

Individual differences in the autonomic origins of heart rate reactivity: The psychometrics of respiratory sinus arrhythmia and preejection period

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Abstract

Heart rate reactivity has been conceptualized, at least implicitly, as a unidimensional construct ranging from low to high, reflecting individual differences in adrenergic reactivity to daily stressors. However, an individual's classification as high in heart rate reactivity ignores possible individual differences in the autonomic origins of this reactivity. Sixty-eight women were exposed to orthostatic and speech stressors to determine the psychometric properties (postural stability, convergent and discriminant validity) of heart rate, preejection period, and respiratory sinus arrhythmia. Results revealed that (a) basal, stress, simple reactivity (stress – baseline), and residualized change indices of heart rate, preejection period, and respiratory sinus arrhythmia were stable across postures and (b) heart rate reactivity was significantly related to preejection period and respiratory sinus arrhythmia reactivity, whereas the latter two measures were unrelated. Reactivity classifications may therefore be significantly improved by attention to concurrent estimates of the activity of both autonomic branches.

Descriptors: Heart rate reactivity, Individual differences, Psychological stress, Posture, Impedance cardiography, Respiratory sinus arrhythmia

Heart rate reactivity to brief psychological stressors has been found to (a) vary as a function of age, genetic, dietary, task, and social factors; (b) reflect cardiac adjustments that exceed metabolic demands; and (c) predict risk for cardiovascular disease (e.g., Matthews et al., 1986; Turner, 1989). Heart rate reactivity, however, has tended to be treated as a unidimensional (and occasionally as a unidirectional) construct ranging from low to high, reflecting individual differences in adrenergic reactivity to daily stressors and behavioral challenges. Although adrenergic activity exerts predominant control over the vasculature and cardiac inotropy, cardiac chronotropy is a joint function of sympathetic and vagal activity. Moreover, psychological stressors are now known to affect vagal as well as sympathetic outflows to the heart (e.g., Allen & Crowell, 1989; Grossman, Stemmler, & Meinhardt, 1990; Porges, 1992), and these outflows can vary independently (Berntson, Cacioppo, & Quigley, 1991).

An individual's classification as high in heart rate reactivity ignores possible individual differences in the autonomic origins

of this reactivity. An individual's classification as high in heart rate reactivity could originate in elevated sympathetic reactivity, vagal withdrawal, or reciprocal activation of the sympathetic and vagal outflows to the heart. Similarly, an individual's classification as low in heart rate reactivity could stem from low sympathetic (and vagal) reactivity or from low to high coactivation of the sympathetic and vagal controls on cardiac chronotropy. Although psychophysiolgists have long recognized these issues (e.g., Pollak & Obrist, 1988), variations in the autonomic origins of heart rate reactivity generally have been relegated to the error term, a practice that may obscure the relationship between autonomic responses to stressors and behavioral, humoral, or clinical outcomes. Quantifying these individual differences requires replacing the conceptualization of heart rate reactivity as a unidimensional (e.g., sympathetic activation) vector with a bivariate autonomic space (i.e., low to high sympathetic activation and low to high vagal activation). We recently outlined such a bivariate autonomic space (Berntson et al., 1991) and reviewed the evidence consistent with the notion that heart rate reactivity can derive from multiple modes of autonomic control (Berntson, Cacioppo, & Quigley, 1993b). There is a paucity of research in the contemporary literature, however, on individual differences in the autonomic origins of heart rate reactivity. Our goals in the present research were to (a) employ a postural manipulation with known autonomic consequences to investigate its effects on respiratory sinus arrhythmia (RSA) and preejection period (PEP) and examine reactivity to a speech

This research was supported by National Science Foundation grant DBS-9211483.

We thank Sandra Sgoutas-Emch and Karen Quigley for assistance in data collection, Bob Kelsey and David Lozano for technical advice, and Stephen W. Porges and Evan Byrne for helpful suggestions.

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stressor using indices based on heart rate (HR), RSA, and PEP; (b) investigate the reliability and postural stability of basal, stress, simple reactivity (stress – baseline), and residualized change indices of HR, RSA, and PEP; and (c) examine the convergent and discriminant validity of RSA and PEP reactivity measures as noninvasive indices of the vagal and sympathetic determinants, respectively, of stress-induced HR reactivity.

