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Borderline Personality Disorder and Emotion Regulation:

Insights from the Polyvagal Theory

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Abstract

The current study provides the first published evidence that the parasympathetic component of the autonomic nervous system differentiates the response profiles between individuals diagnosed with Borderline Personality Disorder (BPD) and controls. Respiratory sinus arrhythmia (RSA), a non-invasive marker of the influence of the myelinated vagal fibers on the heart, and heart period were collected during the presentation of film clips of varying emotional content. The BPD and control groups had similar initial levels of RSA and heart period. However, during the experiment the groups exhibited contrasting trajectories with the BPD group decreasing RSA and heart period and the control group increasing RSA and heart period. By the end of the experiment, the groups were significantly different on both RSA and heart period. The correlation between the changes in RSA and heart period were significant only for the control group, suggesting that vagal mechanisms mediated the heart period responses only in the control group. The findings were interpreted, consistent with the Polyvagal Theory (Porges, 1995, 2001, 2003), as illustrating different adaptive shifts in autonomic state through the course of the experiment with the BPD group ending in a physiological state that supports the mobilization behaviors of fight and flight and the control group ending in a physiological state that supports social engagement behaviors. These finding are consistent with other published studies demonstrating atypical vagal regulation of the heart with other psychiatric disorders.

Borderline Personality Disorder and Emotion Regulation:

Insights from the Polyvagal Theory

The concept of a "borderline personality disorder" dates back to the early 1800s, when clinicians were unsure of the diagnosis of patients who displayed a combination of neurotic and psychotic symptoms. Since clinicians viewed these patients as being on the "border" between neurotic and psychotic, the borderline personality disorder (BPD) evolved as a diagnostic category and was listed as an Axis II diagnosis in 1980 with the publication of *DSM-III* (Hodges, 2003). The current *DSM-IV-TR* emphasizes that patients with BPD express symptoms that include affective instability, intense and tumultuous relationships, difficulty controlling anger, impulsivity, suicidal tendencies, and self-mutilation (American Psychiatric Association, 2000; Rothschild, Haslam, Cleland, & Zimmerman, 2003). This cluster of symptoms indicates that BPD is associated with difficulty in regulating emotions, behavioral states, and relationships. BPD is a severe mental disorder that is more prevalent in women and is believed to impact approximately 1% of the population (American Psychiatric Association, 2000; Hodges, 2003; Torgerson, Kringlen, & Cramer, 2001).

Because BPD is associated with problems in regulating emotions and appropriately responding to daily life events, BPD has been linked with a wide variety of poor outcomes including job-related problems (Zweig-Frank & Paris, 2002), dysfunction in developing strong personal relationships (Daley, Burge, & Hammen, 2000), social maladjustment, and reduced academic achievement (Bagge, Nickell, Stepp, Durrett, Jackson, & Trull, 2004). Due, in part, to the breadth and severity of these frequently observed problems, BPD has been difficult to treat effectively (Hoffman, Buteau, Hooley, Fruzzetti, & Bruce, 2003).

The high correlation between BPD and past sexual abuse and family dysfunction (Weaver & Clum, 1993) has led to the developmental hypothesis that BPD may develop as a result of traumatic experiences early in life. Other adverse events, such as abandonment or fear of abandonment and lack of a secure emotional attachment with a caregiver often accompany BPD (Benjamin, 1996; Gunderson, 1996). Furthermore, BPD is highly comorbid with other mood and anxiety disorders (Hodges, 2003; Skodol, Gunderson, Pfohl, Widiger, Livesley, & Siever, 2002; Weaver & Clum, 1993).

