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ARTICLE *in* REVIEW OF GENERAL PSYCHOLOGY · AUGUST 2006

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## Heart Rate Variability as an Index of Regulated Emotional Responding

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The study of individual differences in emotional responding can provide considerable insight into interpersonal dynamics and the etiology of psychopathology. Heart rate variability (HRV) analysis is emerging as an objective measure of regulated emotional responding (generating emotional responses of appropriate timing and magnitude). This review provides a theoretical and empirical rationale for the use of HRV as an index of individual differences in regulated emotional responding. Two major theoretical frameworks that articulate the role of HRV in emotional responding are presented, and relevant empirical literature is reviewed. The case is made that HRV is an accessible research tool that can increase the understanding of emotion in social and psychopathological processes.

*Keywords:* heart rate variability, emotion, affect, social interaction, psychopathology

Although the nature of emotions has been the subject of much debate, most theorists consider emotions to be multifaceted processes involving coordinated changes in peripheral and central physiology (Thayer & Siegle, 2002), behavior or behavioral tendencies, and cognitive processing. Emotions guide our decisions (Damasio, 2003, pp. 144–150), provide a substrate for social interaction (Keltner & Kring, 1998), and facilitate responses to challenge (Tooby & Cosmides, 1990). Emotions that are expressed with sensitivity to the situational context in which they unfold, both in terms of timing/occurrence and magnitude, are more likely to facilitate adaptive responses. Emotions are matched to their context through various regulatory strategies implemented both during and after initial emotional expression (Gross, 1998). Indeed, the capacity to regulate emotion is vital to social functioning (Eisenberg, 2001) and maintaining mental health (Gross & Munoz, 1995).

The emotions that humans experience while interacting with their environment are associated with varying degrees of physiological arousal (Levenson, 2003). A key system involved in the generation of this physiological arousal is the autonomic nervous system (ANS). The ANS is subdivided into an excitatory sympathetic nervous system (SNS) and an inhibitory parasympathetic nervous system (PNS) that often interact antagonistically to produce varying degrees of physiological arousal. During physical or psychological stress, activity of the SNS becomes dominant, producing physiological arousal to aid in adapting to the challenge. An increased pulse, or heart rate, is characteristic of this state of arousal. During periods of relative safety and stability, the PNS is dominant and maintains a lower degree of physiological arousal and a decreased heart rate. The ease with which an individual can transition between high and low arousal states is dependent on the ability of the ANS to rapidly vary heart rate.

Emotion regulation depends critically on an individual's ability to adjust physiological arousal on a momentary basis (Gross, 1998). A flexible ANS allows for rapid generation or modulation of physiological and emotional states in accordance with situational demands. In contrast, autonomic rigidity results in a lessened capacity to generate or alter physiological and emotional responses in synchrony with

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We are grateful to James Eason of Washington and Lee University for his thoughtful comments on this article.

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changes in the environment. As described later in this article, heart rate variability (HRV) is a measure of the continuous interplay between sympathetic and parasympathetic influences on heart rate that yields information about autonomic flexibility and thereby represents the capacity for regulated emotional responding.

This article provides theoretical and empirical support for the emergence of HRV as an important marker of emotion regulatory ability. The first section of the article provides an introduction to the physiological underpinnings of HRV. The roles of autonomic and central nervous system influences on cardiac functioning are emphasized. The second section describes the methods involved in measuring heart rate and deriving HRV "values" through three major classes of analyses. The third section describes contemporary theoretical models of the self-regulatory function of HRV, including the polyvagal theory (Porges, 1997, 2001) and the model of neurovisceral integration (Thayer & Lane, 2000). A brief review of empirical research supporting the role of HRV in emotion regulation (in a variety of contexts) is presented and evaluated in the fourth section. The final section includes a brief summary and suggested directions for future study. The goal is to provide an introduction to HRV, with the hopes of stimulating future research using this promising physiological index of emotion regulatory ability.

