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 differ significantly on both RSA and heart period. The correlation between the changes in RSA and heart period was significant only for the control group, suggesting that vagal mechanisms mediated the heart period responses only in the control group. The findings were consistent with the Polyvagal Theory [Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage: A Polyvagal Theory. Psychophysiology, 32, 301–318; Porges, S. W. (2001). The Polyvagal Theory: Phylogenetic substrates of a social nervous system. International Journal of Psychophysiology, 42, 123–146; Porges, S. W. (2003). Social engagement and attachment: A phylogenetic perspective. Annals of the New York Academy of Sciences, 1008, 31–47.], illustrating different adaptive shifts in autonomic state throughout the course of the experiment. The BPD group ended in a physiological state that supports the mobilization behaviors of fight and flight, while the control group ended 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.
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1. Introduction

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 2% of the population (American Psychiatric Association, 2000; Hodges, 2003; Swartz, Blazer, George, & Winfield, 1990; Torgersen, Kringlen, & Cramer, 2001)

and approximately 20% of the hospitalized psychiatric patients (Zanarini & Grankenbrug, 2001).

Because BPD is associated with problems in regulating emotions and responding appropriately to daily life events, BPD has been linked to 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 et al., 2004). Due 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 reported 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 et al., 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 et al., 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 the 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 et al., 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 implicated in emotion regulation, such as smaller hippocampal and amygdala volumes (Tebartz van Elst et al., 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). Because the hippocampus and the amygdala are involved in the processing of and responding to emotional stimuli (Anderson & Phelps, 2000; Nolte, 1993), a consequence of volumetric reductions

might be related to the difficulties in emotion regulation that BPD individuals experience.

Other neurophysiological systems mediating processes 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) have been reported in individuals diagnosed with BPD (Hansenne et al., 2002; New & Siever, 2002; Paris et al., 2004; Skodol et al., 2002). BPD may be associated with a hyperresponsiveness of the hypothalamic–pituitary–adrenal system (Rinne et al., 2002), a system implicated in stress responses, anxiety, and emotional reactivity. These findings provide limited evidence that a dysfunction in systems involved in controlling reactivity and emotion accompanying BPD.

Because several features of BPD are related to difficulties in regulating behavioral state and emotional reactivity, measurement of the autonomic nervous system might provide a portal into understanding the neural mechanisms of this disorder. 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 (for detailed review, see Herpertz, Kunert, Schwenger, & Sass, 1999; Schmahl et al., 2004) contrasted physiological responses regulated by the sympathetic nervous system in individuals with BPD and controls. Herpertz et al. (1999) monitored heart rate, skin conductance, and startle responses in a paradigm varying the emotional valence (pleasant, neutral, and unpleasant) and intensity of visual stimuli. Schmahl et al. (2004) measured heart rate, skin conductance, and blood pressure in response to reminders of personal experiences of severe stress (i.e., abandonment, trauma). Neither study found evidence of sympathetic hyperarousal associated with a diagnosis of BPD. However, both studies did not monitor the parasympathetic component of the autonomic nervous system or expose BPD participants to dynamically changing emotional stimuli (e.g., film clips).

The phylogenetic model of the autonomic nervous system described in the Polyvagal Theory (see Porges, 1995, 1997, 2001, 2003), provides an innovative theoretical framework to study the potential involvement of the parasympathetic nervous system in BPD. The theory focuses on the role that autonomic state plays in mediating both prosocial and defensive behaviors. The theory emphasizes an integrated Social Engagement System that regulates the muscles of the face and head involved in social engagement behaviors (e.g., gaze, expression, prosody, and gesture) and a component of the parasympathetic nervous system, the myelinated vagal pathways to the heart that calm visceral state and dampen sympathetic and HPA activity. 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 fol-

low 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 safe, dangerous, or life-threatening contexts. Within this model the nervous system, through a process of ''neuroception,'' is continuously evaluating risk and safety in the environment. Neuroception is not a conscious process, but rather it occurs via unconscious subcortical systems that functionally trigger one of these three adaptive neural circuits. Therefore, based on the Polyvagal Theory, difficulties in emotional regulation that are associated with a diagnosis of BPD could be interpreted as a behavioral expression of a physiological state that has evolved to support defensive strategies in dangerous and life-threatening situations. According to the Polyvagal Theory, the myelinated vagus, which phylogenetically evolved with mammals, is critical for two reasons: to inhibit defensive limbic circuits, and to establish 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 myelinated (i.e., mammalian) vagus actively inhibits the sympathetic nervous system's influence 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) to maintain calm states in social contexts. However, when risk is detected, the vagal brake can be rapidly withdrawn to support defensive mobilization behaviors. 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, the impact of the vagal brake produces a baseline or resting heart rate substantially lower than the intrinsic rate of the pacemaker. When the vagal brake is removed, heart rate can approximate the intrinsic rate of the pacemaker without recruiting sympathetic influences. When cardiac vagal tone via the myelinated vagus 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 change visceral states by slowing or speeding heart rate. Neurophysiologically, the influence of vagal brake is reduced or removed to support the metabolic requirements for mobilization (e.g., fight/flight behaviors) and maintained or increased to support social engagement behaviors. The amplitude of respiratory sinus arrhythmia (RSA) indexes the state of the vagal brake. RSA is a natural rhythm in the heart rate pattern at approximately the frequency of spontaneous breathing. The amplitude of RSA provides a sensitive index of the impact that the myelinated vagus has on the heart (Porges, 1995). By quantifying RSA during various challenges, it is possible to measure the dynamic regulation of the vagal brake.

