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The covariation of acoustic features of infant cries and autonomic state

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HIGHLIGHTS

- Vocal prosody and the heart are regulated by a shared neural pathway.
- A novel automated tool was applied to objectively characterize vocal prosody.
- Vocal prosody is a sensitive index of autonomic activity in human infants.
- Specific acoustic features conveying vocal prosody covary with physiological state.
- Faster heart rate was associated with restricted modulation (i.e., less prosody) of acoustic features.

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ABSTRACT

The evolution of the autonomic nervous system provides an organizing principle to interpret the adaptive significance of physiological systems in promoting social behavior and responding to social challenges. This phylogenetic shift in neural regulation of the autonomic nervous system in mammals has produced a neuroanatomically integrated social engagement system, including neural mechanisms that regulate both cardiac vagal tone and muscles involved in vocalization. Mammalian vocalizations are part of a conspecific social communication system, with several mammalian species modulating acoustic features of vocalizations to signal affective state. Prosody, defined by variations in rhythm and pitch, is a feature of mammalian vocalizations that communicate emotion and affective state. While the covariation between physiological state and the acoustic frequencies of vocalizations is neurophysiologically based, few studies have investigated the covariation between vocal prosody and autonomic state. In response to this paucity of scientific evidence, the current study explored the utility of vocal prosody as a sensitive index of autonomic activity in human infants during the Still Face challenge. Overall, significant correlations were observed between several acoustic features of the infant vocalizations and autonomic state, demonstrating an association between shorter heart period and reductions in heart period and respiratory sinus arrhythmia following the challenge with the dampening of the modulation of acoustic features (fundamental frequency, variance, 50% bandwidth, and duration) that are perceived as prosody.

1. Introduction

The phylogenetic shift in neural regulation of the autonomic nervous system (ANS) in vertebrates has produced a myelinated efferent vagal pathway unique to mammals, which can rapidly regulate visceral state and facilitate social engagement [1–3]. The brainstem origin of this motor-specific vagal pathway shares evolutionary origins with the special visceral efferent pathways traveling through five cranial nerves (trigeminal, facial, glossopharyngeal, vagus, and accessory), which innervate somatic muscles typically not associated with the neurophysiology of the ANS. In addition to its innervation of smooth and cardiac muscles, the mammalian vagus is neuroanatomically linked to the striated muscles of the face and head that regulate social engagement, intimately involved with behaviors such as facial expression, hearing, and vocalization, that contribute to the complex repertoire of both social expressions (i.e., signaling) and social experiences (i.e., receiving) [1–4].

Mammalian vocalizations are part of a social communication system that evolved to communicate and to signal a danger or threat to conspecifics, with many mammalian species modulating their acoustic features of vocalizations to signal states of fear, aggression, pain, and hunger [5–7]. Human infant crying, in particular, serve to elicit parental caregiving, with variations in their cry features known to

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affect adults' perceptions and physiologic responses [8–11]. At the clinical level the acoustic features of infants have been shown to exhibit a wide variance relative to the intensity or degree of a particular challenge and/or medical status [8,12,13]. For example, previous reports have shown a relationship between acoustic characteristics of infant cries and diagnoses related to neurological damage, prematurity, medical conditions, pain (e.g., circumcision), sudden infant death syndrome, and substance exposure during pregnancy [8,14–19]. In particular, the frequency of infant cries may be interpreted as indicators of health status and [20–23], and can indicate distress when infants exhibit high frequency shrill cries [24–27].

Importantly, this covariation between physiological state and the acoustic frequencies of vocalizations is neurophysiologically based on a shared neural pathway, as suggested by the Polyvagal Theory [5]. Prior reports have demonstrated the relationship between variations in infant cry prosody and infant neurobehavioral organization and autonomic activity [28]. For example, higher cry threshold and shorter cry duration have been shown to be predictive of disrupted autonomic regulation [28]. However, the parallel between the acoustic features characterized as vocal prosody (i.e., the cluster of vocal features such as pitch modulation and frequency change) has not been quantified in detail, nor has its parallel with autonomic function been systematically described. To date, only the frequency of vocalization has been correlated to cardiac vagal tone (i.e., respiratory sinus arrhythmia) and then only in healthy, although severely stressed, infants [29]. Here we expand these previous observations to demonstrate the covariation of several prosodic features with autonomic state in human infants.

1.1. Role of vocalizations

The physiological correlates of vocal prosody have been understudied, although in clinical settings vocal prosody has been assumed to be related to health. Most investigations have focused on vocalization as a reflection of neurobehavioral dysregulation, often in relation to a specific disorder [30–32], contextual features (e.g., danger) [33,34], or transitory psychological state (e.g., pain) [35,36] and with little scientific interest in a potential relation between prosody and autonomic state. Consistent with the Polyvagal Theory [5], the current study assumes that the frequency modulations within vocalizations are an effective and efficient form of vocal communication with an adaptive function, capable of conveying the physiological state of the signaler to other members of its species. This 'automatic' communication of the signaler's state is hypothesized to facilitate reproductive, parenting, and social behaviors by indicating when the organism is safe to approach [5].