Despite the prevalence and severity of BPD, few studies have investigated the underlying neurological and physiological mechanisms of the disorder (e.g., Schmahl, Elzinga, Ebner, Simms, Sanislow, Vermetten, McGlashan, & Bremner, 2004). Coccaro and Kavoussi (1991) suggested that an improved understanding of the neurological and physiological mechanisms mediating the clinical symptoms of BPD might lead to the development of more effective treatments. During the past decade, research has begun to identify specific neurobiological features that differentiate between BPD and controls. These features might provide clues to the mechanisms mediating the difficulties in emotion regulation experienced by individuals diagnosed with BPD.

Since impulse control is a characteristic deficit associated with BPD, dysfunction of prefrontal cortex has been hypothesized to be a mediator of BPD. This speculation is based on observations of increased impulsivity following brain damage in prefrontal areas (Blair & Cipolott, 2000). Consistent with this hypothesis, individuals with BPD perform poorer on a go/no-go task, a test of impulse control assumed to evaluate prefrontal function (Völlm, Richardson, Stirling, Elliott, Dolan, Chaudhry, Del Ben, McKie, Anderson, & Deakin, 2004). In

addition, volumetric studies applying imaging techniques have found smaller frontal lobes in BPD participants (Lyoo, Han, & Cho, 1998).

Imaging has also identified in individuals with BPD anomalies in limbic structures involved in emotion regulation, such as smaller hippocampal and amygdala volumes (Tebartz van Elst, Freiburg, Hesslinger, Thiel, Geiger, Haegele, Lemieux, Lieb, Bohus, Hennig, & Ebert, 2003). Volumetric reductions, especially in the hippocampus, are thought to be caused by the excessive stress that BPD patients experience (Schmahl, Vermetten, Elzinga, & Bremner, 2003). Since the hippocampus and the amygdala are involved in the processing of and responding to emotional stimuli (Anderson & Phelps, 2000; Nolte, 1993), the consequences of the volumetric reductions might be related to the difficulties in emotion regulation that BPD individuals experience.

Other neurophysiological systems involved in behaviors such as emotion regulation, impulsivity, and aggressive behavior have been studied. Abnormalities in serotonin, a neurotransmitter linked to aggression, impulsivity, and suicidal behavior (Coccaro, 1989) has been reported in BPD (Hansenne, Pitchot, Pinto, Reggers, Scantamburlo, Fuchs, Pirard, & Ansseau, 2002; New & Siever, 2002; Paris, Zweig-Frank, Ng, Schwartz, Steiger, & Nair, 2004; Skodol, Siever, Livesley, Gunderson, Pfohl, & Widiger, 2002). BPD may be associated with a hyperresponsiveness of the hypothalamic-pituitary-adrenal system (Rinne, de Kloet, Wouters, Goekoop, ReRijk, & van den Brink, 2002), a system involved in stress responses, anxiety, and emotional reactivity. These findings provide limited evidence that accompanying BPD is a dysfunction in systems involved in controlling reactivity and emotion.

Since several features of BPD are related to difficulties in regulating behavioral state and emotional reactivity, the autonomic nervous system might be involved. Thus, it might be

hypothesized that: (a) the sympathetic component of the autonomic nervous system, which supports fight/flight behaviors, would be hyperaroused; and (b) the parasympathetic component, which supports calm visceral states and social engagement behaviors, would be depressed. Previous research contrasted the sympathetic nervous system responses (heart rate, skin conductance, startle response) of individuals with BPD to controls, while viewing stimuli of varying emotional valence (pleasant, neutral, unpleasant) and intensity (Herpertz, Kunert, Schwenger, & Sass, 1999). Also, Schmahl et al. (2004) measured sympathetic variables (heart rate, skin conductance, blood pressure) in response to reminders of personal experiences of severe stress (i.e., abandonment, trauma). These studies found no evidence of sympathetic hyperarousal in the BPD patients, although no research has addressed the parasympathetic component.

