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Atypical autonomic regulation in perpetrators of violent domestic abuse

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Abstract

Perpetrators of domestic violence describe symptoms that are compatible with exaggerated autonomic arousal at the time of the domestic violence. This inappropriate arousal may be reflected in altered heart rate regulation. If heart rate is systematically regulated by vagal mechanisms, then increases in heart rate should correlate with decreases in cardiac vagal activity, as indexed by respiratory sinus arrhythmia (RSA). We hypothesized that perpetrators of domestic violence have an alteration in heart rate regulation. To test this hypothesis we compared the results of a postural shift performed on perpetrators, healthy volunteers, and nonviolent alcoholics. Results showed there were no significant differences in heart rate, RSA, or catecholamines. However, the significant inverse relationship between posture-elicited changes in RSA and heart rate present in the healthy volunteers was not found in perpetrators. These differences in the covariation between heart rate and RSA may represent differences in the neural regulation of heart rate and may be related to difficulties in controlling autonomic state.

Descriptors: Domestic violence, Respiratory sinus arrhythmia, Autonomic nervous system, Alcoholism, Cardiac vagal tone, Heart rate

Domestic violence is a world wide problem with enormous social and medical implications (Bignall, 1993). Acts of domestic violence account for at least 20% of all emergency room visits (Goldberg & Tomlanovich, 1984; McLeer & Anwar, 1989). In the United States, it is estimated that 30% of all women who are murdered are killed by their husbands, boyfriends, or ex-partners (Satcher, 1995).

Current theories concerning the etiology of domestic violence leave many unanswered questions. Many researchers have attributed domestic violence to either the effects of alcohol or to learned behavior patterns (Bergman & Brismar, 1994; Fitch & Papantonio, 1983; Hotaling & Sugarman, 1986). Although these factors play a role in domestic violence, the fact remains that the majority of abusers are sober when they are violent (Eberle, 1982; Kantor & Straus, 1995), and almost half of them have not been exposed to abuse growing up (Caesar, 1988; Kantor & Straus, 1995; Widom, 1989a, 1989b).

When perpetrators of domestic violence become violent, they often exhibit tremulousness and vocal changes that are accompanied by palpitations, breathlessness, flushing, and sweating. These symptoms, characteristic of autonomic nervous system (ANS) arousal, are out of proportion to environmental stimuli and are perceived by the perpetrators as ego-dystonic and not under their conscious control (Bitler, Linnoila, & George, 1994; Margolin, John, & Gleberman, 1988).

Heart rate is frequently studied as an index of autonomic function. Heart rate regulation is a complex feedback system with parasympathetic and sympathetic components that in certain situations appears to be dominated by parasympathetic activity of the vagus nerve. The vagus acts as a brake on the intrinsic rate of the heart and opposes sympathetic activity at the sinoatrial node (Larsen, Schneiderman, & Pasin, 1986; Levy & Martin, 1998; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). The vagus responds rapidly to changing metabolic demands, whether from emotional arousal or from a simple posture change, and immediately adjusts heart rate (Salata & Zipes, 1991).

Numerous studies have shown that noninstitutionalized criminals and conduct disordered/delinquent youth have autonomic disturbances illustrated by lower resting heart rates (Mezzacappa et al., 1997; Raine, 1996a, 1996b). In a recent study, male perpetrators of domestic violence were monitored for changes in autonomic function during a stressful interaction with their wives. Curiously, the men uniformly showed an increase in cardiac vagal activity but less than half showed the expected corresponding decrease in heart rate (Gottman et al., 1995). Previously, we reported that some subjects who initiate domestic violence experience panic symptoms at the time of the violence (George,

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Anderson, Nutt, & Linnoila, 1989). Inasmuch as panic is associated with autonomic nervous system dysfunction (Friedman & Thayer, 1998), altered autonomic regulation may contribute to the etiology of domestic violence.