The measures of RSA and PEP were selected because they represent two of the most promising noninvasive measures of the autonomic control of the heart currently available. The high frequency (i.e., RSA: 0.12–0.40 Hz) component of the oscillations of heart periods provides a marker of vagal control as long as significant variations in respiratory activity are controlled or accounted for (Berntson, Cacioppo, & Quigley, 1993a; Grossman, Karemaker, & Wieling, 1991). For instance, RSA increases with increasing vagal control and is blocked by cholinergic but not by adrenergic blockade (Berntson et al., 1993a). The PEP, on the other hand, is inversely related to sympathetic inotropy (Binkley & Boudoulas, 1986). Shortenings in PEP accompany increases in HR due to adrenergic cardiostimulation but not due to vagal blockade or atrial pacing (e.g., Harris, Schoenfeld, & Weissler, 1967). Studies further suggest that HR per se does not influence the PEP unless changes in HR are associated with inotropic changes or are accompanied by changes in preload or afterload (Lewis, Leighton, Forester, & Weissler, 1974). Harris et al. (1967) reported that infusions of isoproterenol, epinephrine, and norepinephrine are associated with a shortening of PEP, although the effects of catecholamines on the duration of PEP can be complex because of their alpha- and beta-stimulating properties (Lewis et al., 1974). McCubbin, Richardson, Langer, Kizer, and Obrist (1983) found that PEP was negatively correlated with plasma levels of norepinephrine following stressful cognitive tasks (Raven matrices), suggesting that the PEP may provide a useful marker of adrenergic cardiac activation in response to acute psychological stress.

Postural changes are associated with well-defined alterations of vagal and sympathetic outflows to the heart (Berntson et al., 1993b). Standing tends to pool blood in the lower half of the body, resulting in a decrease in filling pressure in the heart, in end-diastolic and end-systolic volume, and in stroke volume (Langou, Wolfson, Olson, & Cohen, 1977). Increases in tonic sympathetic activation of the heart and elevated plasma catecholamines follow (Reid & Dollery, 1978), as do significant decreases in tonic vagal activation of the heart (Hatch, Klatt, Porges, Schroeder-Jasheway, & Supik, 1986). These adjustments are not limited to phasic reactions; many of these adjustments are manifest after prolonged (60 min) standing (Boudoulas, Barrington, Olson, Bashore, & Wooley, 1985). Although RSA decreases with the vagal withdrawal associated with standing (Hatch et al., 1986), PEP increases, despite the increased sympathetic outflows to the heart (Sherwood & Turner, 1993), because of the overriding effects of preload and afterload (e.g., see review by Binkley & Boudoulas, 1986). Variations in PEP appear to reflect the sympathetic control of the heart when posture is constant, however. Therefore, we examined the psychometrics of PEP (e.g., reliability, correspondence to HR) and RSA within and across posture.

Prior research on speech stressors has found that the evaluative speaking task elevates HR (e.g., Saab, Matthews, Stoney, & McDonald, 1989), but its effect on PEP has not been as reliable (e.g., Saab et al., 1992) nor has its effect on RSA been examined. The speech task is an appealing experimental stres-

sor, in part because it has ecological validity. The notion is that the more similar laboratory stressors are to the acute psychological stressors individuals encounter in the course of their daily lives, the more generalizable and predictive the classification of these individuals will be in terms of their cardiac reactivity based on laboratory protocols. However, the speech stressors administered in the laboratory have nearly uniformly tested subjects while seated, a condition that may not be representative of many routine daily activities that involve evaluated speaking. Given the differences in the tonic autonomic control of the heart across posture, it cannot be assumed that brief psychological stressors have the same impact on HR or that HR responses have the same autonomic origins across postures.

Varying posture, therefore, provides a means of examining the reproducibility of the reactivity indices of HR, RSA, and PEP at two different levels of autonomic activation of the heart. Accordingly, our second aim was to investigate the reliability of stress-induced changes in HR, RSA, and PEP reactivity indices across posture. Respiration was also monitored to examine and control for the specificity of the stress-induced changes in RSA. Prior research has established the reliability of HR and HR reactivity when multiple measurements are used and subjects are seated (e.g., Kamark et al., 1992; Sherwood, Dolan, & Light, 1990). To investigate their reliability across two different levels of tonic autonomic control of the heart, we correlated basal HR across posture, task HR across posture, and HR reactivity across posture. Similar analyses were conducted for measures based on RSA and PEP.