The phylogenetic model of the autonomic nervous system, described in the Polyvagal Theory (see Porges, 1995, 1997, 2001, 2003), provides an innovative theoretical framework of how the parasympathetic nervous system may be involved in mediating some of the features of BPD. The Polyvagal Theory emphasizes how neural circuits involved in the regulation of autonomic state evolved to support various adaptive biobehavioral responses to challenges. The theory proposes that autonomic reactions to challenges follow a phylogenetically ordered hierarchy with three distinct adaptive biobehavioral strategies. Each biobehavioral strategy reflects a specialized neurophysiological substrate that evolved to maximize adaptive strategies in different contexts which are safe, dangerous, or life-threatening. Within this model the nervous system, through a process of "neuroception," is continuously evaluating the environment for risks and safety. Neuroception is not be a conscious process, but is proposed to occur via unconscious and subcortical systems that functionally trigger one of these three adaptive neural

circuits. Therefore, problems of emotional reactivity could be, within the Polyvagal Theory, be interpreted as a behavioral expression of a physiological state that evolved to support defensive strategies triggered to deal adaptively with dangerous and life-threatening situations. According to the Polyvagal Theory, the myelinated vagus, which phylogenetically evolved with mammals, is necessary to inhibit defensive limbic circuits and critical for the establishment of social bonds (Porges, 2003).

Phylogenetically, as mammals expressed special visceral efferent pathways to regulate the striated muscles of the face and head (e.g., facial expressions, head gesture), there was a parallel shift in the neural regulation of the heart from an unmyelinated to a myelinated vagus. This new or mammalian vagus actively inhibits the sympathetic nervous system influences on the heart and dampens HPA-axis activity (Porges, 2001). The mammalian vagus functions as an active vagal brake (see Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996) in which rapid inhibition and disinhibition of the vagal tone to the heart can rapidly mobilize or calm an individual. Thus, BPD might be associated with difficulties in regulating the vagal brake in social settings.

The mammalian heart is characterized by a relatively strong vagal influence, via the myelinated pathways, on the heart's pacemaker (i.e., sino-atrial node). Functionally, this results a baseline or resting heart rate substantially lower than the intrinsic rate of the pacemaker. When the vagal tone to the pacemaker is high, the vagus acts as a restraint or brake limiting the rate the heart is beating. When vagal tone to the pacemaker is low, there is little or no inhibition of the pacemaker. Thus, the vagal brake may be used as a construct to describe the functional modulation of heart rate by the myelinated vagal efferent pathways.

The vagal brake provides a neural mechanism to rapidly change visceral state by slowing or speeding heart rate. Neurophysiologically the vagal brake is removed or reduced to support the metabolic requirements for mobilization (e.g., associated with fight/flight behaviors) and maintained or increased to support social engagement behaviors. The state of the vagal brake can be assessed by quantifying the amplitude of respiratory sinus arrhythmia (RSA). RSA is a naturally rhythm in the heart rate pattern at approximately the frequency of spontaneous breathing. The amplitude of RSA can be quantified to provide a sensitive index of the impact of the myelinated vagus on the heart (Porges, 1995). By assessing RSA during various challenges it is possible to measure the dynamic regulation of the vagal brake. In this study, we contrast the regulation of the vagal brake in participants with BPD and controls during the presentation of film clips assumed to reflect emotional content. In addition, the experiment, by requiring an interaction between the participant and the experimenter, provides a secondary context related to social interactions.

Method

Participants

Participants (all female) consisted of 9 borderline personality disorder (BPD) patients and 11 controls between the ages of 18 and 45. The groups were equivalent in education level and age. BPD participants were referred to the study by clinicians in the Washington, D.C. area. The control group consisted of normal volunteers, who were recruited from lists maintained by the National Institutes of Health. Both groups were free of drug and alcohol abuse and were not current users of prescription and illicit drugs. The diagnosis of BPD, based on the criteria of the DSM-IV (American Psychiatric Association, 1994), was confirmed by the Structured Clinical Interview for DSM-III-R (SCID) (Spitzer, Williams, Gibbon, & First, 1990; 1992) and the

Diagnostic Interview for Borderlines (Gunderson, Kolb, & Austin, 1981). Controls were free of psychiatric or neurological disorders. The BPD participants were tested at St. Elizabeth's Hospital (Washington, D. C.). Although the BPD participants did not require inpatient hospitalization, they were electively in residence at the hospital while participating in this study. The control participants were not in residence and were tested either at the hospital or at the Developmental Assessment Laboratory (University of Maryland, College Park) in similar testing environments.

