

# Patterns of sympathetic and parasympathetic reactivity in a sample of children and adolescents

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## Abstract

We hypothesized that patterns of sympathetic and parasympathetic reactivity observed in adults would be apparent in a sample of children and adolescents and that these patterns would be consistent across tasks. We explored the relationship between these patterns and psychosocial risk factors for cardiovascular disease. We measured preejection period (PEP) and an index of respiratory sinus arrhythmia (mean successive difference [MSD] statistic) during three reactivity tasks. We classified participants into four groups based on increases or decreases in PEP and MSD. Ninety percent of the sample exhibited the same pattern during at least two of the tasks. PEP and MSD demonstrated consistency, suggesting individual response stereotypy. Exhibiting a consistent pattern of decreased PEP and increased MSD was associated with less child- and parent-reported family conflict. These results are discussed in the context of vagal regulation of environmental demands.

**Descriptors:** Sympathetic nervous system, Parasympathetic nervous system, Preejection period, Cardiovascular disease, Children and adolescents, Heart rate variability, Blood pressure, Impedance cardiography

Cardiovascular researchers often treat indices of reactivity as separate and independent responses. However, responses of the cardiovascular system occur in concert with one another, and no single index can describe the entire system. Researchers have described reactivity in terms of patterns of cardiac output (CO) and peripheral resistance responses, and the stability of these patterns across tasks and over time (Allen, Obrist, Sherwood, & Crowell, 1987; Kasprowicz, Manuck, Malkoff, & Krantz, 1990). These researchers have focused primarily on mechanisms underlying blood pressure (BP) responses. Their findings suggest that individuals can exhibit similar changes in BP as a result of different underlying cardiac and vascular responses.

Heart rate (HR), like BP, reflects a response determined by underlying mechanisms. Independent sympathetic and parasympathetic innervation of the heart determines the direction and magnitude of HR change. Berntson, Cacioppo, and their colleagues (Berntson et al., 1994; Cacioppo, Uchino, & Berntson, 1994) examined indices of parasympathetic and sympathetic activity as they contribute to HR changes. Specifically, they examined respiratory sinus arrhythmia (RSA) assessed via quantification of HR variability to measure parasympathetic activity. As a measure of sympathetic activity, they examined preejection period (PEP),

an often used, albeit less than perfect index of sympathetic influence on the heart. Lewis, Leighton, Forester, and Weissler (1974) reviewed a number of studies demonstrating the utility of PEP as a sympathetic measure, although it is subject to influence by changes in preload (i.e., venous return) and afterload (i.e., systemic resistance).

Berntson et al. (1994) demonstrated that an orthostatic change from sitting to standing produced a pattern of reciprocal sympathetic activation and parasympathetic withdrawal that characterized a general response. However, they also found considerable individual differences in sympathetic and parasympathetic reactivity to psychological stressors. Although the group means suggested that reaction time, math, and speech stressors produced the reciprocal sympathetic pattern described above, PEP and RSA reactivity were not significantly correlated, indicating individual response stereotypy. Further, PEP and RSA reactivity demonstrated considerable consistency within individuals across tasks, again suggesting individual differences (Berntson et al., 1994).

In a later study, Berntson, Cacioppo, and Fieldstone (1996) examined PEP and RSA responses during mental arithmetic and an illusions task. Again, group means suggested that the mental arithmetic task produced a pattern of sympathetic activation with parasympathetic withdrawal. The illusions task produced a different pattern, however. Overall, participants exhibited parasympathetic activation with a slight sympathetic withdrawal—a pattern not consistent with the idea that mental stress causes sympathetic activation. Individual variability in the degree and pattern of responses for both tasks was present, though PEP and RSA produced significant small to moderate correlations for both tasks. These findings suggest that individual response tendencies may interact

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with task response tendencies to produce a variety of sympathetically and parasympathetically driven HR responses.

