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Respiratory Sinus Arrhythmia: A Transdiagnostic Biomarker of Emotion Dysregulation and Psychopathology

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Abstract

In the past two decades, respiratory sinus arrhythmia (RSA)—an index of parasympathetic nervous system (PNS)-mediated cardiac control—has emerged as a reliable peripheral biomarker of emotion regulation (ER). Reduced RSA and excessive RSA reactivity (i.e., withdrawal) to emotional challenge are observed consistently among individuals with poor ER capabilities, including those with various forms of internalizing and externalizing psychopathology, and those with specific psychopathological syndromes, including anxiety, phobias, attention problems, autism, callousness, conduct disorder, depression, non-suicidal self-injury, panic disorder, and trait hostility. Emerging evidence suggests that low RSA and excessive RSA reactivity index poor ER because they are downstream peripheral markers of prefrontal cortex (PFC) dysfunction. Poorly modulated inhibitory efferent pathways from the medial PFC to the PNS result in reduced RSA and excessive RSA reactivity. According to this perspective, RSA is a non-invasive proxy for poor executive control over behavior, which characterizes most forms of psychopathology.

Keywords

emotion regulation; emotion dysregulation; parasympathetic nervous system; psychopathology; internalizing; externalizing

Introduction

Although the study of human emotion can be traced at least as far back as Darwin [1], for much of the 20th Century psychological science was dominated by behavioral and cognitive paradigms that viewed emotional states as subjective, unquantifiable, and unamenable to scientific inquiry [2]. Beginning in the mid-1990s, these paradigms gave way to the contemporary view that emotional experience and expression are integral to both positive and negative psychological adjustment. This paradigm shift was facilitated by several developments, including a 1990, yearlong seminar on emotion held at the National Institute

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of Mental Health, and a 1991 conference on the development of emotion regulation (ER) that was sponsored by the John D. and Catherine T. MacArthur Foundation Early Childhood Network. From these, landmark volumes on the nature of emotion [3] and ER [4] emerged. Among other important contributions, chapters in these volumes demonstrated how emotional states can be inferred, verified, and quantified by measuring appropriate biological systems [5, 6, 7]. The study of emotion soon became mainstream, and is currently central to the positive and negative valence systems of the Research Domain Criteria (RDoC) [8*].

Emotion Dysregulation and Psychopathology

It is now widely recognized that problems with ER confer vulnerability to a wide range of psychopathological outcomes [9*], and that difficulties with ER characterize almost all forms of psychopathology [10*,11*]. ER can be defined as the set of processes through which emotional experience and expression are shaped in the service of adaptive behavior [12]. Such shaping of emotion may occur through various mechanisms, including attentional, cognitive, social, and behavioral [13*]. In contrast, emotion dysregulation comprises a pattern of emotional experience and/or expression that interferes with appropriate goal directed behavior [14]. In almost all forms of psychopathology, one or more negative emotions (e.g., sadness, anxiety, panic, anger, rage) is experienced either too intensely or too persistently to be adaptive [15*,16*].

Central Nervous System Substrates of Emotion Dysregulation

Recognition that emotion dysregulation plays such a prominent role in vulnerability to psychopathology has led to considerable research on its central nervous system (CNS) substrates. Taken together, this research demonstrates that ER is subserved by top-down, cortical (prefrontal) brain networks that mature into the early 20s [17*,18*,19*]. Trait anxiety, which confers vulnerability to internalizing disorders (e.g., anxiety, depression), is volitionally regulated through prefrontal inhibition of subcortical amygdalar activity and reactivity, whereas trait impulsivity, which confers vulnerability to externalizing disorders (e.g., ADHD, substance use disorders) is volitionally regulated through prefrontal inhibition of subcortical striatal activity and reactivity [20, 21, 22*]. Those with anxiety disorders exhibit less functional connectivity in amygdalar-prefrontal connections than controls [23], and those with externalizing disorders exhibit less functional connectivity in prefrontal-striatal connections than controls [24]. Moreover, deficient top-down control of the amygdala by the PFC, and reduced functional connectivity between the amygdala and the PFC, are observed among those with deficient ER [25].