Procedure

Following informed consent, participants were seated in a quite room facing a television screen. To monitor ECG, from which the heart period and RSA were derived, three disposable Ag-AgCl electrodes were placed on the participant's chest and connected to an ECG preamplifer and output to a Vagal Tone Monitor-II (Delta-Biometrics, Inc.). After the initial baseline period, participants completed a brief questionnaire. Participants were instructed to watch a 10-minute film clip, which was followed by answering several questions pertaining to the film clip just viewed. Two additional 10-minute film clips were shown and each was followed by questions specific to the preceding film clip. Physiological data were collected during each of the four 10-minute segments (i.e., baseline, film clip 1, film clip 2, film clip 3). To allow for transition in physiological state due to the onset of each film clip, the last five minutes of data within each of the four conditions were analyzed to maximize the emotional content of film clips. The experiment lasted approximately 1 hour. Heart period and RSA were collected for the baseline and film clip conditions. Participants were instructed to minimize movements during the experiment.

Film clips 1 and 3 were selected to elicit a strong emotional response. Film clip 1 was a conflict scene with the mother in *Frances*, film clip 3 was a conflict scene with the father in *The Great Santini*. In contrast to these two conflict scenes, film clip 2 was selected to be a neutral scene from *A Handful of Dust.* Participants rated the film clips on a Likert type scale ranging from 0 (not arousing) to 10 (extremely arousing). The rating confirmed the investigators intention in selecting the specific film sequences. The first conflict scene had a mean rating of 7.25 and the second conflict scene had a mean rating of 9.15. In contrast, the neural scene had a mean rating of 1.75. The conflicts scenes were rated as significantly more arousing than the neural scene ($p < .001$). There were no differences in ratings between groups.

Data Quantification

The Vagal Tone Monitor detected the peak of the R-wave with 1-ms accuracy, timed sequential heart periods to the nearest millisecond (Riniolo & Porges, 1997), and stored the heart periods in files for off-line analyses of RSA and heart period. The data files of sequential heart periods (i.e., R-R intervals in milliseconds) were input into MXedit (version 2.21) software (Delta-Biometrics) to edit outlier data produced by movement and digitizing error. Editing consisted of integer addition or division of sequential values.

RSA estimates were calculated using the following procedures (Porges, 1985): (a) the heart period time series were converted to time-based data by resampling at successive 500-msec intervals; (b) a 21-point moving cubic polynomial filter was stepped through the time-sampled series to produce a smoothed template series; (c) the template series was subtracted from the original series to produce a residual time series; (d) the residual time series was processed by a digital bandpass filter with 25 coefficients to extract the variance in the frequency band .12-.40 Hz (i.e., the frequency of spontaneous breathing for adults); and (e) the bandpassed variance was

transformed to its natural logarithm and used to quantify RSA. These procedures result in a sensitive non-invasive marker of the influence of the myelinated vagal fibers on the heart (Porges, 1995; Porges, 2001).

Analyses

Group (Borderline, Control) by Condition (Baseline, Film 1, Film 2, Film 3) analyses of variance (ANOVA) were used to identify statistical effects for RSA and heart period. Data from one participant during the baseline condition was lost due to a technical error. Heart period is calculated as the time interval in msec between successive R-waves of the ECG. The metric for the current analyses represents the average R-R interval in msec for each condition. To evaluate the vagal contribution to the heart period changes during each of the Film clips, correlations between the changes from baseline in heart period and RSA were calculated. High correlations would illustrate that the changes in both variables were mediated by a common mechanism (i.e., vagal regulation of the heart). If the heart period change is totally dependent on vagal regulation, the correlation with RSA should approach 1. In contrast, low correlations would suggest that heart period was not tightly regulated by the vagus and would be mediated by other mechanisms.