The final aim of this study was to examine RSA and PEP reactivity measures as noninvasive indices of the vagal and sympathetic determinants, respectively, of stress-induced HR reactivity. A general increase in the adrenergic activation of the heart is thought to abbreviate PEP and elevate HR, whereas decreased vagal activation of the heart is thought to decrease RSA and increase HR. Therefore, intercorrelations among simple reactivity (stress – baseline) scores and among residualized change scores were examined to determine whether (a) PEP reactivity was correlated negatively with HR reactivity, (b) RSA reactivity was correlated negatively with HR reactivity, and (c) PEP and RSA reactivity were weakly or nonsignificantly correlated.

Method

Subjects

Sixty-eight healthy undergraduate women, ages 17–30 ($M = 18.8$) years, participated in the present research. Subjects received either experimental credit or \$5.00 for approximately 2 hr of participation. The inclusion criteria for participation were that subjects (a) were in good health; (b) were within 20% of their ideal body weight; (c) had no history of psychological disorder or chronic illness; (d) were not on any prescription medication, nonprescription drugs, or tobacco products; (e) exercised on average less than 10 hr/week; (f) consumed on average less than 10 alcoholic beverages per week; (g) had not experienced any recent negative life event (e.g., death in the family); (h) had no class exam on the day of their participation; and (i) were not math, speech, or needle phobic. Subjects were asked to refrain from ingesting anti-inflammatory agents, antihistamines, or alcohol during the 24 hr preceding the test day. One subject was deleted from analyses because of outlier RSA values (i.e., two log units lower than any other subject), leaving a sample of 67 subjects.

The planned analyses involved primarily psychometric tests to examine the postural stability and intercorrelations among HR, RSA, and PEP reactivity indices (e.g., simple change scores, residualized change scores). When a large number of psychometric tests are planned, as in the present research, cross-validation rather than Bonferroni corrections is often preferable. Cross-validation was achieved in the present research by calculating all correlations and reliability coefficients separately for two replications of the study, which differed only in the order in which posture was manipulated. The sample size in Replication 1 was 34 and in Replication 2 was 33. Only psychometric tests that are cross-validated (i.e., statistically significant in both replications) should be viewed as replicable. Median correlations within replication, for instance, were used to assess the convergent and discriminant validity of the RSA, PEP, and HR reactivity indices; median correlations that were statistically significant in both replications represent cross-validated estimates of the relationships among these reactivity indices.

Procedure

Following a description of the study and tasks, subjects signed an informed consent document and spent approximately 20 min in the laboratory completing an innocuous set of questionnaires. Among these scales was a health/lifestyle questionnaire that assessed their exercise habits, caffeine consumption, family history of hypertension, and number and nature of major negative life events during the last year. Following completion of these surveys, the cardiovascular recording sites were cleaned with alcohol and electrodes were attached.

Once stable recording levels were obtained, cardiovascular and respiratory measures were obtained over 2-min baseline periods while the subjects were seated and while they were standing. The order of postural testing was counterbalanced across subjects, and 30 s were allowed after the assumption of a given posture before baseline measures were initiated.

After baseline testing, a speech stressor was introduced (Saab et al., 1989). Subjects were seated and were asked to imagine that they were in a department store shopping when a security guard falsely accused them of shoplifting. Subjects were instructed to prepare a 4-min speech to (a) tell their side of the story, (b) tell the manager what the security guard did wrong and why the security guard may have suspected them of shoplifting, (c) say how they can prove they did not steal the item, (d) specify what should happen to the security guard for the mistake, and (e) summarize their points. Subjects were instructed to give intelligent and well-thought out answers because their speech would be recorded and compared with the speeches of others. Subjects were given 4 min to prepare and 4–5 min to present their speeches, with recordings obtained only during the latter period. Approximately half (2 min) of the speech was delivered while seated, and approximately half (2 min) of the speech was delivered while standing. The same counterbalance order for postural testing used during baseline were used during the presentation of the speech. Subjects assumed the initial posture (sitting or standing) during the final 30 s of the speech preparation. Recordings were not initiated, however, until subjects began their speech. After speaking for 2 min, the recordings were paused surreptitiously, and subjects were instructed to change posture and to continue their speech. Subjects assumed the alternative posture (standing or sitting) and continued speaking. Thirty seconds later, the recordings were again surreptitiously initiated and continued for another 2 min, at which point recordings were

stopped and subjects were instructed that they had done well and could stop. Afterwards, subjects were debriefed, thanked, and dismissed.