To better understand the autonomic regulation of heart rate in perpetrators of domestic violence, we employed a postural challenge. The act of standing up is a natural physiological stressor that raises heart rate without the confounding effects of psychological input (Stein, Tancer, & Uhde, 1992). We reasoned that if perpetrators of domestic violence manifest alterations in autonomic function when they are violent, then they may also manifest alterations when they are not violent. These alterations may be reflected in the regulation of heart rate during a posture challenge.

Heart rate regulation is a complex process involving sympathetic and parasympathetic components; however, if heart rate is being systematically regulated by vagal mechanisms, then increases in heart rate should correlate with decreases in cardiac vagal activity, as indexed by respiratory sinus arrhythmia (RSA) (Berntson, Cacioppo, & Quigley, 1993). For example, during exercise, when sympathetic excitation supplements the vagal withdrawal to the heart to promote increased cardiac output, research has found a reliable relationship between RSA and heart rate (Hatfield et al., 1998).

To investigate heart rate control, we compared the effects of a postural shift on the dynamic relationship between heart rate and RSA in violent perpetrators and nonviolent healthy volunteers. We hypothesized that a lack of systematic vagal regulation of the heart might be related to the reported difficulties in autonomic regulation noted in violent perpetrators. Because of the prevalence of alcoholism among violent perpetrators and the reported effects of alcohol on the ANS (Ito, Miller, & Pollock, 1996; O'Farrell & Murphy, 1995; Weise, Muller, Krell, Kielstein, & Koch, 1985), we also studied a group of alcoholics with no history of violence.

Methods

Participants

We performed a posture challenge in three groups: perpetrators of domestic violence, nonviolent alcoholics, and healthy volunteers. Perpetrators consisted of 13 male and 3 female participants who had exhibited repeated episodes of physical violence toward a spouse or significant other (e.g., hitting, pushing, choking, and threatening with a weapon). The perpetrators were recruited through newspaper advertisements that sought individuals who lose control and are violent toward their spouses or significant others. Interviews with significant others were conducted to document the presence of recent violence. To avoid the confounding effects of alcohol-induced disinhibition, we required that at least some of the violent acts occurred when the subjects were not under the influence of alcohol. Fifteen of the perpetrators fulfilled DSM III-R criteria (American Psychiatric Association, 1987) for alcohol dependence/abuse during their lifetime and 10 were actively drinking 3 weeks prior to the study. To address the possibility that chronic heavy alcohol ingestion could alter the autonomic nervous system (Ito et al., 1996; O'Farrell & Murphy, 1995; Weise et al., 1985), we also studied a nonviolent alcoholic comparison group. Nonviolent alcoholics fulfilled DSM III-R criteria for alcohol abuse or dependence and included 10 men and 3 women recruited from our inpatient alcoholism treatment program. Healthy volunteers consisted of 13 male and 2 female participants who did not fulfill criteria for any Axis I diagnosis. Healthy volunteers were recruited from the normal volunteer program of the National Institutes of Health in Bethesda, Maryland, and were the only group reimbursed for their participation in the study. Healthy volunteers and alcoholics had no history of interpersonal violence confirmed by a significant other.

All participants underwent an extensive physical examination to ensure that they were in good physical health. All participants were medication free for at least 3 weeks prior to the study and had negative urine drug screens. Social and economic status was measured by the method of Hollingshead (Hollingshead & Redlich, 1958). Lifetime alcohol consumption was determined for each participant using a structured research questionnaire and expressed as kilograms consumed over the lifetime (Eckardt, Parker, Noble, Feldman, & Gottschalk, 1978). Perpetrators had an electroencephalogram and MRI of the brain to rule out central nervous system pathology that could contribute to their violent behavior. Subjects with a history of seizures or major head trauma (defined as a period of unconsciousness exceeding 1 hr) were excluded from participation. Participants with a history of IV drug abuse, schizophrenia, bipolar disorder, or organic brain dysfunction were excluded from participation. Written informed consent was obtained from all participants after the nature and possible consequences of the study were explained. The National Institute of Alcohol Abuse and Alcoholism Institutional Review Board approved the study.