It was hypothesized that the BPD participants, unlike typically behaving individuals, will have difficulties maintaining the vagal brake in social contexts. Thus, in response to social stimuli, BPD participants should rapidly shift from a calm state (i.e., high amplitude RSA) to a state of agitation (i.e., low amplitude RSA). To test this hypothesis, we contrasted the regulation of the vagal brake by measuring the amplitude of RSA 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.

2. Method

2.1. Participants

Participants (all female) consisted of 9 borderline personality disorder (BPD) patients and 11 controls between the ages of 18 and 45. Only females were recruited, since females represent the majority of individuals diagnosed with BPD and to remove gender as a possible source of variance in the neurophysiological regulation of autonomic state. The groups were equivalent in education level and age. BPD participants were referred to the study by clinicians in the Washington, DC area. The BPD participants were identified and screened to eliminate co-morbid diagnoses by NIMH clinical researchers at St. Elizabeth's Hospital (Washington, DC). The control group consisted of 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 or illicit drugs. The diagnosis of BPD, based on DSM-IV criteria (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). The BPD participants were tested at St. Elizabeth's Hospital. The BPD participants, while participating in the study, were off medication and as a precautionary procedure were in residence at the hospital. Controls were free of psychiatric or neurological disorders. The control participants were not in residence at the hospital and were tested either at the hospital or at the Developmental Assessment Laboratory (University of Maryland, College Park) in similar testing environments.

2.2. Procedure

Following informed consent, participants were seated in a quiet 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, connected to an ECG amplifier, and output to a Vagal Tone Monitor-II (Delta-Biometrics, Inc.). After the initial baseline period, participants were instructed to watch three 10-min film clips. Each film clip was followed by questions specific to the film clip just viewed. Physiological data were collected during each of the four 10-min segments (i.e., baseline, film clip 1, film clip 2, film clip 3). To facilitate the measurement of a stable indicator of autonomic state in response to the specific emotional content of each film clip and to allow for a transition in physiological state due to the onset of each film clip, the last 5 min of data within each of the four conditions were analyzed. The experiment lasted approximately one hour. Heart period and RSA were collected for the baseline and film clip conditions. Participants were instructed to minimize their movements during the experiment. The experimenter remained in the room with the participant during the experimental procedures, since BPD is associated with an inability to tolerate being alone (see Gunderson, 1996). While in the experimental room, the experiment ran the equipment and asked questions about each film clip.

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*, and 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 scale ranging from 0 (not arousing) to 10 (extremely arousing). Participants' ratings confirmed the assumed emotional content of each film sequence. 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 neutral scene had a mean rating of 1.75. The conflict scenes were rated as significantly more arousing than the neutral scene ($p < .001$). There were no significant differences in ratings between groups.

2.3. Data quantification

The Vagal Tone Monitor detected the peak of the R-wave with 1-ms accuracy, timed sequential heart periods to the nearest ms (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 ms) were input into MXedit (version 2.21) software (Delta-Biometrics) in order 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-ms 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 of .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. Data from our laboratory (Denver, in press) demonstrate that this methodology accurately captures the heart rate variability associated with spontaneous breathing. Denver (in press) report a correlation of .99 between the frequency observed in the heart rate and respiration spectra. Moreover, the amplitude of the heart rate periodicity derived from spectral analysis was correlated .99 with the values derived from the MXedit analysis. These procedures result in a sensitive, non-invasive marker of the influence of the myelinated vagal fibers on the heart (Porges, 1995, 2001).

2.4. 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 were lost due to technical error. Data from this participant were analyzed during the experimental conditions. Heart period was calculated as the time interval in ms between successive R-waves of the ECG. The metric for the current analyses represents the average R–R interval in ms for each condition. To evaluate the vagal contribution to 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 1.0. In contrast, low correlations would suggest that heart period is not tightly regulated by the vagus and would be mediated by other mechanisms.