Measures

A Minnesota Impedance Cardiograph (Model 304B) was used to measure electrocardiogram (EKG), basal thoracic impedance (Z_0), and the first derivative of the impedance signal (dZ/dt). Disposable EKG spot electrodes were placed in the tetrapolar configuration (Sherwood, Royal, Hutcheson, & Turner, 1992). Although band electrodes provide more accurate magnitude measures of cardiac output values, PEP can be measured with equal accuracy using band or spot electrodes (Sherwood et al., 1992). The two outer (current) electrodes were placed over the fourth cervical vertebra and the ninth thoracic vertebra, whereas the two inner (recording) electrodes were placed 4 cm above the clavicle, and over the sternum at the fourth rib. A 4-mA AC current at 100 kHz was passed through the two outer electrodes, and Z_0 and dZ/dt were recorded from the two inner electrodes. The EKG, Z_0 , and dZ/dt signals were digitized at 500 Hz, and interbeat intervals were derived from a custom software package.¹ The impedance data were ensemble averaged within 1-min epochs, and each waveform was verified or edited prior to analyses. The PEP was quantified as the time interval in milliseconds from the onset of the EKG Q-wave to the B-point of the dZ/dt wave. Mean PEP was calculated for each minute for each subject. These minute-by-minute means were averaged over minutes within posture and period prior to the analysis of variance and hierarchical regression to increase reliability.

The interbeat intervals were checked and edited for artifacts using the detection algorithm of Berntson, Quigley, Jang, and Boysen (1990). The interbeat intervals were subsequently verified by visual inspection. Mean HR was calculated for each minute for each subject. In addition, the interbeat interval data were converted to a time-series of successive 500-ms samples. The magnitude of RSA was extracted as a noninvasive index of cardiac vagal activity using a PC-based software package (MXedit 2.01, Delta-Biometrics, Bethesda, MD). As suggested by Porges and Bohrer (1990), the complex trend in the heart period time series was removed with a 21-point cubic digital filter that was moved stepwise through the data. The resulting smoothed time series was subtracted from the original time series to generate a residual series. The natural logarithm of the variance of the heart period pattern within the frequency bandpass associated with respiration (i.e., 0.12–0.40 Hz) was calculated for the estimate of vagal cardiac activity. Mean RSA activity was calculated across each 2-min period within posture for each subject.

Respiration was recorded on a Grass Model 7D polygraph using a Grass strain gauge placed below the lowest current electrode. The analog signal was quantified at 250 Hz, and the digital data were subjected to a 10-point boxcar filter. Our recording and filtering procedures were developed in pilot testing on the speech stressor. The respiration data were verified or edited to eliminate artifacts, and the mean respiration rate,

¹We thank Robert Kelsey and William Guethlein for providing us with copies of their data acquisition and reduction software for impedance cardiography and for their helpful advice. The analyses reported in the text were also performed on heart period and on pre-ejection period corrected for heart rate using the formula recommended by Binkley and Boudoulas (1986). The pattern of results paralleled those reported in the text.

respiration amplitude, and I-fraction were calculated for each minute for each subject. These minute-by-minute means were averaged within posture and period prior to analyses of variance and hierarchical regressions to increase their reliabilities.

Results

Effects of Posture and Period

To determine the general effects of posture and speech stressor on cardiovascular and respiratory activity, we performed 2 (posture: sitting vs. standing) \times 2 (period: baseline vs. task) analyses of variance. The results are summarized in Table 1. Consistent with prior research, significant main effects for posture confirmed that standing was associated with higher HR, lower RSA, and longer PEP.

Main effects for period indicated that the speech stressor elevated HR and shortened PEP. This pattern of results might lead one to attribute the effect of the speech stressor on HR to increased sympathetic cardiac activity, particularly in light of the nonsignificant main effect for period on RSA. However, significant Period \times Posture interactions were observed for HR and RSA, and the form of these interactions suggest that it may be an error to characterize the speech stressor simply as a beta-adrenergic stressor. First, HR reactivity was significantly larger when subjects were seated than when they were standing (see Table 1). Second, the speech stressor had very different effects on RSA in these two postures: The stressor was associated with diminished RSA when subjects were sitting but was associated with an increase in RSA when subjects were standing ($ps < .05$). As expected, basal RSA levels were reduced when subjects were standing, in contrast to when they were sitting. The increase in RSA in response to the speech stressor when subjects were standing, however, was unexpected. Although modest, the increase in RSA was statistically significant, making it unlikely that the effects of the stressor on RSA reactivity when subjects were standing reflects the operation of a floor effect.