Berntson, Cacioppo, and colleagues (Berntson et al., 1994, 1996; Berntson, Cacioppo, & Quigley, 1991, 1993; Cacioppo et al., 1994) proposed a taxonomy for classifying individuals based on their sympathetic and parasympathetic responses during psychological stress. The reciprocal sympathetic pattern is evidenced by sympathetic activation and parasympathetic withdrawal; that is, PEP increases and RSA decreases during stress. A reciprocal parasympathetic pattern involves parasympathetic activation (RSA increases) and sympathetic withdrawal (PEP increases). Coactivation describes sympathetic and parasympathetic activation, whereas coinhibition describes withdrawal of both sympathetic and parasympathetic influence. Functionally, three of these patterns can result in HR increases, considering the degree of change in the two autonomic branches. A reciprocal sympathetic pattern should always result in HR increases. Coactivation and coinhibition could result in HR increases or decreases, considering the degree in change of one branch relative to the other. A reciprocal parasympathetic response should result in HR decreases. Clearly, then, HR alone is an imprecise index of autonomic influences on the heart.

Research examining the stability of PEP and RSA reactivity is sparse. Berntson, Cacioppo, and colleagues assessed stability across tasks and postures during speech, math, and reaction time stressors. Correlations within the same task across posture for healthy undergraduate women ranged from .52 to .73 (Cacioppo et al., 1994) and intertask correlations ranged from .48 to .92 for RSA and .42 to .69 for PEP (Berntson et al., 1994). Sloan, Shapiro, Bagiella, Gorman, and Bigger (1995) examined RSA reactivity only in healthy adult men and women. Intraclass correlations for combined tasks across sessions averaged .66 for measures of total variability and low frequency power, but were smaller for high frequency variability and low/high frequency ratio, ranging from .26 to .38. Within the same session, intertask correlations for reactivity ranged from .58 to .85 (Sloan, Shapiro, Bagiella, Fishkin, et al., 1995).

Aside from RSA's role in mechanisms underlying of HR reactivity, examining RSA responses separately is in and of itself important. RSA as an index of vagal tone may be a marker for poor cardiovascular function and risk for cardiovascular disease. Reviews of this literature (Kristal-Boneh, Raifel, Froom, & Ribak, 1995; van Ravenswaaij-Arts, Kollee, Hopman, Stoeltinga, & van Geijn, 1993) illustrate the utility of RSA as a marker of severe coronary artery disease and prognosis following a cardiac event. Others have linked decreased vagal tone to psychosocial correlates of cardiovascular disease such as hostility (Sloan et al., 1994), anxiety (Friedman & Thayer, 1998a, 1998b), and depression (Glassman & Shapiro, 1998; Musselman, Evans, & Nemeroff, 1998). Within a sample of 11-year-old boys at risk for juvenile delinquency, boys with higher internalizing and externalizing pathology exhibited lower resting RSA (Pine et al., 1998). Porges, Doussard-Roosevelt, Portales, and Greenspan (1996) demonstrated that infants who showed smaller decreases in RSA in response to an attention task had greater behavioral problems at 3 years of age.

Porges and his colleagues suggested that vagal tone serves to maintain homeostasis and promote growth and restoration. Thus, decreased vagal tone allows sympathetic input to exert more influence on the cardiovascular system, leaving the system at greater risk of coronary events. The relationship of vagal tone to psychosocial attributes suggests that reduced tonic parasympathetic activity may derive from or lead to risk-related personality characteristics. Porges also has suggested that vagal activity functions as a "brake"

to quickly regulate responses to environmental demands. An unresponsive "vagal brake" suggests an inability to adjust to environmental demands properly (Porges, 1994; Porges et al., 1996). This dysregulation might manifest in personality and behavior.

Our laboratory has focused on the development of biological and psychosocial risk factors for cardiovascular disease in children and adolescents. This work, as well as that of others, has been fruitful in charting the developmental course of cardiovascular disease risk (Ewart & Kolodner, 1994; Matthews, Woodall, & Stoney, 1990; Treiber, Musante, Strong, & Levy, 1989; Woodall & Matthews, 1989). More recently, we have become interested in patterns and clustering of risk factors and examining multiple factors that, in conjunction, can place an increased burden on the cardiovascular system. In the present investigation, we examined patterns of PEP and RSA reactivity as individual response stereotypy in children and adolescents. We hypothesized that patterns of sympathetic and parasympathetic reactivity observed in adults would be apparent in children and adolescents and that these patterns would be consistent across tasks. Further, we hypothesized that this classification scheme would relate to changes on other cardiovascular variables. Finally, we explored whether the patterns showed relationships with psychosocial risk factors for cardiovascular disease, namely hostility, as well as an index of maladaptation to the environment, namely family conflict. We chose these factors to reflect Porges' ideas of vagal dysregulation manifested in personality and behavior.