The following two case reports illustrate behaviors and describe symptoms frequently reported by perpetrators:

Subject 1 was a 31-year-old married tradesman who was physically abused by his alcoholic mother. During grade school, he initiated numerous fights and, as an adult, he was violent toward numerous girlfriends. He reported hitting his wife on multiple occasions, once striking her in the face with a closed fist and giving her a black eye. Prior to episodes of domestic violence, he felt confused and had a sense of losing control. He had a rapid heart beat, sweaty palms, and he paced the floor. He was preoccupied with staying in control so that he would not be violent, and, on occasion, he locked himself in his room to avoid getting into a fight with his wife.

Subject 2 was a 34-year-old, twice-married man who reported that his violent behavior resulted in divorce from his first wife and separation from his present wife. He recounted numerous episodes of hitting his present wife in the face. He also recounted punching her, grabbing her throat, and pulling her hair. Prior to the violence, he typically felt his heart race, he trembled and sweat, and he felt hot behind the back of his head. He described feeling enraged and said, "I can't believe I am doing these things, but I can't stop." He avoided alcohol in an effort to decrease the violence.

Measures of Autonomic Function

Autonomic nervous system function has been assessed in various clinical populations by examining the baroreceptor reflex during a posture challenge (Rudorfer, Ross, Linnoila, Sherer, & Potter, 1985; Stein et al., 1992). During a postural challenge, blood pools in the legs and causes a fall in cardiac output. This decrease in cardiac output activates arterial baroreceptors, which trigger an immediate compensatory decrease in heart period (heart rate increase) mediated by the vagus nerve. Studies suggest that vagal regulation of the heart is occurring via nerve fibers originating in the nucleus ambiguus, a brainstem structure that coordinates cardiac output with breathing and responds to changing metabolic demands. Because vagal fibers from the nucleus ambiguus have an inherent respiratory rhythm, the tonic level of vagal action on the heart can be indexed by the amplitude of respiratory sinus arrhythmia. Respiratory sinus arrhythmia (RSA) is a rhythmical fluctua-

tion in heart rate that occurs at a frequency similar to spontaneous breathing and is characterized by a respective lengthening and shortening of the R-R interval (Akselrod et al., 1985; Berntson et al., 1993; Porges, 1995; Richter & Spyer, 1990; Sargunaraj et al., 1996).

In contrast to the immediate effects of the vagus on the heart, the actions of the sympathetic nervous system are delayed in onset but sustained. With increased metabolic demand, the sympathetic system is assumed to stimulate the heart via cardiac nerves terminating on the sinoatrial node and the release of epinephrine from the adrenal medulla. This system also increases peripheral resistance with the subsequent spillover of norepinephrine into the circulation. Changes in sympathetic nervous system activity can be inferred from changes in the peripheral blood norepinephrine concentration that, for example, normally doubles upon standing (Goldstein & Kopin, 1988; Larsen et al., 1986; Siever, 1987; Wallin et al., 1992). Although techniques utilizing low frequency heart rate fluctuations have been proposed as an estimate of sympathetic activity, there is no consensus regarding the validity of this strategy (Kingwell et al., 1994; Saul, Rea, Eckberg, Berger, & Cohen, 1990).

Procedure

All participants with a diagnosis of alcoholism were abstinent from alcohol for at least 3 weeks prior to the study. Nonalcoholic participants abstained from alcohol for at least 72 hr prior to the study. To eliminate possible dietary-induced variations on the catecholamine determinations, all participants were on a low monoamine diet for at least 72 hr and fasted overnight at the Warren Grant Magnusen Clinical Center of the National Institutes of Health. On the morning of the study, participants remained at bed rest while an intravenous catheter was placed in the antecubital vein and kept open with a slow infusion of normal saline. Following a 30-min baseline period, participants were asked to stand quietly by the bedside for 5 min. We sought a normal spontaneous breathing pattern that did not require the participant to be trained or to attend to the demands required by paced breathing methods. Blood for epinephrine and norepinephrine was drawn at the end of the baseline period, and after 5 min of standing. Blood samples were immediately placed on wet ice, centrifuged, and the plasma was stored at -70° C prior to analysis. The plasma norepinephrine concentrations were measured with high-pressure liquid chromatography using electrochemical detection. The interassay coefficient of variability was 10.9% with a lower limit of detection of 10 pg/ml. Plasma epinephrine concentrations were determined by the same method, and the interassay coefficient of variability was 10.7% with a lower limit of detection of 5 pg/ml (Eisenhofer et al., 1986). Blood pressure (BP) was measured (Dinamap automated BP cuff, Critikon Co., Tampa, FL) from the nondominant arm immediately before and 5 min after standing.