An alternative interpretation for the RSA changes is that they reflect changes in respiratory amplitude or period. As illustrated in Table 1, analyses of the measures of respiration revealed

(a) the expected main effect for period for the measure of respiration period, reflecting a lengthening of the respiration cycle during the speech; (b) the expected main effect for period for the measure of I-fraction, indicating that the lengthening of the respiratory cycle was due primarily to an increase in expiration when speaking; and (c) Posture \times Period interactions for the measures of respiration amplitude and respiration period. Respiration amplitude decreased during the stressor when subjects were seated but did not change significantly when subjects were standing, and respiration period increased more during the stressor when subjects were standing than when they were sitting (see Table 1). However, these respiratory differences were quite small. To determine whether these respiratory changes were underlying the stress-induced RSA changes, we conducted a hierarchical regression analysis of RSA. Independent variables in their entry order and their associated statistics were (a) basal respiration amplitude (beta = $-.01$, n.s.); (b) task respiration amplitude (beta = $.62$, $p < .05$); (c) basal respiration period (beta = $.26$, $p < .01$); (d) task respiration period (beta = $.08$, n.s.); (e) basal respiration I-fraction (beta = $.05$, n.s.); (f) task respiration I-fraction (beta = $-.11$, n.s.); (g) posture (beta = $-.31$, $p < .01$); (h) period (beta = $-.06$, n.s.); and (i) Posture \times Period (beta = $.39$, $p < .01$). In sum, the significant effects found for respiration amplitude and period on RSA are consistent with prior research, but the main effect of posture and the interactive effect of posture and speech stressor on RSA remained statistically significant even after extracting the variance in RSA attributable to respiration using hierarchical regression.

Reliability of Cardiac Control Indices Across Postures and Periods

The results suggest that the speech stressor was associated with reciprocal sympathetic activation and vagal withdrawal during sitting and with coactivation (primarily sympathetic activation) during standing. Because these modes of autonomic control of HR reactivity to the speech stressor differed across posture, the reliability of HR reactivity across posture was investigated to determine its stability as an index of individual differences in

Table 1. Mean (SEM) Cardiovascular and Respiratory Activity as a Function of Posture and Period

Measure ^a	Sitting		Standing		F ratios		
	Base	Task	Base	Task	Period	Posture	Period \times Posture
HR	74.27 (1.32)	91.79 (1.90)	86.71 (1.26)	96.57 (1.76)	131.66**	121.02**	24.96**
RSA	7.05 (0.12)	6.43 (0.15)	5.88 (0.11)	6.24 (0.12)	1.27	84.06**	55.79**
PEP (ms)	84.27 (1.27)	74.09 (2.06)	92.91 (1.39)	83.03 (1.92)	47.73**	59.14**	<1
Respiration amplitude	5.90 (0.08)	5.77 (0.09)	5.87 (0.08)	5.84 (0.09)	2.40	<1	5.00*
Respiration period (s)	3.74 (0.09)	4.46 (0.11)	3.68 (0.08)	4.68 (0.10)	57.08**	1.10	5.93*
Respiration I-fraction	.40 (.004)	.33 (.005)	.40 (.004)	.32 (.005)	173.28**	2.07	2.52

^aHR = heart rate; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period.
* $p < .05$. ** $p < .01$.

Table 2. Pearson Correlations Across Posture

Measure ^a	Replication 1 (n = 34)	Replication 2 (n = 33)
Basal HR	.82**	.81**
Task HR	.91**	.88**
HR Δ	.82**	.63**
HR Res. Δ	.83**	.64**
Basal RSA	.64**	.58**
Task RSA	.87**	.74**
RSA Δ	.53*	.52*
RSA Res. Δ	.72*	.62*
Basal PEP	.74**	.85**
Task PEP	.82**	.64*
PEP Δ	.64*	.70**
PEP Res. Δ	.73**	.65**

^aHR = heart rate; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; Δ = change scores; Res. Δ = residual change scores. * $p < .01$. ** $p < .001$.

cardiac reactivity. Basal HR, task HR, and HR reactivity (calculated as a simple change score and as a residualized change score) during sitting were highly predictive of the corresponding measure during standing (Table 2). These reliability coefficients are adequate to excellent from a psychometric perspective, and the statistical tests confirmed that these reliability estimates were replicable (i.e., cross-validated). We also computed the Spearman correlation for each measure to determine the stability of the rank orderings across posture. In every instance, the Spearman coefficient was statistically significant at the $p < .01$ level.