### Physiological Underpinnings of HRV

HRV reflects the degree to which cardiac activity can be modulated to meet changing situational demands. Although HRV is influenced by numerous physiological and environmental factors, two are particularly prominent and of psychophysiological importance: the influence of the ANS on cardiac activity and ANS regulation by the central autonomic network (CAN). The heart is innervated by the sympathetic and parasympathetic (vagal) branches of the ANS, which exert a regulatory influence on heart rate by influencing the activity of the primary pacemaker of the heart, the sinoatrial node. The sinoatrial node generates action potentials that course throughout the cardiac tissue, causing regions of the myocardium (heart muscle) to contract in the orchestrated fashion that characterizes a heartbeat. Activation of

sympathetic fibers has an excitatory influence on the firing rate of the sinoatrial node and, among other things, results in increased heart rate. In contrast, parasympathetic activation has an inhibitory influence on the pacemaking activity of the sinoatrial node and produces decreased heart rate. Alternatively, it can be said that the two autonomic branches regulate the lengths of time between consecutive heartbeats, or the interbeat intervals, with faster heart rates corresponding to shorter interbeat intervals and vice versa. The PNS and SNS act antagonistically to influence cardiac activity. For example, an increase in heart rate could arise from either increased sympathetic activity or decreased parasympathetic inhibition (vagal withdrawal). Although both autonomic branches exert a constant influence on heart rate, parasympathetic influence is predominant at rest and serves to maintain resting heart rate well below the intrinsic firing rate of the sinoatrial node (Berntson et al., 1997).

The two branches of the ANS rely on different signaling mechanisms with different temporal effects. Sympathetic influence on heart rate is mediated by neurotransmission of norepinephrine and possesses a slow course of action on cardiac function. That is, changes in heart rate due to sympathetic activation unfold rather slowly, with peak effect observed after about 4 s and return to baseline after about 20 s. In contrast, parasympathetic regulation of the heart is mediated by acetylcholine neurotransmission and has a very short latency of response, with peak effect at about 0.5 s and return to baseline within 1 s (Berntson et al., 1997; Pumpila, Howorka, Groves, Chester, & Nolan, 2002). The ability of the PNS to rapidly modulate cardiac activity allows for flexibility in responding to environmental demands with physiological and emotional arousal. Owing to the difference in their latencies of action, the oscillations in heart rate produced by the two autonomic branches occur at different speeds, or frequencies. This serves as the basis for the frequency-based HRV analyses described below.

Breathing air into the lungs temporarily gates off the influence of the parasympathetic influence on heart rate, producing a heart rate increase (see Berntson, Cacioppo, & Quigley, 1993). Breathing air out of the lungs reinstates parasympathetic influence on heart rate, resulting in a heart rate decrease. This rhythmic os-

cillation in heart rate produced by respiration is called respiratory sinus arrhythmia (Bernardi, Porta, Gabutti, Spicuzza, & Sleight, 2001; Berntson et al., 1993). As only cardiac parasympathetic activity possesses a latency of action rapid enough to covary with respiration, respiratory sinus arrhythmia is a phenomenon known to be entirely mediated by the PNS. In fact, a large majority of parasympathetically mediated variation in heart rate is produced by respiratory sinus arrhythmia (Berntson et al., 1997), and many researchers have reported the magnitude of respiratory sinus arrhythmia as an index of parasympathetically mediated HRV. Historically, researchers assumed that the magnitude of respiratory sinus arrhythmia could serve as a linear index of "vagal tone," or the electrochemical activity of the vagus nerve (Grossman, Stemmler, & Meinhardt, 1990; Saul & Cohen, 1994). However, this view has been challenged (Pyetan & Akselrod, 2003), and strictly speaking, respiratory sinus arrhythmia is an estimate of parasympathetically mediated HRV rather than of vagal tone.