Heart period was recorded continuously during the study via a Vagal Tone Monitor (Delta Biometrics, Bethesda, MD). The Vagal Tone Monitor measured the R-R interval (heart period) to the nearest millisecond. The heart period data were edited off-line to remove R-wave detection artifact (Porges & Byrne, 1992). RSA was quantified according to the technique of Porges (Porges, 1985; Porges & Bohrer, 1990). This methodology accurately quantifies the variance in the heart period within the estimated frequency band associated with spontaneous breathing. The method provides two advantages over traditional spectral analyses. First, the method is designed to dynamically detrend the data to remove the influence of shifting baselines and very slow periodicities that may confound the application of spectral analysis. Second, the method enables the calculation of RSA during relatively short time periods. Because we are investigating the dynamic shift in RSA during posture shift, the nature of this physiological challenge promotes a heart period pattern that violates the assumption of stationarity and thus precludes the appropriate application of spectral analysis. Alternative methods of quantifying heart rate variability, such as measures of approximate entropy, have not been systematically related to neurophysiological mechanisms (e.g., vagal pathways) or physiological processes (e.g., RSA).

The Porges method quantifies RSA via the following steps: (a) R-R intervals are timed to the nearest millisecond to produce a time series of sequential heart periods; (b) the sequential heart periods are converted to time-based data by resampling into sequential 500-ms intervals; (c) the time-based series is detrended by a 21-point cubic moving polynomial (Porges & Bohrer, 1990) that is stepped through the data to create a smoothed template; (d) the template is subtracted from the original time series to generate a detrended residual series; (e) the detrended time series is band passed to extract the variance in the heart period pattern associated with the band of frequencies associated with spontaneous breathing (i.e., 0.12–0.40 Hz); and (f) the natural logarithm of the variance of the band passed time series is calculated as the measure of RSA (Riniolo & Porges, 1997). These procedures are statistically equivalent to frequency domain methods (i.e., spectral analysis) for the calculation of RSA when the heart period data are stationary. The amplitude of RSA was calculated during 10 sequential 30-s epochs before and after standing. Standing (the moment the knees locked) was completed at the end of the 10th 30-s epoch. The average of the epoch values within each condition (i.e., baseline and standing) was used in the data analysis.

Statistical analyses were accomplished using the STATISTICA software program. ANOVA was used to test differences between groups for baseline variables. Repeated measures ANOVA was used to look at differences between groups from a lying to a standing posture. To examine the possibility that perpetrators have a disturbance in vagal control of heart rate during a posture challenge, we examined the relationship between changes in RSA and changes in heart period from lying to standing using regression analysis. We also examined this relationship after eliminating the transition period, (the minute before and the minute after the subjects assumed an upright posture) to eliminate the potential confounding effects of an initial standing-induced heart rate instability or altered breathing pattern. In addition, we attempted to model the change in heart period for each group utilizing a multiple regression analysis using baseline heart period and change in RSA as predictors of change in heart period. To insure that by averaging the period after standing we did not overlook transitory shifts in RSA and heart period, we examined the relationship between the two on a minute-by-minute basis, starting a minute after standing. For each of these 4 min the participants were standing, we computed the change in RSA and change in heart period from the averaged first 4 min of baseline. We used the test of parallelism because distributions of regression lines slopes are relatively immune to the influence of range (Cohen & Cohen, 1983). To examine the sympathetic influence on heart rate, we examined the relationship between changes in catecholamines (norepinephrine together with epinephrine) and changes in heart period by calculating multiple regression analyses within each group, applying heart period change as the dependent variable.