Peripheral Nervous System Markers of Emotion Dysregulation

Although the CNS substrates of ER are well characterized [16], measuring CNS processes requires neuroimaging protocols that are expensive, limited in ecological validity, and difficult to use with certain populations, such as young children and those with severe psychopathology. For these reasons and others, many researchers have turned to peripheral nervous system markers of ER, most notably respiratory sinus arrhythmia (RSA). The term RSA refers to ebbing and flowing of heart rate (HR) across the respiratory cycle. This cyclic

pattern of HR occurs due to increases in inhibitory parasympathetic efference during exhalation, and decreases in inhibitory parasympathetic efference during inhalation [10, 26*]. RSA can be assessed using any of several methods [27*], the most common being spectral analysis of the electrocardiographic R-wave time series. Spectral analysis isolates parasympathetic influence on the heart—as indexed by RSA—by filtering out low- and mid-frequency heart rate variability (see Figure 1). This is necessary because these frequency bands include parasympathetic, sympathetic, diurnal, and non-neural components [28*].

RSA demonstrates two important qualities that establish its validity as a transdiagnostic biomarker of emotion dysregulation. First, abnormally low resting RSA and/or large reductions in RSA specifically during emotion evocation are associated with symptoms of both internalizing and externalizing psychopathology [29, 30], and with numerous psychopathological syndromes, including anxiety [31], phobias [32], attention problems [33], autism [34], callousness [35], conduct disorder [15], depression [36], non-suicidal self-injury [37], panic disorder [38], and trait hostility [39]. This remarkably long list suggests that RSA marks one or more core self-regulatory functions that are disrupted across diverse forms of psychopathology [10, 11]. Importantly, *excessive RSA withdrawal among psychopathological groups is specific to emotion evocation, and is not observed in many other stimulus conditions* [40].

Second, mounting evidence suggests that RSA reflects prefrontal cortex (PFC) function, and therefore indexes—albeit peripherally—CNS substrates of ER [11]. This assertion, which is articulated in Thayer's neurovisceral integration theory [41, 42*], is based on several considerations, including existence of inhibitory neural efferent pathways from the medial PFC to the PNS; (2) positive associations between resting RSA and performance on executive function tasks; and (3) positive correlations between RSA and PFC activity during neuroimaging tasks.

Inhibitory efferent pathways from the medial PFC to the PNS have been characterized for some time [43]. The prefrontal, cingulate, and insular cortices form an interconnected neural network that exhibits feed-forward and feedback connections with the amygdala [41]. Activation of the central nucleus of the amygdala via this network provides inhibition of the nucleus solitary tract, which in turn inhibits vagal motor neurons in the dorsal motor nucleus and the nucleus ambiguus [41]. These structures provide inhibitory input via the PNS to the sinoatrial node [26]. Through this structural network, PFC function is translated into RSA. Since most forms of psychopathology are characterized by PFC dysfunction [44, 45*], they are also characterized by low resting RSA and excessive RSA reactivity, peripheral biomarkers of poor executive control [41].

Consistent with this interpretation, positive associations between resting RSA and performance on executive function (EF) tasks have been reported. For example, among military personnel, those who score high on baseline RSA outperform those who score low on RSA on stimulus detection and addition tasks [46, 46].

Positive correlations between RSA and PFC function have also been demonstrated using positron emission tomography. During emotion-induction, RSA correlates with cerebral

blood flow in both the PFC and the anterior cingulate cortex [47*]. Induction of various emotions reduces both RSA and blood flow in these regions.

Taken together, this research provides an explanation for the remarkably consistent findings of low resting RSA and excessive RSA reactivity among those with diverse forms of psychopathology. Non-specific vulnerability to psychopathology is conferred by poor executive (i.e., prefrontal) control over behavior [44, 45], which is reflected in measures of RSA given structural and functional connections between the PFC and PNS efferents to the heart via the vagus nerve. Specific forms of psychopathology are determined by interactions of PFC dysfunction with largely independent subcortical neural circuits that generate approach- and avoidance-related affect. According to this perspective, emotions are *generated* by phylogenetically old, subcortical neural circuits, but *regulated* by phylogenetically new, cortical neural circuits [10, 11].

Conclusions

A primary aim of psychophysiological research is to use peripheral measures to make inferences about CNS processes that are difficult and in some cases impossible to index noninvasively [26]. However, much of the literature on RSA-behavior relations is either agnostic with respect to central nervous system substrates of RSA, or implies that the PNS plays a causal role in ER. As reviewed above, it is far more likely that the PNS—via the vagus nerve—mediates links between the PFC and cardiovascular function. In future research, authors should interpret their findings not only at the ANS level, but at the CNS level as well. Furthermore, since cortical PFC dysfunction interacts with subcortical neural systems to confer vulnerability to specific forms of psychopathology, researchers are encouraged to assess multiple CNS and/or autonomic systems concurrently. Doing so provides considerably more specificity in distinguishing among psychiatric disorders [10, 48]. Such an approach is fully consistent with the RDoC initiative [8], which assumes that multiple neurobiological systems interact to affect behavior. By studying such interactions, we are likely to advance our understanding of psychopathology in the upcoming decade [11].