3. Results

Across all 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 the Group × Condition interaction, $F(3, 51) = 3.62$, $p < .05$. As illustrated in Fig. 1a, the trajectory of RSA during the experimental session differed between the groups. The values for RSA were similar for the two groups during baseline. However, over the course of the experiment, the control participants exhibited an increase in RSA, and the BPD participants exhibited a decrease in RSA. These distinct patterns reflect different neural strategies. The BPD participants exhibited a vagal withdrawal, which
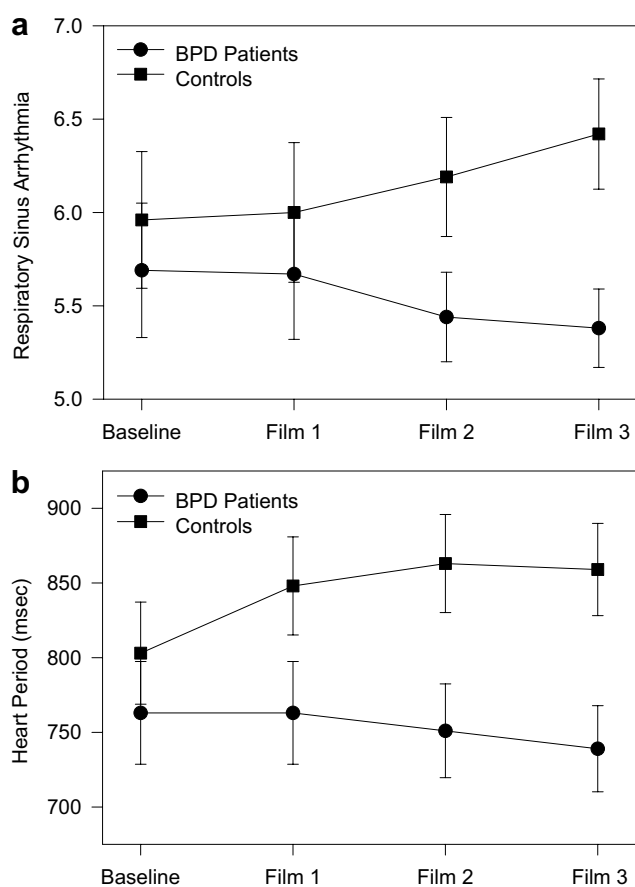
Fig. 1. Means (±SE) for respiratory sinus arrhythmia (a) and heart period (b) across test conditions.

would support the increased metabolic demands of fight/flight behaviors. The control participants exhibited an increase in vagal influences to heart, which would support social engagement behaviors. As illustrated in Fig. 1b, there was a significant Group × Condition interaction for heart period, $F(3,51) = 6.49$, $p < .05$. The heart period response pattern during the experiment confirms the observed group specific shifts in cardiac vagal tone. The BPD group progressively exhibited shorter heart periods

(i.e., faster heart rate), while the control group exhibited longer heart periods (i.e., slower heart rate). Simple effects tests (see Table 1) confirm that at the end of the experiment, group differences were pronounced for both variables. Consistent with the RSA data, across conditions there was a Group effect for heart period, $F(1,77) = 14.2$, $p < .05$.

The vagal contribution to 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 change from baseline to a specific film clip were under vagal control. As illustrated in Fig. 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 the vagal control of the heart, but 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 demonstrate that BPD participants have poor vagal regulation and the changes in heart period may be due, in addition, to other influences such as sympathetic activation.

4. 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 et al., 1999; Schmahl et al., 2004). In contrast, by focusing on the parasympathetic limb of the autonomic nervous system, the current study provides the first report of unique characteristics of autonomic regulation associated with a diagnosis of BPD.

Based upon the limited literature studying the psychophysiology of PTSD (e.g., Sahar, Shalev, & Porges, 2001), we assumed that BPD patients and controls would have similar levels of RSA during baseline. This assumption was confirmed. By challenging the participants with film clips of both high (clips 1 and 3) and low (clip 2)

Table 1
Simple effects for ANOVA

	BPD patients ($n = 9$)	Controls ($n = 11$)	*t*-value	df	*p*-value
Respiratory sinus arrhythmia					
Baseline	5.69[a] (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[b]
Heart period (ms)					
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[b]
Film 3	739 (86)	859 (103)	−2.78	18	.0123[b]