The autonomic influences on heart rate are regulated remotely by the distributed network of brain areas composing the CAN (Benarroch, 1993). The CAN supports regulated emotional responding by flexibly adjusting physiological arousal in accordance with changing situational demands. Thus, the CAN is critically involved in integrating physiological responses in the services of emotional expression, responding to environmental demands, goal-directed behavior, and homeostatic regulation. The neuroanatomical composition of the CAN includes cortical (medial prefrontal and insular cortices), limbic (anterior cingulate cortex, hypothalamus, central nucleus of the amygdala, bed nucleus of the stria terminalis), and brainstem (periaqueductal gray matter, ventrolateral medulla, parabrachial nucleus, nucleus of the solitary tract) regions. The CAN receives input from visceral afferents regarding the physiological conditions inside the body and input from sensory processing areas in the brain regarding the external sensory environment (Benarroch, 1993). This input allows the CAN to dynamically adjust physiological arousal, including arousal associated with emotional expression and regulation, in response to changes in internal and external conditions. The output of the CAN is transmitted to the sinoatrial node (and many other or-

gans) through the SNS and PNS and directly influences heart rate. Therefore, HRV reflects the moment-to-moment output of the CAN and, by proxy, an individual's capacity to generate regulated physiological responses in the context of emotional expression (Thayer & Lane, 2000; Thayer & Siegle, 2002).

### Assessment and Calculation of HRV

HRV measures are derived by estimating the variation among a set of temporally ordered interbeat intervals. Obtaining a series of interbeat intervals requires a continuous measure of heart rate, typically electrocardiography (ECG). A simple lead II configuration (electrodes on the right arm and left leg, plus a ground) is sufficient for HRV analysis. Data should be sampled at a rate rapid enough to provide a high-resolution signal (e.g., 500–1,000 Hz; Berntson et al., 1997). After collection, the series of interbeat intervals must be corrected for abnormal beats and artifacts (see Kamath & Fallen, 1995). Commercially available software is then used to define the interbeat intervals within an ECG recording. Several waveforms can be identified from a raw ECG recording, each corresponding to different components of a heartbeat. In HRV analysis, interbeat intervals are almost always defined as the temporal distance between R-spikes (see Figure 1), the prominent waveform that corresponds to the contraction of the heart's ventricles. Because in theory HRV analyses are to be applied to a set of interbeat intervals defined by consecutive "normal" beats (i.e., produced by sinoatrial depolarizations), the interbeat intervals are often referred to as "normal-to-normal" (NN) intervals. Although in practice HRV analyses can be applied to a *corrected* series of interbeat intervals, not true normal-to-normal intervals, the abbreviation *NN* has been incorporated into the names of many analyses.

The obtained set of interbeat intervals is used as input data for computing three classes of HRV analyses. The statistical class of analyses consists of variance-based calculations (described below) performed on the set of interbeat intervals and yielding numerical estimates for HRV in temporal units (e.g., milliseconds). Within the frequency class of analyses, a technique known as power spectral analysis is used to partition the variance of the interbeat inter-

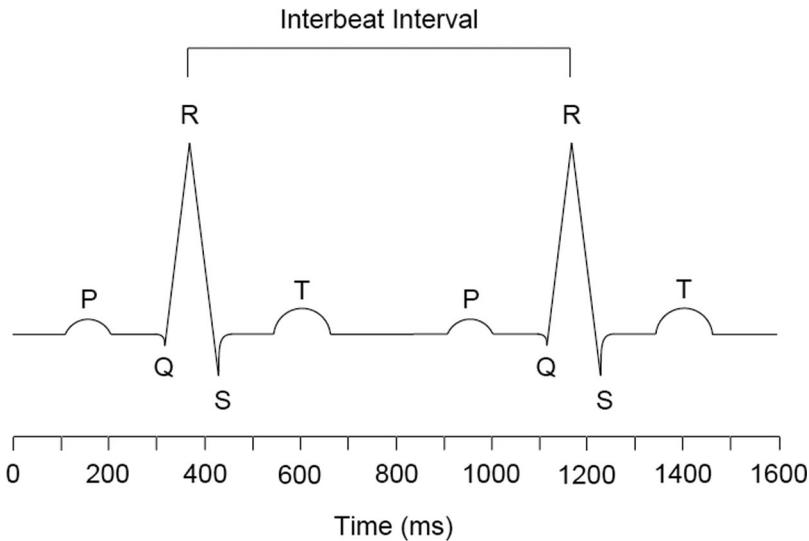


Figure 1. Idealized electrocardiograph segment representing two heartbeats. Waveforms are labeled with letters and correspond with specific electrophysiological events during a heartbeat. The interbeat interval is defined by the temporal distance between R-spikes, the waveforms corresponding to depolarization of the heart's ventricles.