Results

Collapsed across conditions there was a significant Group effect for RSA, $F[1,77] = 7.16$, $p < .05$. The Control group had significantly higher RSA than the BPD group. This main effect was functionally determined by the Group trajectories during the experiment and statistically represented in a significant Group X Condition interaction, $F[3, 51] = 3.62$, $p < .05$. As illustrated in Figure 1A, the trajectory of RSA during the experimental session differed between the groups. The values for RSA are similar for the two groups during baseline. However, over the course of the experiment, the control participants exhibit an increase in RSA and the BPD

participants exhibit a decrease in RSA. These distinct patterns reflect a different neural strategy with the BPD participants exhibiting a vagal withdrawal that would support the increased metabolic demands of fight/flight behaviors and the Control participants exhibiting an increased in vagal influences to heart consistent with a calm physiological state that would support social engagement behaviors. As illustrated in Figure 1B, there was a statistically significant Group X Condition interaction for heart period, $F[3,51] = 6.49$, $p<.05$. The heart period response pattern during the experiment confirms the impact of the shift in cardiac vagal tone with the BPD group progressively exhibiting shorter heart periods (i.e., faster heart rate) and the Control group exhibiting longer heart periods. Simple effects tests (see table 1) confirm that at the end of the experiment, group differences are pronounced for both variables. Consistent with the RSA data, collapsed across conditions there was a significant Group effect for heart period, $F[1,77] = 14.2$, $p < .05$.

The vagal contribution to the heart period changes during the experiment was estimated by correlating the change from baseline in heart period with change from baseline in RSA. Correlations were calculated within each group to evaluate whether the observed heart period responses were under vagal control. As illustrated in Figure 2, the relations between changes in RSA and heart period are significantly correlated only for the Control group. Thus, the changes in heart period are strongly linked to an increase in vagal control of the heart only in the Control group. In the BPD group, although there is a consistent vagal withdrawal, the changes in RSA are not sufficient to account for the changes in heart period. These findings suggest that either the BPD participants have poor vagal regulation or that the changes in heart period may be due to other influences, such as sympathetic activation.

Discussion

Previous investigations of the autonomic nervous system of BPD participants focused on indices of the sympathetic nervous system. These studies did not identify differences between BPD and controls (Herpertz, Kunert, Schwenger, & Sass, 1999; Schmahl, Elzinga, Ebner, Simms, Sanislow, Vermetten, McGlashan, & Bremner, 2004). In contrast, the current study, by focusing on the parasympathic limb of the autonomic nervous system, provides the first report of differences in autonomic regulation between BPD and controls.

Based upon a limited literature with PTSD subjects (Sahar, Shalev, & Porges, 2001), we assumed that BPD patients and controls would have similar levels of RSA during baseline. This was confirmed. By challenging the participants with film clips of both high (clips 1 and 3) and low (clip 2) emotional content, the experiment was designed to elicit a withdrawal of the vagal brake (i.e., lower RSA and shorter heart period) in both groups to the high emotion stimuli and a return to baselevel for the low emotion stimulus. However, we anticipated that the BPD group, due to their sensitivity to affective stimuli and difficulty in state regulation, would exhibit an exaggerated withdrawal of the vagal brake (i.e., lower RSA and shorter heart period). Interestingly, neither group exhibited a film clip related response. Instead as illustrated in Figures 1A and 1B, there was a trajectory of increasing vagal influences on the heart for the control group and decreasing vagal influences on the heart for the BPD group.