We conducted comparable analyses for basal RSA, task RSA, and RSA reactivity and for basal PEP, task PEP, and PEP reactivity. The results closely paralleled those for HR. High and statistically significant correlations across posture were found for basal RSA, task RSA, and RSA reactivity measures and for basal PEP, task PEP, and PEP reactivity measures (Table 2). Spearman correlations produced the same pattern of results (all $ps < .01$). Thus, RSA and PEP level and RSA and PEP reactivity during standing met psychometric as well as cross-validation thresholds for reliability.

Intercorrelations Among Cardiac Reactivity Indices

Our final aim was to determine whether the interrelationships among the reactivity measures were consistent with the use of RSA and PEP reactivity as noninvasive indices of the vagal and sympathetic determinants, respectively, of stress-induced HR reactivity. The intercorrelations among the cardiac reactivity measures for each replication are presented in Table 3. These analyses were performed using simple change scores and residualized change scores. The indices of postural stability for HR, RSA, and PEP reactivity indices are depicted in Table 2. These coefficients are uniformly high, so a low correlation between reactivity indices in Table 3 is unlikely an artifact of low measurement reliability.

The eight coefficients in the upper left corner of each matrix in Table 3 designate the correlations between stress-induced changes in RSA and HR. As expected, these correlations are all negative, reflecting the negative chronotropic effects of vagal input to the heart. That is, individuals who displayed stress-induced increases in RSA also were likely to show small increases in HR, whereas individuals who showed stress-induced decreases

Table 3. Intercorrelations Among Reactivity Change Scores (Residualized Change Scores)

	Sitting RSA	Standing RSA	Sitting PEP	Standing PEP
Replication 1				
Sitting HR	-.69** (-.72**)	-.39* (-.59**)	-.62** (-.66**)	-.69** (-.70**)
Standing HR	-.52** (-.56**)	-.63** (-.74**)	-.52** (-.52**)	-.58** (-.59**)
Sitting RSA			.31 (.31)	.52** (.56**)
Standing RSA			.28 (.29)	.23 (.42*)
Replication 2				
Sitting HR	-.68** (-.68**)	-.31 (-.31)	-.74** (-.72**)	-.54** (-.48**)
Standing HR	-.54** (-.53**)	-.28 (-.28)	-.52** (-.50**)	-.50** (-.45**)
Sitting RSA			.40* (.43*)	.32 (.31)
Standing RSA			.10 (.12)	-.12 (-.14)

Note: HR = heart rate; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period.

* $p < .05$. ** $p < .01$.

in RSA (reflecting vagal withdrawal) also displayed large increases in HR. The relationship between RSA and HR reactivity was not as robust in the upright posture, possibly due to low basal RSA levels in this posture in Replication 2. Nevertheless, the median correlation among these measures was statistically significant ($ps < .05$) in both replications.

The coefficients in the upper right corner of the matrices in Table 3 represent the correlations among stress-induced changes in PEP and HR. Consistent with the notion that stress-induced sympathetic cardiac activation shortens PEP and elevates HR, these correlations were uniformly large and negative. The median correlation among these measures was also statistically significant in each replication.

The coefficients in the lower right corner of each correlation matrix represent the correlations between the RSA and PEP reactivity measures. Consistent with the notion that stress-induced changes in RSA and PEP can vary independently, most of these correlations were nonsignificant and none of these intercorrelations proved replicable. The median correlation among these measures was not significant in either replication. Parallel analyses were performed using partial correlations to statistically control for any effects attributable to respiration. The pattern and levels of statistical significance were unchanged.