## Method

### Participants

We recruited 201 participants from Pittsburgh, Pennsylvania area school districts. We excluded one participant due to a fever, and one due to protocol problems. We required that participants had no history of cardiovascular disease, no condition that would require medication affecting the cardiovascular system, no alcohol or drug abuse, no history of mental illness, and no psychiatric counseling within the past year. Other eligibility requirements were <80% above ideal weight (to ensure adequate impedance cardiography signals) and no smoking in the prior 12 hours before the laboratory session. We recruited approximately equal numbers of Black ( $n = 97$ ) and White ( $n = 102$ ) participants, as well as approximately equal numbers of male ( $n = 101$ ) and female ( $n = 99$ ) participants. A more complete description of the participant sample is provided in Allen and Matthews (1997).

### Psychosocial Measurement

The protocol included two measurements relating to social adjustment: hostility and family environment. We measured hostility via the 23-item version of the Cook-Medley Hostility Scale (Cook & Medley, 1954; Costa, Zonderman, McCrae, & Williams, 1985). This scale demonstrates a high correlation with the original 50-item scale ( $r = .95$ ) and good test-retest reliability and internal consistency (Woodall & Matthews, 1993). We made minor wording changes to the version given to children to make some questions more appropriate (e.g., we changed "acquaintances" to "school friends"). Participants rated statements as true or false and all items were summed such that higher scores indicated higher hostility. For our purposes, we collected responses on only the angry affect, cynical attitudes, and aggressive responding subscales. We chose these subscales based on the results of Barefoot, Dodge, Peterson, Dahlstrom, and Williams (1989), who demonstrated that these three subscales were related to coronary heart disease mortality.

We measured family environment using the conflict subscale of the Family Environment Scale (FES; Moos & Moos, 1981). The conflict subscale describes the amount of openly expressed anger, aggression, and conflict among family members. The subscale consists of nine true-false statements. We administered the conflict subscale to participants and their parents to assess family environment from both perspectives. The FES exhibits high internal consistency (Chronbach's  $\alpha = .89$ ), is valid against a number of family supportiveness measures, and predicts psychosomatic complaints and depression in adult family members (Holahan & Moos, 1981, 1982).

### Physiological Measurement

The computation of PEP and RSA reactivity required the measurement of electrocardiographic (ECG), impedance cardiographic (ZKG), and BP signals. These signals also allowed computation of stroke volume (SV), CO, and total peripheral resistance (TPR). A more complete description of the physiological measures and protocol for the current sample can be found in Allen and Matthews (1997).

A standard Lead II electrode configuration with three Cleartrace LT disposable Ag-AgCl electrodes was used to collect the ECG. The two active electrodes were placed on each side of the abdomen below the bands for the ZKG measurement and the ground electrode was placed beside the navel.

A Minnesota Impedance Cardiograph (Model 304-B, Bio-Impedance Technology, Inc., Chapel Hill, NC) measured the ZKG signals according to published guidelines using a tetrapolar, aluminum/mylar electrode band configuration (Sherwood et al., 1990). The cardiac output program (COP) acquired, stored, and processed the ECG and ZKG signals via a PC and A/D board. The COP program calculated SV for ensemble-averaged waveforms using the Kubicek equation (Kubicek, Karnegis, Patterson, Witsoe, & Mattson, 1966). The COP program computed CO as the product of SV and HR for a given period.

We monitored systolic (SBP) and diastolic (DBP) blood pressure using an IBS Model SD-700A sphygmomanometer (IBS Corp., Waltham, MA) and a standard occluding cuff placed on the non-dominant arm. BP measurements occurred at 1.5-min intervals during the tasks and coincided with measurement periods for ZKG and ECG. The BP readings were entered into the COP program for scoring. The COP program computed TPR using the formula  $TPR = \frac{[(SBP - DBP)/3] + DBP}{CO} \cdot 80$ .

Customized software extracted continuous interbeat intervals (IBI) from COP program output files. The IBI values were screened and edited for artifactual values. For each data series (e.g., initial rest), the mean successive difference statistic (MSD) was computed. This statistic reflects the mean of the difference between successive IBIs. MSD filters out low frequency sources of variability in IBI series and has been validated against pharmacologically manipulated cardiac vagal control and spectral analytic statistical techniques (Hayano et al., 1991).