vals into a spectrum of frequencies. The amount of variance within a given frequency range, represented by the area under the curve, is referred to as power. The geometrical class of analyses derives estimates of HRV from the geometric properties of the sample density distributions of either the interbeat intervals or the differences between consecutive interbeat intervals (see Malik, 1995). Unlike the statistical class of analyses, geometrical analyses are less affected by aberrations in the ECG input data but require more extended sampling of ECG and provide less precise estimates of HRV (Task Force, 1996). Therefore, they are less frequently reported and are not described here. The interested reader is referred to Malik (1995) and Task Force (1996).

Statistical analyses are frequently reported and can be computed to represent overall HRV or HRV at different frequencies (see Kleiger, Stein, Bosner, & Rottman, 1995). For example, *SDNN* refers to the standard deviation of NN intervals, and *SDANN* refers to the standard deviation of the average NN interval computed across all 5-min recording segments. *SDNN* reflects overall HRV, whereas *SDANN* reflects HRV occurring at cycles longer than 5 min. Also among the statistical analyses are those

involving differences between interbeat intervals, such as the mean squared difference of successive NN intervals (MSSD), the square root of MSSD (RMSSD), the number of pairs of adjacent NN intervals differing by more than 50 ms (NN50), and the proportion derived by dividing the NN50 by the total number of NN intervals (pNN50). The MSSD, RMSSD, NN50, and pNN50 are thought to represent parasympathetically mediated HRV (Task Force, 1996).

The frequency-based technique of power spectral analysis is a relatively new and sophisticated approach to quantifying HRV. Power spectral analysis parses from the ECG signal the quantity of HRV occurring at different frequencies (these frequencies are expressed in hertz, calculated as cycles per second). The technique results in a power spectrum, a distribution of variance in heart rate at different frequencies (see Figure 2). Various approaches are used to extract the oscillatory components of HRV from a set of sequential interbeat intervals, the most common being the fast Fourier transform and autoregressive modeling techniques. Both approaches have been compared in detail (Cerutti, Bianchi, & Mainardi, 1995) and found to yield similar results (Task Force, 1996).

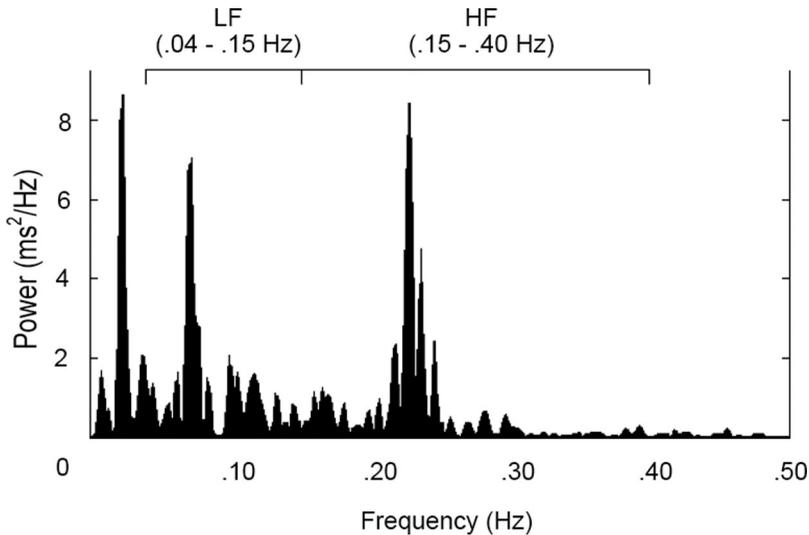


Figure 2. An example of a heart rate variability power spectrum obtained using the fast Fourier transform on a 5-min recording obtained from a resting subject in supine position. The low-frequency (LF) component occurs between .04 and .15 Hz and the high-frequency (HF) component occurs between .15 and .40 Hz. Hz = cycles per second.