Table 1. *Descriptive Statistics (mean ± SEM)*

	Perpetrators (n = 16)		Healthy volunteers (n = 15)		Alcoholics (n = 13)	
Variable	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*
Age (years)	37.25	1.79	37.33	2.50	40.08	2.12
Height (cm)	175.47	2.16	176.60	2.39	173.96	1.84
Weight (kg)	79.21	2.25	78.42	4.54	77.27	2.88
Hollingshead	3.94	0.23	4.07	0.18	3.85	0.22
Lifetime alcohol consumption* (kg)	305.44	70.95	14.00	5.82	336.62	57.23
Smoking* (cigarettes per day)	17.06	4.81	1.33	1.33	11.15	3.50

*There was a significant group difference for lifetime alcohol consumption and smoking at $p \leq .01$.

Results

There were no significant[1] group differences for age, weight, height, or socioeconomic status. The healthy volunteers smoked less and consumed significantly less alcohol than the perpetrators and the alcoholics, who did not differ from each other on these variables (Table 1). Each group exhibited a significant decrease in heart period (i.e., increase in heart rate) upon standing. Concomitantly, standing elicited a significant decrease in the amplitude of RSA (i.e., reduced vagal influences to the heart) and a significant increase in epinephrine, norepinephrine, and diastolic blood pressure. There were no significant group differences on any of these measures. The perpetrators tended to have greater increases in systolic and diastolic blood pressure, but this did not reach significance at the $p \leq .01$ level (Table 2).

Regression analyses of the relationship between changes in RSA and changes in heart period were performed for the three groups. The analyses identified a significant group effect for the slopes, $F(2,38) = 6.05$, $p \leq .01$. The pairwise contrasts for equality of slopes are as follows: healthy volunteers versus alcoholics, $F(1,24) = 0.54$, $p = .47$; healthy volunteers versus perpetrators, $F(1,27) = 10.0$, $p \leq .01$; alcoholics versus perpetrators, $F(1,25) = 7.78$, $p \leq .01$. The tests for zero regression coefficients in the three groups are as follows: healthy volunteers, $t(13) = 3.15$, $p \leq .01$, $R^2 = .43$, $B = 73.3$; alcoholics, $t(11) = 2.86$, $p = .02$, $R^2 = .43$, $B = 50.4$; perpetrators, $t(14) = -1.03$, $p = .32$, $R^2 = .07$, $B = -16.7$. Examining this data after eliminating the transition period between lying and standing produced a similar finding.[2] The tests for significant multiple regression within each group, applying change in heart period as the dependent variable and initial heart period and change in RSA as the independent variables, are presented with the overall tests preceding the individual variable tests. For healthy volunteers: $F(2,12) = 11.60$, $p \leq .01$, $R^2 = .66$; initial heart period, $t(12) = -2.82$, $p = .02$, $B = -0.42$; change in RSA, $t(12) = 3.25$, $p \leq .01$, $B = 62.3$. For alcoholics: $F(2,10) = 9.84$, $p \leq .01$, $R^2 = .66$; initial heart period, $t(10) = -2.65$, $p = .02$, $B = -0.30$; change in RSA, $t(10) = 3.09$, $p < .02$, $B = -10.4$. For perpetrators: $F(2,13) = 8.04$, $p \leq .01$, $R^2 = .55$; initial heart period, $t(13) = -3.74$, $p \leq .01$, $B = -0.24$; change in RSA, $t(13) = -0.88$, $p = .39$, $B = 44.4$.