At the start of the experiment vagal influences on the heart were similar in both groups. However, as the experiment progressed, there was an intriguing divergence between the groups. The Polyvagal Theory provides two insights into the adaptive nature of the physiological state for each group at the end of the experiment. First, the Polyvagal Theory emphasizes that the physiological states, which characterized the two groups at the end of the experiment, support different classes of behavior. For the BPD group the physiological state, characterized by a

vagal withdrawal, would support the mobilization behaviors of fight and flight. In contrast, for the Control group the physiological state, characterized by increased vagal influence on the heart, would support spontaneous social engagement behaviors. Interestingly, imbedded in the diagnosis of BPD are features related to compromised social engagement behaviors, including a hyper-reactivity or emotional dysregulation during social interactions. Thus, although the experimental conditions provided the same context and task demands for all subjects, the BPD group reacted with a visceral state to promote defensive behaviors, while the f the Control group reacted with a visceral state to promote increase spontaneous social engagement behaviors. Second, the Polyvagal Theory (Porges, 2004) proposes a mechanism, neuroception, to trigger defense strategies. Based on the clinical features of BPD, social interactions with a novel individual (i.e., the experimenter) might provide a social stimulus to trigger to the nervous system and to elicit a physiological state that would support fight and flight behaviors. Alternatively, the film clips with the depictions of social interactions might have contributed to the need to mobilize. It would be interesting to speculate whether in the absence of a person in the experimental room, the physiological states of the two groups would diverge during the film clips. Perhaps, both groups would have been more similar throught the experiment in the absence of the human experimenter. The BPD group might have maintained a stable autonomic state, because in the absence of the human experimenter the nervous system would not detect risk and elicit a defensive strategy. Also, the Control group might have maintained stable autonomic state, because in the absence of the same human experimenter the nervous system would not detect the cues to trigger spontaneous social engagement behaviors.

The correlations between RSA and heart period provide additional support for the hypothesis that in individuals without psychiatric disturbances, there is a strong link between

vagal regulation and the control of heart period. The lack of correlation between RSA and heart period for the BPD group is consistent with other studies demonstrating atypical vagal regulation of the heart in other with psychiatric disorders (Sahar, Shalev, & Porges, 2001; Umhau, George, Reed, Petrulis, Rawlings, & Porges, 2002) characterized by difficulties in regulating behavioral and psychological state.

One interpretation is that the nervous system of BPD participants does not accurately evaluate whether environments are safe or pose a threat. Thus, rather than being calm in the presence of another "nonthreatening" human, the autonomic nervous system is regulated to a state that supports fight and flight and not a state that would support spontaneous social engagement. This may partially explain why BPD patients have a variety of poor outcomes, especially related to social relationships and emotional instability in real world situations.

In summary, this study provides the first documented evidence of autonomic nervous system differences between controls and individuals diagnosed with BPD, and provides a theoretical framework to explain the emotional reactivity that is linked with the disorder. The study, however, has several limitations, including small sample size, an isolated experimental manipulation, and no measures of test-retest reliability. However, this investigation into the autonomic response profile of BPD patients may lead to research using RSA, as a noninvasive marker of the vagal brake, in a variety of social challenges to confirm the above speculations. Monitoring RSA during social challenges may provide a measurable index of neuroception in BPD and provide a method to delineate the features in the social environment that would trigger autonomic states that support defensive behaviors.