Discussion

One major aim of this research was to investigate the use of RSA and PEP reactivity measures as noninvasive indices of the vagal and sympathetic determinants, respectively, of stress-induced HR reactivity. Intercorrelations revealed that (a) PEP reactivity was correlated negatively with HR reactivity, as expected if PEP were to serve as a marker generally of cardiac adrenergic activation; (b) RSA reactivity was correlated negatively with HR

reactivity, as expected if RSA were to mark vagal control of the heart; and (c) PEP and RSA reactivity were not reliably correlated, as would be expected if PEP reactivity were selectively sensitive to changes in the sympathetic outflows and RSA reactivity were selectively sensitive to vagal outflows to the heart. Thus, the intercorrelations of the reactivity indices support the hypothesis that stress-induced changes in RSA and in PEP can vary independently and that each predicts unique autonomic determinants of HR reactivity.

A second major aim was to verify the effects of posture on HR, RSA, and PEP and to examine the additional effects of a speech stressor. Our results replicate prior research showing that standing produced higher HR, lower RSA, and longer PEP than did sitting. The elevation of HR and reduction in RSA by standing replicate the results of Hatch et al. (1986) and support the validity of RSA as an index of the vagal activation of the heart. The effect of posture on the PEP is also consistent with results of prior research (e.g., Boudoulas et al., 1985; Sherwood & Turner, 1993) and underscores the multiply determined nature of the PEP. Although the increased sympathetic outflow to the heart that results from standing abbreviates PEP, this effect is typically overwhelmed by the effects of standing on preload or afterload (Spodick, Meyer, & St. Pierre, 1972). Thus, even though the PEP may provide an index of sympathetic cardiac activation in response to a stressor when posture is constant, caution should be exercised when interpreting PEP as a noninvasive index of sympathetic cardiac activity across postures. Additional research is also needed to determine whether task-induced changes in preload or afterload (e.g., by alpha-adrenergic responses) must also be considered in the utility of PEP as a marker of beta-adrenergic activity.

HR reactivity to brief psychological (e.g., speech) stressors generally exceeds metabolic demands and varies as a function of age, genetic, dietary, task, and social factors (e.g., Kamarck, 1992; Turner, 1989). Furthermore, cardiac reactivity predicts endocrine and immunologic responses to laboratory stressors and risk for cardiovascular health (e.g., see Kiecolt-Glaser, Cacioppo, Malarkey, & Glaser, 1992). This research is typically interpreted as reflecting individual differences in behaviorally evoked sympathetic nervous system activation. A cursory inspection of Table 1 might suggest that our results are consistent with this conceptualization. The speech stressor elevated HR and shortened PEP in our study of undergraduate women. Saab et al. (1992) recently used a similar speech stressor and reported that it had no significant effects on PEP even though HR was elevated; their study, however, involved men rather than women, their sample size was much smaller than ours, and the HR and PEP responses evoked by their speech stressor were substantially smaller than what we observed. Because the speech stressor tends to evoke greater reactivity in women than men (Stoney, Matthews, McDonald, & Johnson, 1988) and our sample size was more than twice as large as that used by Saab et al. (1992), our observation that the speech stressor abbreviated the PEP likely reflects stress-induced increases in beta-adrenergic activity.

Closer inspection of Table 1 indicates that there are serious limitations in the conceptualization of HR reactivity as varying along a single (e.g., beta-adrenergic activation) dimension. Recent research in which HR and RSA have been measured has provided evidence that psychological challenges such as mental arithmetic and a video game can produce vagal withdrawal (e.g., Allen & Crowell, 1989). Subjects in prior research have typically performed tasks while sitting, a posture in which we also found

a significant reduction in RSA in response to the psychological stressor even after covarying out the effects of respiration. Thus, the HR reactivity evoked by the stressor when subjects were sitting appears to have been subserved by both sympathetic activation and vagal withdrawal.

Basal RSA was lower when subjects were standing than when they were sitting. However, the speech stressor evoked an increase rather than a decrease in RSA when subjects performed the task standing. Regression analyses indicated that changes in respiration could not explain away the changes in RSA. The increase in RSA was statistically significant, making it unlikely that the effects of the stressor on RSA reactivity when subjects were standing reflects the operation of a floor effect. In addition, research by Boudoulas et al. (1985) indicates that the effects of posture on the autonomic control of the heart stabilize quickly, so it is also unlikely that the increase in RSA reflects any kind of rebound or delayed adjustment in tonic RSA due to standing. Instead, these data suggest that the speech stressor produced reciprocal sympathetic cardiac activation when subjects were sitting but produced primarily sympathetic activation when subjects were standing. The net effects of these autonomic changes would be to augment any sympathetically mediated elevation in HR in the sitting condition but to mitigate any sympathetically mediated increase in HR in the standing condition.