We also examined the relationship between RSA and heart period on a minute-by-minute basis, starting a minute after standing. Analysis of the slopes indicated the regression slopes of the perpetrators and the healthy volunteers are different and they are consistently different during the initial 1-min evaluations, with healthy volunteers having a steeper slope. Similarly, the alcoholics strongly tended to have a steeper slope than the perpetrators. (Table 3). The tests for zero regression coefficients in the three groups at each minute are presented in Table 4. These results indicated that although change in RSA in response to posture shift was systematically related to change in heart period in healthy volunteers, and was initially related to change in heart period change in alcoholics, it did not systematically influence heart period change in perpetrators.

The tests for significant multiple regression within each group, applying heart period change as the dependent variable and change in catecholamines as the independent variables, are as follows: healthy volunteers, $F(2,12) = 2.94$, $p = .09$, $R^2 = .33$; alcoholics, $F(2,10) = 3.86$, $p = .06$, $R^2 = .44$; perpetrators, $F(2,13) = 0.22$, $p = .81$, $R^2 = .03$. We also investigated the individual influences of changes in norepinephrine and epinephrine on change in heart period by testing the individual regression coefficients. The tests for change in norepinephrine for each group are: healthy volunteers, $t(12) = -1.76$, $p \leq .10$; alcoholics, $t(10) = -2.68$, $p = .02$; perpetrators, $t(13) = 0.62$, $p = .55$. The tests for change in epinephrine for each group are: healthy volunteers, $t(12) = -2.32$, $p = .04$; alcoholics, $t(10) = -1.66$, $p = .13$; perpetrators, $t(13) = 0.44$, $p = .67$. Although none of the relationships between changes in catecholamines and heart period were significant after Bonferroni correction, the effects were always weakest for the perpetrators. These findings are limited by the fact that peripheral catecholamines may not accurately reflect the neural activity of the sympathetic nervous system at the level of the sinoatrial node (Goldstein & Kopin, 1988; Wallin et al., 1992).

[1]Because of the numerous statistical tests performed, a significance level of $p \leq .01$ has been used in the analyses.

[2]The following results were obtained from the data after eliminating the minute before and the minute after the subjects assumed an upright posture. Similar to the results reported above, there were no differences between groups for the change in heart period or change in RSA from a lying to a standing position, $F(2,41) = 1.22$, $p = .31$ and $F(2,41) = 1.04$, $p = .36$, respectively. We again examined the relationship between changes in RSA and changes in heart period by performing regression analysis. A test for the equality of slopes was performed: $F(2,38) = 5.02$, $p \leq .01$ for the three groups. The tests for zero regression coefficients in the three groups are as follows: healthy volunteers, $t(13) = 3.57$, $p \leq .01$, $R^2 = .50$, $B = 78.1$; alcoholics, $t(11) = 2.38$, $p = .04$, $R^2 = .34$, $B = 44.2$; and perpetrators, $t(14) = -0.52$, $p = .61$, $R^2 = .02$, $B = -11.0$. The pairwise comparisons for equality of slopes are: healthy volunteers versus alcoholics, $F(1,24) = 1.24$, $p = .28$; healthy volunteers versus perpetrators, $F(1,27) = 8.58$, $p \leq .01$; alcoholics versus perpetrators, $F(1,25) = 3.62$, $p = .07$.

Table 2. *Cardiovascular Measures During Orthostatic Challenge (mean ± SEM)*

	Perpetrators (n = 16)		Healthy volunteers (n = 15)		Alcoholics (n = 13)	
Measure	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*
Heart period (msec)						
Lying	983.78	34.22	1001.06	34.12	932.82	40.43
Standing	789.82	25.63	785.24	28.76	759.60	33.06
Respiratory sinus arrythmia						
Lying	6.12	.29	6.42	.25	5.87	.34
Standing	5.11	.21	4.94	.24	4.57	.36
Systolic blood pressure (mm/Hg)	(n = 15)					
Lying	116.07	3.90	115.60	2.24	114.85	3.53
Standing (Minute 5)	122.4	4.12	114.27	2.67	101.85	5.06
Diastolic blood pressure (mm/Hg)	(n = 15)					
Lying	66.53	2.55	67.60	3.01	68.54	2.75
Standing (Minute 5)	78.47	2.99	72.07	3.18	72.00	3.61
Epinephrine (pg/ml)						
Lying	14.44	1.20	13.93	5.56	10.23	1.45
Standing	27.25	3.25	39.27	16.93	46.85	15.09
Norepinephrine (pg/ml)						
Lying	194.13	25.26	160.27	17.73	180.62	25.20
Standing	451.13	43.46	385.60	53.17	443.92	82.14