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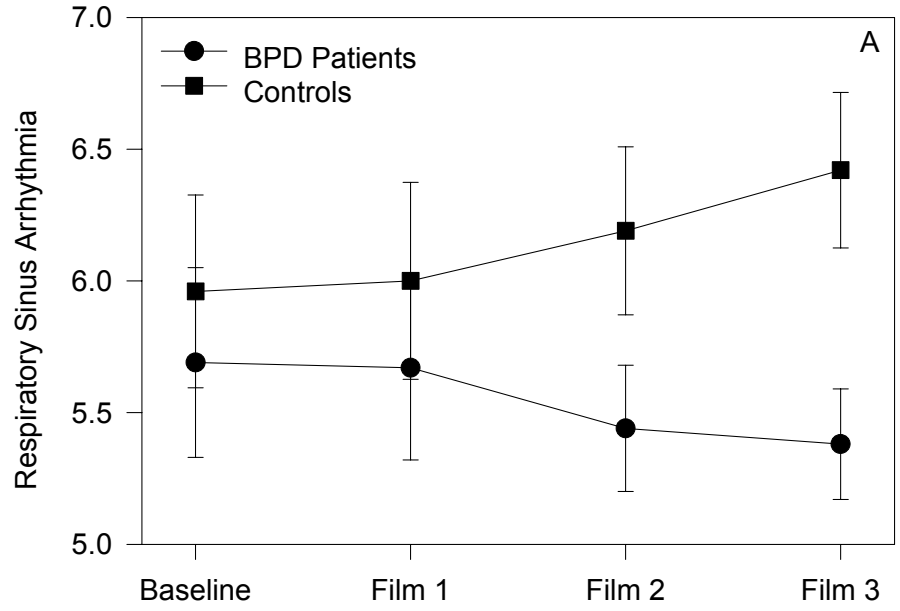
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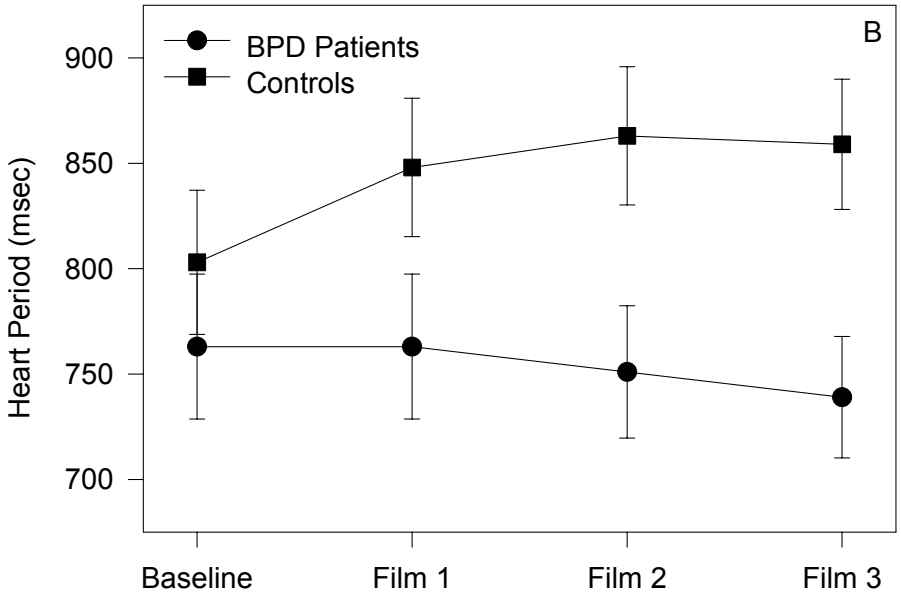
Table 1

Simple Effects for ANOVA

	BPD Patients (n = 9)	Controls (n = 11)	t-value	df	p-value
Respiratory Sinus Arrhythmia					
Baseline	[a]5.69 (1.0)	5.96 (1.2)	- .520	17	=.6098
Film 1	5.67 (1.1)	6.00 (1.2)	- .636	18	=.5329
Film 2	5.44 (.72)	6.19 (1.1)	-1.81	18	=.0867
Film 3	5.38 (.64)	6.42 (.98)	-2.74	18	=.0134*
Heart Period (msec)					
Baseline	762 (123)	803(114)	- .748	17	=.4645
Film 1	763 (103)	848 (121)	-1.66	18	=.1134
Film 2	751 (94)	863 (109)	-2.43	18	=.0256*
Film 3	739 (86)	859 (103)	-2.78	18	=.0123*