### Reactivity Tasks

We chose reactivity tasks based on their ability to elicit  $\beta$ -,  $\alpha$ -, or mixed-adrenergic patterns of response. As described by Allen, Bouquet, and Shelley (1991), the reaction time task produces a  $\beta$ -adrenergic response, as evidenced by increased cardiac reactivity. We chose the mirror-tracing task because of its ability to produce an  $\alpha$ -adrenergic response, resulting in increases in vasomotor tone (Kasprowicz et al., 1990). The Social Competence interview (Ewart & Kolodner, 1991) served as the mixed  $\alpha/\beta$ -adrenergic

task, as past research has shown that this task elicits both cardiac and vascular increases.<sup>1</sup>

### Laboratory Protocol

Adolescents and parents signed a consent form prior to participation. Children signed an assent form. The Psychosocial Institutional Review Board at the University of Pittsburgh approved all forms prior to study initiation. Participants and a parent/guardian arrived at the laboratory at 8:30 a.m. An experimenter applied the necessary electrodes and transducers for measurement of ECG, ZKG, and BP. Participants then sat quietly during an initial 10-min rest period and then completed the tasks in a counterbalanced order with intervening 8-min rest periods. Subsequent to completion of the tasks, participants sat quietly during a final 10-min rest. Next, the experimenter removed the electrodes and transducers. Participants completed psychosocial questionnaires and an interview and were paid \$75 plus money earned during the reaction time.

We recorded ECG and ZKG signals during all tasks and rest periods. We ensemble averaged the ZKG data by individual minutes and created means for each period. BPs were taken at regular intervals throughout the rests and tasks and were averaged to reflect values for these periods. TPR and CO reactivity values reflect percent change from baseline. Percent change scores for CO and TPR are preferable to difference scores because of concerns regarding the accuracy of absolute values of impedance-derived measures (Miller & Horvath, 1978; Sherwood et al., 1990).

We classified participants according to the exhibited pattern of PEP and MSD reactivity during each task. Specifically, we classified participants exhibiting a decline in PEP as showing sympathetic activation and participants exhibiting an increase in PEP as showing sympathetic withdrawal. Similarly, we classified participants exhibiting an increase in MSD as showing parasympathetic activation and participants exhibiting a decrease in MSD as showing parasympathetic withdrawal. A cross-tabulation of this delineation produced four categories of responses, or four patterns of PEP/MSD reactivity, named here according to the terminology of Berntson, Cacioppo, and colleagues (Berntson et al., 1994, 1996; Berntson, Cacioppo, & Quigley, 1991, 1993; Cacioppo et al., 1994). *Coinhibitors* exhibited PEP increases and MSD decreases, *reciprocal parasympathetic responders* exhibited PEP increases and MSD increases, *reciprocal sympathetic responders* exhibited PEP decreases and MSD decreases, and *coactivators* exhibited PEP decreases and MSD increases.<sup>2</sup>

<sup>1</sup>All participants engaged in a fourth reactivity stressor, a forehead cold pressor. For this task, the experimenter placed on the participants' foreheads a 2-quart bag of two parts crushed ice and one part water for 1 min. However, we did not utilize data from this task in the present analysis. This task also produces a large vasoconstrictive response leading to blood pressure increases. In this case, the probable effects of afterload on PEP make its usefulness as an index of sympathetic  $\beta$ -adrenergic activation questionable.

<sup>2</sup>We defined our groups based on changes in PEP and MSD that were not zero. This method resulted in a few individuals classified into a change group who exhibited little change in one parameter or another. For example, a few participants with a PEP reactivity score of 0.5 ms were considered "sympathetic responders" when, in fact, the change in PEP was negligible. We conducted an additional analysis in which we removed individuals who showed less than or equal to a  $\pm .25$  standard deviation change from zero on either PEP or MSD. This analysis resulted in smaller cell sizes for each pattern (12, 11, 96, and 4 for the coinhibition, coactivation, reciprocal sympathetic, and reciprocal parasympathetic groups, respectively). The differences between the less inclusive classification scheme were more striking and were consistent with the original patterns of cardiovascular and psychosocial results. We took this finding to suggest that including the nonresponders as responders resulted in more conservative tests, increasing the likelihood of making a type II error. Therefore, we presented the more inclusive classification scheme.