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Highlights

RSA is a valid and reliable biomarker of emotion regulation capacity in humans.

Low tonic RSA and excessive RSA reactivity to emotion evocation mark PFC dysfunction.

Effective cortical regulation of subcortical neural circuits marks psychological health.

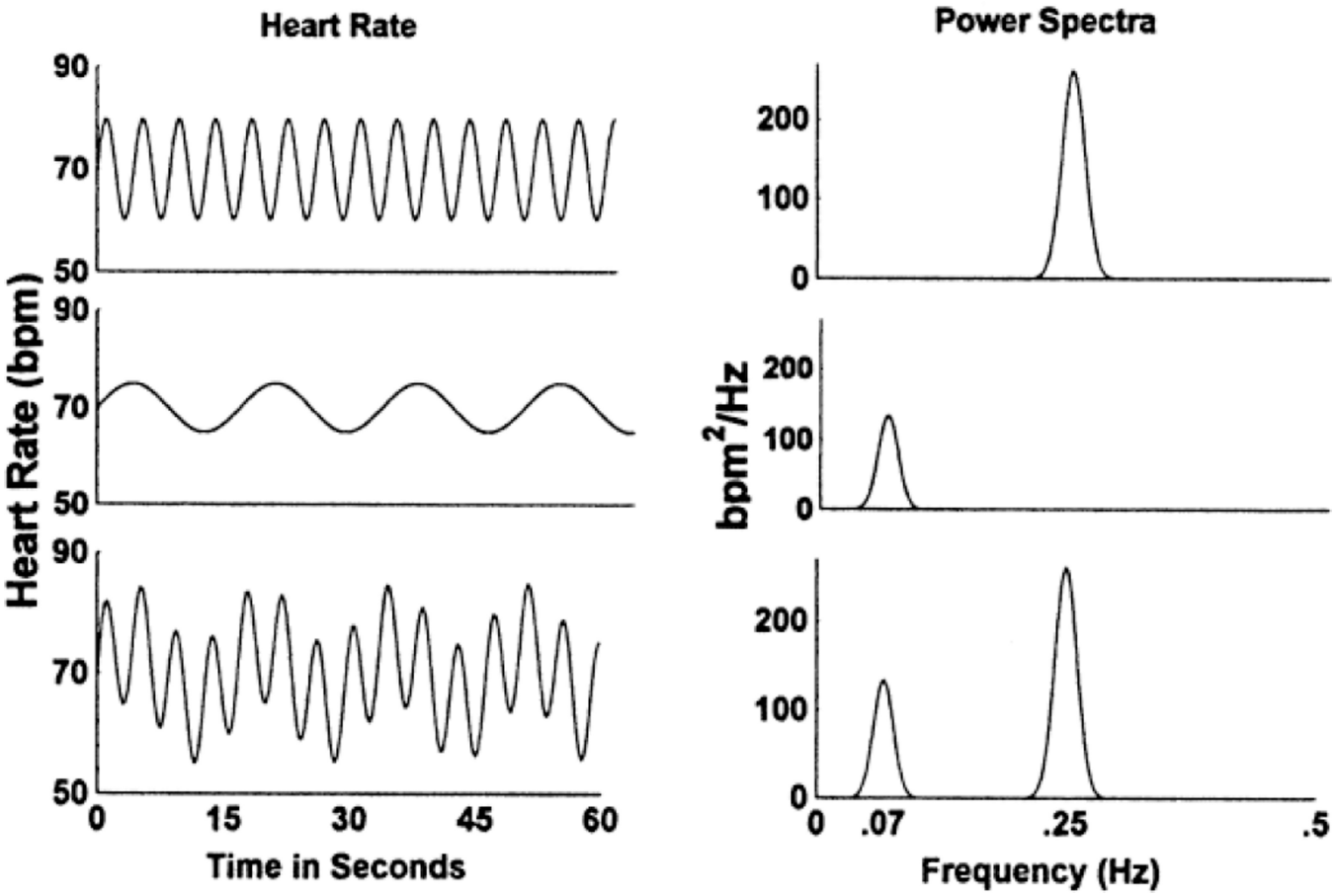

Figure 1.

Fictitious heart rate (HR) signals and associated power spectra. The top two panels represent pure high-frequency HR variability (0.25 Hz), as associated with RSA. The middle two panels represent low-frequency HR variability (0.07 Hz), which is of parasympathetic, sympathetic, diurnal, and nonneural origin. The bottom panels represent the combined signal including both high- and low-frequency components. Actual heart rate signals include spectral power at additional frequencies.