The power spectrum contains prominent bands representing the major oscillatory components of HRV. The high-frequency (HF) component occurs at the frequency of adult respiration, .15–.40 Hz and primarily reflects cardiac parasympathetic influence due to respiratory sinus arrhythmia. The low-frequency (LF) component occurs within .04–.15 Hz. Controversy exists over whether LF HRV is a direct marker of SNS activity, is a product of both SNS and PNS activity (Malliani, Pagani, Lombardi, & Cerutti, 1991; Pumprla et al., 2002; Task Force, 1996), or is mostly parasympathetically mediated (Eckberg, 1997). Owing to the concern that the LF component is contaminated by parasympathetic influence, many researchers have reported the ratio of LF to HF power as an index of “sympathovagal balance” (Task Force, 1996; but also see Eckberg, 1997). As the SNS and PNS operate antagonistically in their control of the heart, this ratio represents relative shifts or biases toward sympathetic or parasympathetic dominance over cardiac function (Pumprla et al., 2002). Statistical filters, such as Porges’s (1986) moving polynomial algorithm and Grossman’s peak-valley statistic (Grossman, van Beek, & Wientjes, 1990), have been developed that remove influences on HRV

that are extraneous to respiratory sinus arrhythmia.

### Psychophysiological Theories of HRV

Two major theories causally relate the autonomic flexibility represented by HRV with regulated emotional responding. Porges’s (1997, 2001) polyvagal theory is based within an evolutionary framework, which understands aspects of human functioning in terms of acquired, genetically based characteristics that are presumed to have aided in survival and/or reproduction throughout human phylogenetic history. Specifically, the polyvagal theory posits that the human ANS evolved in three stages, each characterized by the acquisition of an autonomic structure that plays a unique role in social processes. First acquired was the dorsal vagal complex, a slow-responding, unmyelinated vagus nerve that supports simple immobilization (e.g., freezing) in response to threat. This “vegetative vagus” slows heart rate through tonic inhibition of sinoatrial node activity. The capacity for active mobilization responses (e.g., fight or flight) became supported with the subsequent acquisition of the SNS. Most recently acquired was the ventral vagal

complex, consisting of a fast-acting, myelinated vagus that can rapidly withdraw and reinstate its inhibitory influence on sinoatrial node activity. A key premise of the polyvagal theory is that the ventral vagal complex has afferent fibers terminating in the nuclei of the facial and trigeminal nerves and includes portions of cranial nerves that mediate facial expression, head turning, vocalization, listening, and other socially relevant behaviors. The connection of the ventral vagal complex and these cranial nerves provides a mechanism by which cardiac states can be coordinated with social behaviors (Porges, 1997, 2001).

The polyvagal theory states that the ability of the ventral vagal complex to rapidly withdraw its inhibitory influence allows humans to rapidly engage and disengage with their environment without the metabolic cost of activating the slower responding SNS. The dynamic nature of many social processes (e.g., nonverbal communication, romantic courtship; see Porges, 1998) requires this rapid management of metabolic resources. Only when ventral vagal complex withdrawal is insufficient to meet demands are other autonomic subsystems enlisted. In this respect, the polyvagal theory emphasizes the relation of respiratory sinus arrhythmia (which purportedly indexes ventral vagal complex activity; Porges, 2001) and the regulation of the emotional processes underlying social behavior.

Thayer and colleagues (Thayer & Lane, 2000) have outlined the model of neurovisceral integration, which relates emotional responding with HRV through a dynamical systems perspective. Within the dynamical systems framework, large-scale patterns (e.g., fluctuations in bird populations) are considered to be products of a number of smaller scale interacting forces (e.g., food supply, presence of predators) that influence one another in nonlinear fashions (see Gleick, 1987). According to the model of neurovisceral integration, the array of behavioral, cognitive, and physiological processes involved in emotion are considered to be subsystems of a larger, self-organizing system. Specific emotion states emerge from interactions among these lower level elements along the lines of certain control parameters. The model of neurovisceral integration cites a growing body of literature suggesting the dimensions of valence (aversive vs. appetitive) and arousal represent the control

parameters that guide the organization of emotional responses.

