in vocal jitter during beta-adrenergic blockade of the SNS stimulatory effects[43] is documented, but less frequently. The absence of universal trends may be explained, at least in part, by individual differences in response and susceptibility to various stressors as well as the possibility of parasympathetic influences as contrasted with the more traditionally accepted model of SNS and HPA arousal.

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