[a]Means (and standard deviations); *statistically significant $p < .05$


A
BPD Patients
Controls
Respiratory Sinus Arrhythmia
7.0
6.5
6.0
5.5
5.0
Baseline
Film 1
Film 2
Film 3


B
BPD Patients
Controls
Heart Period (msec)
900
850
800
750
700
Baseline
Film 1
Film 2
Film 3

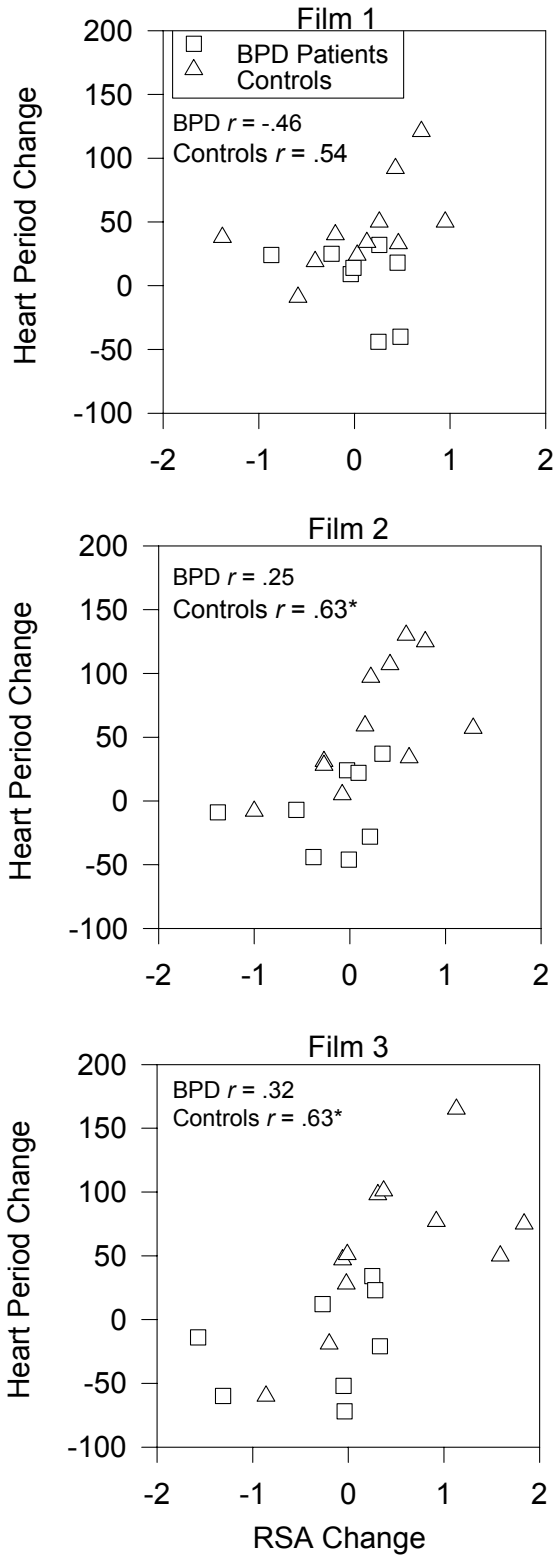

Film 1
BPD Patients
Controls
BPD r = -.46
Controls r = .54
Heart Period Change
200
150
100
50
0
-50
-100
-2
-1
0
1
2
Film 2
BPD r = .25
Controls r = .63*
Heart Period Change
Film 3
BPD r = .32
Controls r = .63*
Heart Period Change
RSA Change

Figure Captions

Figure 1: Means (±SE) for Respiratory Sinus Arrhythmia (Panel A) and Heart Period (Panel B) across test conditions.

Figure 2: Correlations of the change from baseline in Heart Period with change from baseline in Respiratory Sinus Arrhythmia from films 1, 2, and 3. *$p < .05$.