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

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