More important, the model of neurovisceral integration views the CAN as the neurophysiological command center governing cognitive, behavioral, and physiological elements into regulated emotion states (Hagemann, Waldstein, & Thayer, 2003; Thayer & Lane, 2000). The CAN does this by inhibiting other potential responses. This inhibition requires reciprocal communication among system components (e.g., feedback loops), sensitivity to the initial conditions of the system, and the existence of multiple pathways to a response (e.g., combinations of sympathetic and parasympathetic activity), all elements of a neuroviscerally integrated dynamical system (Thayer & Seigle, 2002). Such inhibition is thought to be mediated synaptically in the brain and vagally in the periphery (Thayer & Friedman, 2002). From this perspective, HRV can be considered a proxy for the CAN's ability to regulate the timing and magnitude of an emotional response through inhibition in accordance with contextual factors.

Both theories presented above are similar in that they (a) specify a critical role for parasympathetically mediated inhibition of autonomic arousal in emotional expression and regulation and (b) maintain that HRV measures are informative about individuals' capacity for this aspect of regulated emotional responding. However, some differences between the theories do exist. For example, the model of neurovisceral integration is premised on neuroanatomical links between the ANS and brain regions associated with emotional processing (e.g., cortical and limbic areas of the CAN), whereas the polyvagal theory largely rests on the neural connections between the vagus nerve and other cranial nerves that control peripheral structures involved in the behavioral expression of emotion (e.g., the muscles of the face and head). Attending to different neuroanatomical substrates has led to divergent extensions of each theory, mostly to affective dysfunction for the model of neurovisceral integration and to social and developmental processes for the polyvagal theory. Integrating our understanding of the neuroanatomical components from both models should help researchers generate more intricate hypotheses about the interaction of autonomic, cognitive, and behavioral aspects of emotional expression and regulation.

In addition to differing in their neuroanatomical emphases, the model of neurovisceral integration and the polyvagal theory are also based in different theoretical frameworks. The evolutionary perspective within which the polyvagal theory is presented concerns itself with recognizing biologically based adaptations to challenges that have recurred throughout human history. On the other hand, the dynamical systems perspective that characterizes the model of neurovisceral integration aims to uncover broad-based rules that produce emerging patterns within various systems. However, it appears that the dynamical systems and evolutionary frameworks are complementary rather than contradictory, and the evolutionary process itself can be considered a dynamical system (Kenrick, 2001). A synthesis between these perspectives might provide a more complete understanding of the role of HRV in emotional responding.

### Empirical Research With HRV

Although interest in cardiac rhythms may have existed as early as the first half of the 18th century, academic interest in HRV did not emerge until the 1960s (Berntson et al., 1997). Since then, medical researchers have established diminished HRV as an independent risk factor for several negative cardiovascular outcomes (Curtis & O'Keefe, 2002) and as a proxy for underlying cardiovascular disease processes (Stys & Stys, 1998). A growing body of psychological research supports an association between HRV (particularly parasympathetically mediated HRV) and various measures and outcomes relevant to regulated emotional responding. One such outcome is coping, a construct highly intertwined with emotion regulation. *Coping* refers to a set of regulatory strategies that are motivated by emotions (often negative emotions) and that frequently serve an emotion regulatory function but generally involve either nonemotional actions or nonemotional goals, or both (Gross, 1998). Higher levels of resting respiratory sinus arrhythmia have been associated with greater self-reported emotion regulation and the use of constructive coping strategies in university students (Fabes & Eisenberg, 1997). This relation between resting respiratory sinus arrhythmia and constructive coping was mediated by negative emotional arousal. Fabes

and Eisenberg concluded that those with lower resting respiratory sinus arrhythmia experienced greater negative emotional arousal in response to stress, which interfered with their ability to implement adaptive coping strategies. Similarly, higher resting HRV was associated with reduced indices of distress in grade school children watching an upsetting film (Fabes, Eisenberg, & Eisenbud, 1993) and higher social competence in young children (Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994). Although a few studies have associated higher resting respiratory sinus arrhythmia with lower ratings of social competence (Eisenberg et al., 1995, 1996), results have generally supported the premise that higher HRV reflects a greater capacity for regulated emotional responses.