Prosodic vocalization characteristics are generally expanded during positive social interactions among humans, and reduced during stress or illness [29,37,38]. For example, distressed infants often demonstrate a high-pitched cry with little frequency modulation and shortened duration [16,29]. Consistent with the polyvagal hypothesis, the decreased neural tone seen in distressed or unhealthy infants is believed to reduce the inhibitory effect on the heart and bronchi and the contraction of laryngeal muscles, thereby producing dramatic increases in both heart rate and respiration rate as well as the fundamental frequency of the cry [5,29,39,40]. This convergence of cardiac vagal tone and vocal prosody has also been demonstrated in healthy, though severely stressed, newborns following circumcision. Cardiac vagal tone, measured by respiratory sinus arrhythmia (RSA), was significantly reduced, and individual differences in resting cardiac vagal tone were shown to correlate with the pitch of the infants' high frequency pain cries [29].

1.2. Shared neurophysiology underlying vocal prosody and autonomic regulation

The evolutionary development of the striated muscles necessary for mammalian vocalizations paralleled the evolutionary changes in vagal regulation of the heart [41]. Ultimately, this phylogenetic convergence of several neural circuits has served to form an integrated functional social engagement system [4,42,43]. Importantly, the same brainstem structures are involved in both the regulation of the heart rate via the myelinated branch of the vagus (i.e., cardiac vagal tone measured via respiratory sinus arrhythmia) and vocalizations via the laryngeal and pharyngeal muscles [44,45]. Thus, the vagal output to the laryngeal and pharyngeal muscles reflected in the prosodic features of vocalization may mirror the vagal influence to the heart. Therefore, procedures that disrupt homeostatic processes may result in both depressed cardiac vagal tone and vocalizations characterized by a higher pitch and less prosody [5]. Moreover, a chronically depressed cardiac vagal tone would reflect, on an individual level, poor homeostasis and a neurophysiological vulnerability to a challenge.

The brain substrates mediating vocalizations remain relatively conserved across most mammalian species [46], and with the exception of some cortical influences, the brain structures reside primarily in the subcortical forebrain — namely limbic structures and the hypothalamus, as well as the periaqueductal gray of the midbrain, leading to the motor neurons that innervate the larynx [46,47]. Regulation of special visceral efferents to the laryngeal and pharyngeal muscles involved in vocalization, as well as general visceral efferents producing a rhythmic oscillation in heart associated with breathing (i.e., RSA), stem from the myelinated vagal pathway originating in the nucleus ambiguus (NA).

Vocalizations represent direct central regulation of laryngeal and pharyngeal muscles, with the neural regulation of these muscles occurring in the same brainstem nuclei involved in regulation of myelinated vagal pathways to the heart [46,47]. In particular, specific vocalization features are plausibly linked to the neural regulation of the laryngeal and pharyngeal muscles. For example, a positive relationship between the height of the larynx muscle and vocal fundamental frequency exists, with vocal pitch changing relative to changing larynx height (i.e., the relaxation and contraction of the muscle) [48,49]. Thus, rapid change in fundamental frequency during a vocalization represents modulation of neural tone to the laryngeal muscles. The laryngeal muscles provide active opening of the glottis (i.e., the combination of the vocal cords and the space in between the folds) to enhance the flow during inspiration, and partial closing to reduce air flow and increase subglottal pressure during expiration. During expiration, the posterior cricoarytenoid (PCA) muscle (regulated by the recurrent laryngeal branch of the vagus) is phasically active while activity of the cricothyroid (CT) muscle (regulated by superior laryngeal nerve) tends to increase. Likewise, the thyroarytenoid (TA) muscle (regulated by superior laryngeal nerve) is also more active during expiration. The TA is comprised of two divisions — with the external division (TA-X) adducting the vocal fold, and the vocalis division (TA-V) modulating sound quality [50].

The activity of the laryngeal and pharyngeal muscles parallel the alternating increase and decrease in myelinated vagal output to the heart during exhalation and inhalation, respectively (Table 1). This occurs because respiration modulates vocalizations and "gates" the efferent action of the myelinated vagus on the sino-atrial node [51]. Moreover, with the laryngeal and pharyngeal muscles and heart both

Table 1
Laryngeal regulation of the glottis parallels the alternating increase and decrease in myelinated vagal input to the heart during exhalation and inhalation (i.e., RSA).

Inhalation	Exhalation
Less vagal output	More vagal output
Laryngeal muscles abduct	Laryngeal muscles adduct
Posterior cricoarytenoid muscle (via recurrent laryngeal nerve)	Thyroarytenoid muscle (via recurrent laryngeal nerve)
Cricothyroid muscle (via superior laryngeal nerve)	
Glottis opens	Glottis closes