Discussion

In all three groups, the decrease in heart period associated with standing was accompanied by (a) a decrease in RSA reflecting a withdrawal of parasympathetic tone, and (b) an increase in epinephrine and norepinephrine reflecting sympathetic activation. There were no significant baseline or postural group differences in catecholamines, blood pressure, heart period, or RSA. However, the strong correlation between change in heart period and change in RSA observed in healthy volunteers was not present in the perpetrators. Although RSA change did not predict heart period change in perpetrators, it did so in healthy volunteers. Even though the results for alcoholics did not reach traditional levels of statistical significance, the percentage of variance accounted for (40%) was not trivial. Notably, the pattern of correlations obtained using averaged standing values was similar to the pattern noted with the elimination of the transition period, and noted when examining the initial individual minute-by-minute segments. This pattern, namely, that changes in RSA and changes in heart period were correlated in healthy volunteers but not in perpetrators, remained the same despite the changing nature of the initial hemodynamic adjustments associated with standing. Although the regulation of heart rate during the first 5 min of standing is complex, with changing sympathetic and parasympathetic components, this suggests that the effect we observed was not dependent on the precise timing of posture shift. It also suggests that our methods were not adversely affected by acute changes (i.e., respiratory rate fluctuations) associated with the act of standing.

The correlations between changes in RSA and changes in heart period were clearly not as robust in alcoholics as they were in healthy volunteers. However, our data does not allow us to conclude that the lack of correlation between changes in RSA and changes in heart period is secondary to alcohol consumption alone, as there was no difference in the relationship between healthy volunteers and alcoholics. Although healthy volunteers smoked less than the other groups, this factor did not affect our results, as

Table 3. *Test of Parallelism Between Changes from Baseline in RSA and Changes from Baseline in Heart Period for Each Minute of Standing*

Time	Group effect	Perpetrators vs. healthy volunteers	Perpetrators vs. alcoholics	Healthy volunteers vs. alcoholics
Minute 2	$F(2,38) = 6.72$ $p \leq .01$	$F(1,27) = 10.42$ $p \leq .01$	$F(1,25) = 6.50$ $p = .02$	$F(1,24) = 0.55$ $p = .47$
Minute 3	$F(2,38) = 6.34$ $p \leq .01$	$F(1,27) = 10.66$ $p \leq .01$	$F(1,25) = 5.78$ $p = .02$	$F(1,24) = 1.26$ $p = .27$
Minute 4	$F(2,38) = 4.68$ $p = .02$	$F(1,27) = 8.25$ $p \leq .01$	$F(1,25) = 3.08$ $p = .09$	$F(1,24) = 1.35$ $p = .26$
Minute 5	$F(2,38) = 4.14$ $p = .02$	$F(1,27) = 6.81$ $p = .02$	$F(1,25) = 3.20$ $p = .09$	$F(1,24) = 1.19$ $p = .29$

Table 4. *Test for Zero Regression Coefficients Between Changes from Baseline in RSA and Changes from Baseline in Heart Period at Each Minute of Standing*