Other evidence has supported a relation between adaptive coping strategies and HRV. Recently bereaved individuals with higher resting respiratory sinus arrhythmia scored higher on measures of active coping and acceptance and lower on measures of passive coping (O'Connor, Allen, & Kaszniak, 2002). Female graduate students classified as repressive copers demonstrated lower resting LF and HF HRV near the time of a major examination than women classified as low anxious (Fuller, 1992), and women with lower parasympathetically mediated HRV (RMSSD) during experimentally induced fear states reported greater use of defensive coping (Pauls & Stemmler, 2003). Finally, those who exhibited submissive behavior during an interpersonal stressor had lower HRV (SDNN and RMSSD) at rest and during the task (Sgoifo et al., 2003).

It would be expected that attention allocation, which can be considered both an emotion regulation strategy (e.g., attentional deployment; Gross, 1998) and a cognitive component of emotion (e.g., fearful vigilance), is associated with HRV. Insofar as attention allocation is involved in selecting meaningful information from the sensory environment, it is linked with the emotional responses to that information. Both the polyvagal theory (Porges, 1992) and the model of neurovisceral integration (Thayer & Lane, 2000) suggest that strong vagal regulation of the heart provides the capacity to quickly adjust cardiac parasympathetic influence to foster attentional engagement or disengagement with the sensory environment. Supporting this notion is the finding that dental-

phobic persons with higher parasympathetically mediated HRV throughout the experiment were better able to inhibit attentional processing of the linguistic content of dental-related words in a modified version of the Stroop task (Johnsen et al., 2003).

The model of neurovisceral integration considers anxiety disorders, such as generalized anxiety disorder and panic disorder, to result from a breakdown in the inhibitory processes of the CAN. CAN disinhibition is thought to account for the continual state of excessive worry and behavioral avoidance that characterizes generalized anxiety disorder and the potential for seemingly uncued panic attacks found in panic disorder (Friedman & Thayer, 1998b). This disinhibition would also be expected to produce reduced parasympathetically mediated HRV and attentional vigilance toward threat. Biases in attentional processing toward threat-related stimuli have also been observed in panic disorder and generalized anxiety disorder (Bradley, Mogg, White, Groom, & de Bono, 1999; Mathews, Ridgeway, & Williamson, 1996). As predicted by the model, patients with generalized anxiety disorder have shown lower parasympathetically mediated HRV relative to controls during rest and during intense worry (Thayer, Friedman, & Borkovec, 1996). Lower overall and parasympathetically mediated HRV (aggregated across several tasks) have been observed in nonclinical panickers and blood phobics relative to controls (Friedman & Thayer, 1998a). Other manifestations of anxiety, such as trait anxiety (Fuller, 1992), social anxiety (Mezzacappa et al., 1997), and self-perceived stress-induced anxiety (Sgoifo et al., 2003) have been associated with reduced resting parasympathetically mediated or overall HRV, suggesting the possibility that diminished autonomic flexibility may be an underlying causal factor.

As with anxiety, it would be expected that diminished HRV would accompany depressive states given that a core feature of depression is the inability to generate appropriate positive and negative emotions (Rottenberg, Kasch, Gross, & Gotlib, 2002). Consistent with this view, bereaved individuals (O'Connor, Allen, & Kaszniak, 2002) and patients being treated for melancholic major depression with amitriptyline (Rechlin, Weis, Spitzer, & Kaschka, 1994) exhibited diminished resting parasympathetically mediated HRV, and patients with bi-

polar depression showed reduced overall resting HRV (Cohen et al., 2003). One study found decreased resting parasympathetically mediated HRV in depressed men, but increased HRV in depressed women (Thayer, Smith, Rossy, Sollers, & Friedman, 1998). An inability to generate well-regulated autonomic responses to stress has been observed in depression, as those reporting greater depressive symptoms exhibited larger decreases from baseline in parasympathetically mediated HRV during a stressful speech task and smaller increases in parasympathetically mediated HRV during a cold pressor task (Hughes & Stoney, 2000), indicating a lessened capacity to regulate cardiac activity to meet the task demands. Resting overall and parasympathetically mediated HRV have interestingly been shown to increase with successful treatment of depression (Balogh, Fitzpatrick, Hendricks, & Paige, 1993), suggesting that resting HRV is related to within-person variation in regulated emotional responding over time. Finally, there is accumulating evidence that suggests reduced parasympathetic control of the heart (as indexed by HRV) may partially mediate the well-documented link between depression and heart disease (Grippo & Johnson, 2002; Sheffield et al., 1998).