**Table 1.** Proportions of Participants Classified in Groups Based on PEP and MSD Reactivity

Pattern group:	SNS	PNS	Task			Across tasks <sup>a</sup>
			Reaction time	Interview	Mirror	
			f (%)	f (%)	f (%)	
Coinhibition	–	–	7 (4%)	56 (29%)	27 (15%)	19 (11%)
Reciprocal parasympathetic	–	+	4 (2%)	8 (4%)	7 (4%)	4 (2%)
Reciprocal sympathetic	+	–	136 (73%)	114 (59%)	124 (68%)	131 (75%)
Coactivation	+	+	39 (21%)	16 (8%)	24 (13%)	21 (12%)

$\chi^2 (1, N = 175) = 235.9,$   
 $p = .000$

Note: PEP = prejection period; MSD = mean successive difference; SNS = sympathetic nervous system; PNS = parasympathetic nervous system.

<sup>a</sup>Participants who exhibited the same SNS/PNS pattern for at least two of the three tasks.

**Results**

**Patterns of PEP/MSD Response**

Table 1 presents the number of participants that we classified by the four autonomic patterns for each of the three tasks. Each task elicited distributions among all four patterns and these distributions were significantly different from each other,  $\chi^2 (6, N = 562) = 50.85, p < .001$ . The largest proportion of participants during each task exhibited a reciprocal sympathetic pattern (PEP and MSD decreases), though the proportion of individuals exhibiting this pattern was not equal across tasks. The reaction time tasks produced the most individuals classified within the reciprocal sympathetic activation pattern, followed in order by the mirror tracing and interview tasks. Given that this is the pattern presumed to underlie all reactivity during these tasks, it is of particular interest that a substantial proportion of participants did not exhibit this pattern. The second most common pattern was not the same for each task. Coactivation was the second most common pattern exhibited during the reaction time task, whereas coinhibition, a pattern involving sympathetic withdrawal, was the second most common pattern exhibited during the interview. Almost equal proportions of participants exhibited these two patterns during the mirror-tracing task. Only a small proportion of participants exhibited reciprocal parasympathetic patterns. These data contradict the view that these standard reactivity tasks produce a ubiquitous sympathetic response.

**Consistency of PEP/MSD Patterns**

Next, we classified participants exhibiting the same pattern during at least two of the three tasks as a consistent responder for that pattern. Table 1 also provides the distribution of participants classified as consistent responders. Of the 194 participants with both PEP and MSD values for at least two tasks, 175 exhibited the same pattern during at least two of the three tasks. Of the 170 participants with both PEP and MSD values for all three tasks, 87 exhibited the same pattern during all three tasks. Thus, these patterns reflect individual differences, at least in the context of laboratory reactivity tasks.

To further test the consistency of sympathetic and parasympathetic responses, we performed correlations between PEP and MSD among tasks as well as average intraclass correlations across all three tasks. Table 2 presents these correlations. The correlations are substantial and suggest a greater intertask consistency and reliability for MSD reactivity than for PEP reactivity.

Table 3 presents the correlations between PEP, MSD, and HR. These correlations demonstrate moderate to large significant relationships between HR and both PEP and MSD. As PEP shortens and MSD decreases, HR increases, indicating that these measures reflect at least in part sympathetic and parasympathetic control of HR, respectively. Further, the lack of significant correlations between PEP and MSD reactivity during the interview and mirror-tracing tasks reflects the independence of sympathetic and parasympathetic input and the presence of individual differences. The correlation between PEP and MSD during the reaction time task is small but significant. However, this correlation is not surprising considering that this task included the largest proportion of participants who exhibited the reciprocal sympathetic pattern. In other words, the greater the situation response specificity of a task to produce the same pattern in all individuals, the greater the correlations between PEP and MSD.

**Patterns and Hemodynamic Responses**

Next, we examined responses of the cardiovascular system as a whole based on categorization by the patterns of PEP and MSD response. Because only four participants were classified as reciprocal parasympathetic responders, we excluded this group from the analysis. For illustrative purposes, the means for this group are presented in Figure 1. The means for the three patterns included in the analyses are also presented in Figure 1 for the purpose of comparison.

We performed univariate analyses of variance (ANOVAs) using the GLM general factorial procedure in SPSS 8.0 (SPSS, Inc.,

**Table 2.** Correlations Between PEP and MSD Responses Across Tasks

	Reaction time-mirror	Reaction time-interview	Interview-mirror	Intraclass correlation
PEP	.51** (197)	.36** (194)	.54** (194)	.69** (194)
MSD	.79** (199)	.78** (196)	.78** (197)	.91** (196)

Note: ns for the correlations are provided in the parentheses. PEP = prejection period; MSD = mean successive difference. \*\* $p < .01$ .