Vocalizations may compromise the estimation of RSA, rendering it a less valid index of vagal control during the speech than during the baseline. There are several lines of evidence arguing against this possibility, although it cannot be ruled out. For instance, the correlations between basal and task (i.e., speech) RSA were positive and statistically significant for both standing and sitting. Furthermore, RSA decreased when the speech stressor was performed in the sitting posture, consistent with our RSA measure indexing the vagal innervation of the heart during speaking. Analyses also indicated that the RSA reactivity scores were reliable, were significantly and negatively related to HR reactivity (convergent validity), and were nonsignificantly associated with PEP reactivity (discriminant validity). Whether one or more of these speculative accounts proves to be heuristic, the data from this study clearly call into question the conceptualization of HR reactivity as a unidimensional construct ranging from low to high that reflects individual differences in sympathetic reactivity.

A third aim of this research was to investigate the reliability and postural stability of basal, stress, simple reactivity (stress - baseline), and residualized change indices of HR, RSA, and PEP. Although the literature on the psychometrics of HR reactivity is consistent with its use as an individual difference variable, especially when assessments are based on multiple HR measures (Kamarck, 1992), the prior research on the reliability of stress-induced RSA and PEP responses has been less definitive. No psychometric investigation of RSA reactivity has been published. Research on the test-retest reliability of reactivity indices based on the PEP is more extensive, but the variability in these estimates across tasks or studies suggests there may be factors operating within sessions that are important to consider to achieve stable and reliable assessments of individual differences in adrenergic reactivity to acute psychological stressors outside the laboratory. For instance, Saab et al. (1992) calculated generalizability coefficients assessing the 2-week stability of cardiovascular parameters including PEP. Reactivity was calculated based on change scores to a speech stressor (preparation and delivery), mirror tracing, and cold pressor task. The

generalizability coefficients for the reactivity measures for PEP ranged from .23 for the mirror tracing task to .74 for the cold pressor task.

Given the relative dearth of research on RSA reactivity indices and some concern about variability across tasks in the reliability of PEP reactivity indices, we focused in the present research on the reproducibility of the reactivity indices of HR, RSA, and PEP at two different levels of autonomic activation of the heart (i.e., across postures) within a single experimental session. Results revealed that HR, RSA, and PEP reactivity based on subjects' performance when they were sitting significantly predicted the corresponding cardiac reactivity index based on subjects' performance when they were standing. The rank orderings of subjects in terms of their HR reactivity, RSA reactivity, and PEP reactivity were also reproducible across postures, with residualized change indices of RSA reactivity performing somewhat better than simple change scores. Thus, the stability of individual difference classifications based on RSA and PEP reactivity indices appear to be on par with those for HR reactivity.

In sum, recent research has distinguished between individuals who show reactivity primarily in terms of cardiac output from those who show reactivity primarily in terms of total peripheral resistance. However, cardiac output reactivity, like HR reactivity, has been conceptualized as ranging from low to high and reflecting individual differences in beta-adrenergic

reactivity. The importance of determining the autonomic origins of individual differences in HR reactivity is apparent when considering the putative relationship between HR reactivity and changes in catecholamine levels in individuals under stress or disease. HR reactivity is typically thought to be positively related to changes in circulating catecholamines in response to a stressor (McCubbin et al., 1983). For "high" HR reactors, risk factors and catecholamines may vary consistently depending on the autonomic origins of the individual's HR reactivity. Individuals whose HR reactivity is attributable to sympathetic activation, for instance, would be expected to show stronger correlations between HR reactivity and catecholamine levels than individuals whose comparable stress-induced HR response is attributable to vagal withdrawal or to reciprocal sympathetic activation. At present, these individual differences in reactivity are relegated to the error term in unidimensional conceptualizations of HR (or cardiac output) reactivity, a practice that may obscure the relationship between cardiac sympathetic reactivity and humoral (e.g., catecholaminergic) responses, immunologic changes, and health outcomes. Thus, studies of individual differences in cardiac reactivity may be further advanced by expanding the unidimensional concept of sympathetic reactivity to a bivariate autonomic space with separate vagal and sympathetic activation dimensions and by obtaining concurrent estimates of both autonomic branches in research on behaviorally evoked HR reactivity.

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(RECEIVED February 24, 1993; ACCEPTED September 29, 1993)