### Summary and Future Directions

HRV is emerging as an objective measure of individual differences in regulated emotional responding, particularly as it relates to social processes and mental health. HRV can be derived through a number of statistical, geometrical, and frequency-based analyses that provide information about sympathetic, parasympathetic, and/or overall autonomic regulation of the heart. Two theoretical models hypothesize that the ability of the nervous system to track changes in the environment and respond with physiological arousal that is both proportionate to the context in which it occurs and well-integrated with behavior and cognition is critical to emotional expression and regulation. The polyvagal theory posits that evolutionary forces led to the acquisition of a rapidly responding vagus nerve that supports emotional expression and regulation through its neural connections with peripheral structures involved with social functioning. The model of neurovisceral integration equates the CAN to a dynamical system

that organizes changes in the physiological, cognitive, and behavioral subsystems from which emotion states emerge along the dimensions of valence and arousal.

These theories have generated studies linking greater HRV to the use of adaptive emotion regulation and coping strategies and reduced HRV with various outcomes indicative of emotional dysregulation, such as anxiety, depression, and rigid attentional processing of threat. Findings generally support the predictions of the polyvagal theory and the model of neurovisceral integration, but future research is needed to establish that the relation of HRV to regulated emotional responding is independent of other factors, such as intelligence or personality. Further investigation should attempt to elucidate those contexts in which the autonomic flexibility represented by greater HRV is particularly adaptive, as well as situations in which greater HRV may be maladaptive. Although theorists and researchers have emphasized the importance of parasympathetically mediated HRV in regulated emotional responding, the relative contribution of sympathetic regulation of the heart has not yet been clarified. It would be worthwhile to identify those contexts in which the two branches of the ANS differentially contribute to emotional responding. Finally, research assessing the role of HRV in the regulation of positive emotions is generally lacking and would be beneficial in defining the relation of HRV to emotion regulation.

HRV analysis may be applicable to numerous areas of investigation. For example, a new view of pain as an emotion that motivates homeostatic behaviors (Craig, 2003) suggests that individual differences in emotional responding (as assessed via HRV) may account for variation in pain sensitivity and the ability to regulate pain through various strategies (e.g., attention allocation). HRV can also be assessed through the use of ambulatory ECG monitors, allowing researchers to assess changes in both sympathetic and parasympathetic activity throughout the course of emotionally charged episodes (e.g., interpersonal conflict) in naturalistic settings. Finally, it is unknown whether HRV is influenced by early environmental influences during periods of greater neural plasticity. HRV analysis might help elucidate a neurophysiological pathway linking early family experiences with documented alterations in autonomic re-

sponding in adulthood (Luecken, Rodriguez, & Appelhans, 2005). Potential clinical applications of HRV also exist, as increasing an individual's capacity for inhibitory emotion regulation through HRV biofeedback (Lehrer et al., 2003) may have therapeutic implications for mood, anxiety, and impulse control disorders.

## Conclusions

Research and theory support the utility of HRV as a noninvasive, objective index of the brain's ability to organize regulated emotional responses through the ANS and as a marker of individual differences in emotion regulatory capacity. Unlike other psychophysiological variables, HRV provides information regarding both PNS and SNS activity, thereby permitting inferences about both inhibitory and excitatory processes in emotion regulation. As regulated emotional responding plays a vital role in social processes and mental health, researchers have much to gain by incorporating HRV into their research designs. HRV analysis also has great potential to illuminate the role of individual differences in emotion regulation in other aspects of human functioning. It is hoped and anticipated that only a fraction of the utility of HRV for understanding emotional responding has thus far been realized, which is a truly an exciting prospect for social scientists.

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Received May 8, 2005

Revision received June 20, 2005

Accepted September 15, 2005 ■

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