**Table 3.** Correlations Between PEP, MSD and Heart Rate for Each Task

	Heart rate			MSD		
	Reaction time	Interview	Mirror	Reaction time	Interview	Mirror
PEP	-.66**	-.39**	-.37**	.18*	.04	.12
MSD	-.52**	-.53**	-.59**	—	—	—

Note: PEP = preejection period; MSD = mean successive difference. \**p* < .05, \*\**p* < .01.

Chicago, IL) for all tests on cardiovascular and psychosocial variables. The three patterns (coinhibitors, reciprocal sympathetic responder, and coactivators) served as the grouping variable. The cardiovascular variables used were HR, SBP, DBP, SV, CO, and TPR. Table 4 presents the means, *F* values, and significance for these ANOVAs. Significant effects emerged for HR, SV, CO, and TPR. No significant effects emerged for SBP or DBP.

Post hoc tests were performed using the Scheffé *S* multiple comparison procedure, as this statistic is appropriate for unequal numbers of subjects. These comparisons revealed that reciprocal sympathetic responders exhibited the highest HR reactivity overall, whereas coactivators exhibited the lowest. Coinhibitors' HR reactivity did not differ significantly from the other two groups. Coinhibitors exhibited larger decreases in SV, a decrease in CO, and larger increases in TPR, compared with coactivators and reciprocal sympathetic responders.

**Patterns and Psychosocial Attributes**

We examined if these distributions differed by age cohort, gender, or ethnic group with additional chi-square tests. To maintain adequate cell frequencies, it was necessary to create separate distributions for sympathetic and parasympathetic activity. For example, we performed two chi-square tests for ethnic group, one testing the 2 × 2 distribution of ethnic group with PEP reactivity (increase or decrease), and the other testing the 2 × 2 distribution of ethnic group with MSD reactivity (increase or decrease). The chi-square test of the distribution of ethnic group with MSD reactivity was

significant,  $\chi^2(1, N = 175) = 4.42, p = .036$ . White participants were more likely to show a consistent pattern of parasympathetic activation than Black participants (20% and 8% within ethnicity, respectively). The other chi-square tests for age cohort and gender were not significant.

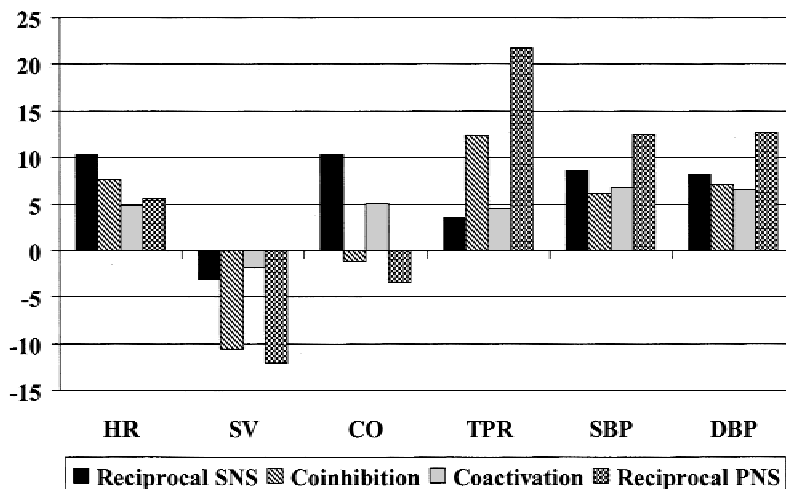
Next, we examined if consistent responders differed in their psychosocial characteristics by the PEP/MSD pattern exhibited. We performed comparable univariate ANOVAs using each of the following psychosocial characteristics as dependent variables: Cook-Medley subscales (cynicism, hostile affect, aggressive responding) and the Family Environment Scale conflict subscale completed by both the child and a parent. The results of these univariate ANOVAs are presented in Table 4.

We found significant effects for the both the child and parent responses on the conflict subscale of the Family Environment Scale. Post hoc tests using Scheffé *S* revealed similar patterns for child's and parent's level of perceived family conflict; coinhibitors and their parents reported significantly higher conflict than coactivators, and reciprocal sympathetic responders reported levels between the two. The results for hostile affect showed a similar pattern, with coinhibitors reporting the highest hostile affect and coactivators the least. However, this effect reached only marginal significance.