Time	Perpetrators	Healthy volunteers	Alcoholics
Minute 2	$t(14) = -1.00$ $p = .34$ $R^2 = .07$ $B = -20.1$	$t(13) = 3.71$ $p \leq .01$ $R^2 = .51$ $B = 66.9$	$t(11) = 3.20$ $p \leq .01$ $R^2 = .48$ $B = 48.3$
Minute 3	$t(14) = -0.75$ $p = .46$ $R^2 = .04$ $B = -14.2$	$t(13) = 2.99$ $p \leq .01$ $R^2 = .41$ $B = 69.4$	$t(11) = 2.50$ $p = .03$ $R^2 = .36$ $B = 44.0$
Minute 4	$t(14) = -0.03$ $p = .97$ $R^2 = .00$ $B = -0.70$	$t(13) = 3.00$ $p \leq .01$ $R^2 = .41$ $B = 64.9$	$t(11) = 1.79$ $p = .10$ $R^2 = .23$ $B = 37.7$
Minute 5	$t(14) = 0.31$ $p = .76$ $R^2 = .01$ $B = 5.66$	$t(13) = 3.52$ $p \leq .01$ $R^2 = .49$ $B = 77.6$	$t(11) = 1.57$ $p = .15$ $R^2 = .18$ $B = 28.9$

in none of the three groups did daily cigarette consumption correlate (at the $p \leq .05$ level) with changes in RSA, changes in heart period, or initial heart period.

Although the mechanism for the altered relationship between RSA and heart period in perpetrators is not known, several possible mechanisms could contribute to this dysregulation. First, the response characteristics of the sinoatrial node may be different. These differences could result from either receptor changes or from the interaction between sympathetic and parasympathetic nerves that influence the sinoatrial node (Levy & Martin, 1998; Salata & Zipes, 1991). Second, there may be differences in sympathetic nervous system activity. In the controls, there was a trend towards a relationship between the change in heart period and the change in epinephrine and norepinephrine (implying a more organized autonomic nervous system with coordinated sympathetic and vagal activity). However, in the perpetrators this trend was absent. Third, there may be differences in parasympathetic activity not reflected by RSA, that is, from fibers arising from the dorsal motor nucleus of the vagus (Porges, 1995, 1997; Standish, Enquist, & Schwaber, 1994), or the potential influence of vagal fibers from the nucleus ambiguus that do not have respiratory rhythm (Reed, Ohel, David, & Porges, 1999). Fourth, the trend towards a greater diastolic blood pressure response in perpetrators suggests that there may be altered central regulation of the baroreceptor reflex. This reflex is modulated by the hypothalamus. Interestingly, electrical stimulation of the medial hypothalamus in the cat inhibits this reflex concomitant with the onset of a rage reaction (i.e., raised fur, broad-sided stance, arched back, loud vocalizations, unsheathing of the claws; Mifflin, Spyer, & Withington-Wray, 1988; Spyer, 1989; Wasman & Flynn, 1962). We believe this constellation of symptoms in the cat is analogous to the intimidating behaviors displayed by perpetrators at the time of domestic violence. If this is the case, it is conceivable that pathways mediating their aggressive behavior are chronically dysregulated, and interfere with normal baroreceptor function even when perpetrators do not outwardly appear enraged.

Our results suggest that during an orthostatic challenge, perpetrators have an altered ability to systematically modulate heart rate via vagal pathways originating in the nucleus ambiguus. This could parallel an altered ability of the nucleus ambiguus to regulate its innervation of other key structures involved with the expression of emotion and rage, namely the larynx, pharynx, esophagus, and bronchi (Porges, 1998; Richter & Spyer, 1990). The nucleus ambiguus, with its central connections, may facilitate the appropriate expression of emotion in response to changing social interactions by enabling "rapid" and "efficient" shifts in mood and behavior (Porges, 1997, 1998; Porges, Doussard-Roosevelt, & Maiti, 1994).

In summary, perpetrators of domestic violence showed a different neural strategy in the regulation of heart rate during a posture shift that warrants further investigation. In assessing these results, it is important to emphasize that domestic violence is the culmination of a complex set of interactions between the perpetrator, the victim, the environmental circumstances and cultural mores in which the violence takes place. Future studies may confirm that altered heart rate regulation provides a measurable index of autonomic dysregulation, which, in turn, may be found to affect the perpetrators' ability to modulate emotion and to control aggression.

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