To examine if the ethnic differences in parasympathetic reactivity could be explained by differences in a child's and parent's level of perceived family conflict, we performed two univariate ANOVAs with ethnicity as the grouping variable and each conflict variable as the dependent variables. No significant differences were found between ethnic groups on either child's or parent's level of perceived family conflict (*F*s < 1.81, *p*s > .18).

**Discussion**

The present analysis of children and adolescents' responses to stress demonstrate the utility of simultaneously considering sympathetic and parasympathetic reactivity. PEP and RSA reactivity during a variety of demanding tasks was stable across tasks within individuals. The intertask and intraclass correlations demonstrated considerable consistency, and we were able to classify participants as consistent responders. In other words, underlying autonomic responses during reactivity task reflect the degree of individual differences. This finding builds on the work of Berntson, Ca-



**Figure 1.** Means of cardiovascular responses for preejection period (PEP) and mean successive difference statistic (MSD) patterns, including the reciprocal parasympathetic group. HR = heart rate; SV = stroke volume; CO = cardiac output; TPR = total peripheral resistance; SBP = systolic blood pressure; DBP = diastolic blood pressure; SNS = sympathetic nervous system; PNS = parasympathetic nervous system.

**Table 4.** Mean Cardiovascular Reactivity and Psychosocial Attributes by PEP/MSD Group

	Reciprocal sympathetic (n = 130)		Coinhibition (n = 19)		Coactivation (n = 21)		F	p
	M	SD	M	SD	M	SD		
<b>Cardiovascular</b>								
Heart rate (bpm)	10.2 <sup>a</sup>	(5.0)	7.6 <sup>a,b</sup>	(4.8)	4.8 <sup>b</sup>	(6.4)	11.25	.000
Systolic BP (mmHg)	8.5	(6.5)	6.1	(5.6)	6.8	(6.6)	1.65	.20
Diastolic BP (mmHg)	8.2	(7.3)	7.1	(7.0)	6.6	(7.2)	0.54	.58
Stroke volume (ml)	-4.5 <sup>a</sup>	(8.0)	-10.6 <sup>b</sup>	(7.7)	-1.9 <sup>a</sup>	(7.4)	6.53	.002
Cardiac output (l/min)	8.4 <sup>a</sup>	(0.9)	-1.2 <sup>b</sup>	(2.2)	5.1 <sup>a,b</sup>	(2.1)	8.52	.000
Total peripheral resistance (%, dyne-sec/cm <sup>5</sup> )	3.9 <sup>a</sup>	(11.4)	12.4 <sup>b</sup>	(15.6)	4.5 <sup>a,b</sup>	(10.1)	4.33	.02
<b>Psychosocial</b>								
C-M hostile affect	2.4	(1.5)	3.0	(1.5)	1.8	(1.4)	2.78	.07
C-M aggressive responding	4.5	(2.0)	4.3	(2.3)	4.2	(2.1)	0.28	.76
C-M cynicism	6.7	(2.4)	6.5	(2.3)	5.6	(2.1)	1.74	.18
Conflict (child report)	3.2 <sup>a</sup>	(2.0)	3.8 <sup>a</sup>	(2.1)	2.1 <sup>b</sup>	(2.0)	4.49	.01
Conflict (parent report)	3.3 <sup>a</sup>	(2.2)	5.0 <sup>b</sup>	(2.6)	3.4 <sup>a,b</sup>	(2.3)	4.19	.02

Note: Means sharing the same superscript do not differ at the  $p < .05$  level. PEP = preejection period; MSD = mean successive difference; C-M = Cook-Medley subscales.

cioppo, and their colleagues, who demonstrated the presence and individual stereotypy of these patterns in adults. These patterns suggest that stability of reactivity is not simply the result of an older, less flexible cardiovascular system, but begins early when the cardiovascular system is still relatively healthy and adaptable to environmental demands. The finding that these patterns were stable across tasks is particularly telling in the present study because the individual tasks were chosen to elicit different cardiovascular responses, namely cardiac and vascular responses. Yet, 90% of participants exhibited the same pattern during at least two of the tasks and half of participants with complete data for all tasks exhibited the same pattern during all three.

Given the presence of individual consistency in reactivity, task-driven situation response specificity also was evident in the distributions of participants across the four patterns. A full 94% of participants exhibited one of the sympathetic activation patterns during the reaction time task, whereas 66% and 81% exhibited these patterns during the interview and mirror-tracing tasks, respectively. A considerable proportion of children and adolescents exhibited coinhibition during the interview task, suggesting that this task may be particularly adept at eliciting PEP and RSA decreases. Measuring HR alone would not reveal this difference in autonomic control, as coinhibition can produce an increase in HR by releasing inhibition of the intrinsic firing rate of the sinoatrial node.

These patterns were related to previously described patterns of cardiac and vascular responses. Coinhibitors exhibited what has been traditionally referred to as a vascular pattern of response. They responded with decreases in SV and CO and large increases in TPR. Although we have used PEP as an index of sympathetic activation based on prior work demonstrating that PEP responses to stress disappear under  $\beta$ -adrenergic blockade, PEP is also affected by preload and afterload (Lewis et al., 1974). An increased afterload produces a lengthening of PEP to decrease contractility and decreases in SV. It is likely that afterload was having a significant effect on the responses of the coinhibitors and reciprocal parasympathetic responders, as evidenced by their increases in TPR and decreases in SV.

The coactivator and reciprocal sympathetic responder groups both exhibited a cardiac pattern of cardiovascular response. Further, they did not differ significantly in reactivity for SV, CO, or TPR. However, the two groups did differ significantly in HR reactivity, suggesting that sympathetic responders are not simply a homogenous group of cardiac responders. As expected, coactivators produced smaller changes in HR than produced by reciprocal sympathetic responders. Parasympathetic activation did in fact buffer the HR responses of coactivators, such that they had the lowest HR reactivity of the three groups. Reciprocal sympathetic responders exhibited the highest HR reactivity. Utilizing RSA to examine differences in parasympathetic reactivity distinguished between two types of cardiac responders, those with exaggerated HR reactivity, and those with buffered HR responses.

Coactivators perceived their family environments as involving less conflict compared with reciprocal sympathetic responders and coinhibitors, suggesting more adaptive interactions with their environments. This finding tentatively supports Porges' vagal brake hypothesis (Porges, 1994; Porges et al., 1996) in that children whose parasympathetic nervous systems responded as active brakes during sympathetic activation also exhibited comparatively adaptive behavior. Children exhibiting patterns of parasympathetic withdrawal reported more conflict, suggesting more maladaptive behavior. Of interest is that these differences occurred in a relatively normal sample, free of cardiovascular or major behavioral problems.

We did not find any differences in the distribution of participants among the patterns by age group or gender. We did find a greater proportion of White participants exhibiting parasympathetic activation relative to Black participants. This finding suggests that Black participants did not show the buffering effect of parasympathetic activation on HR reactivity. Results from this sample reported by Allen and Matthews (1997) tentatively support this proposal. Black adolescents exhibited greater HR reactivity during the Ewart interview than did White adolescents, though ethnic differences in HR reactivity were not found for the reaction time and mirror tracing tasks. Also, differences in family conflict were not found, suggesting that the psychosocial factors found to be related to the overall patterns do not explain ethnic differences

in parasympathetic reactivity. More research is warranted before making any strong conclusions from the ethnicity results.

Because of the small number of children who consistently exhibited a reciprocal parasympathetic pattern, we were unable to include this group in the reactivity and psychosocial analyses. However, the pattern of means in Figure 1 suggests that reciprocal parasympathetic responders exhibited the most extreme BP and TPR reactivity. Further research including a larger group of reciprocal parasympathetic responders is warranted before we can draw any conclusions about this pattern.

Our study of children and adolescents suggests that reactivity patterning occurs in younger populations, including those in their first decade of life. According to the reactivity hypothesis (Krantz & Manuck, 1984; Matthews et al., 1986), exaggerated cardiovascular responses to stress play a role in the development of cardio-

vascular disease. Children exhibiting stable maladaptive patterns of cardiovascular reactivity may be progressing down the path toward disease, suggesting that childhood may be the optimal time to target preventative measures.

This research supports the idea of patterning of multiple reactivity responses to describe more fully the synergistic nature of the autonomic nervous system's response to stress. Historically, psychophysiological research has focused primarily on sympathetic responses, and more recently on parasympathetic influences, often examining responses individually. Combining the two provides a more accurate picture of stress responses and is useful for describing a response profile linked to psychosocial characteristics. Future research should focus on the stability of these patterns across longer time spans and their relationship to cardiovascular disease risk.

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