[a] Means (and standard deviations).
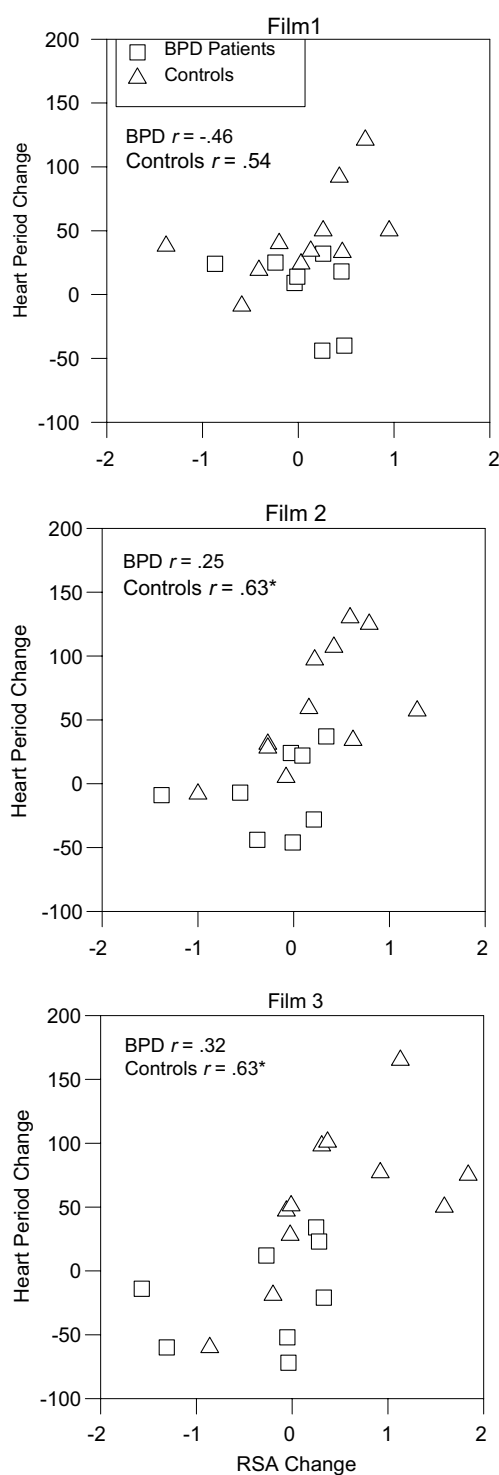[b] Statistically significant $p < .05$.

Fig. 2. Correlations of change from baseline in Heart Period and change from baseline in Respiratory Sinus Arrhythmia from films 1, 2, and 3; *$p < .05$.

BPD participants, 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, we found that neither group exhibited a film clip related response. Instead as illustrated in Figs. 1a and b, the control group exhibited a trajectory of increasing vagal influences on the heart across the entire duration of the experiment, while the BPD group exhibited decreasing vagal influences on the heart.

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 as the experiment progressed. First, the Polyvagal Theory emphasizes that the physiological state that characterizes each group at the end of the experiment supports 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, embedded 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 participants, the BPD group reacted with a visceral state to promote defensive behaviors, while the control group reacted with a visceral state to promote increased spontaneous social engagement behaviors. Second, the Polyvagal Theory (Porges, 2004) proposes a mechanism (i.e., neuroception) that triggers defense strategies. Based on the clinical features of BPD, social interactions with a stranger (i.e., the experimenter) might provide a social stimulus that would trigger the nervous system and elicit a physiological state supporting fight and flight behaviors. However, the film clips in this experiment, with their depictions of social interactions, might have contributed to the need to mobilize. It would be interesting to see whether in the absence of a person in the experimental room, the physiological states of the two groups would still diverge during the film clips. Perhaps, the physiological states of both groups would have been more similar and more stable throughout the experiment in the absence of the experimenter. Under these conditions, the BPD participants might not detect risk and elicit a defensive strategy and the control participants would not detect the cues from the experimenter 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. In previous research, we have reported that this covariation

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 during high emotion stimuli with an expectation that the vagal brake would be reinstated (i.e., increases in RSA and heart period) during the low emotion stimulus. However, we anticipated that the

is compromised in other psychiatric disorders that have difficulties in regulating behavior (Sahar et al., 2001; Umhau et al., 2002). The lack of correlation between RSA and heart period for the BPD group is consistent with these studies.

It is possible that the neuroception of the environment provides an invalid indicator of risk for individuals with BPD. Thus, rather than being calm in the presence of another "non-threatening" 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. If invalid neuroception related to social stimuli accurately describes individuals with BPD, it may partially explain why BPD patients have a variety of poor outcomes, especially related to social relationships and emotional instability in real world situations.

This study has provided the first documented evidence of autonomic nervous system differences between controls and individuals diagnosed with BPD. Thus, it offers a theoretical framework to explain the emotional reactivity that is linked to BPD. The study, however, has several limitations, including small sample size, isolated experimental manipulation, and no measures of test-retest reliability. However, this investigation into the autonomic response profile of BPD patients may lead to additional research to confirm our hypotheses relating autonomic state and neuroception in a variety of other social challenges. Thus, 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 and promote defensive behaviors.

Acknowledgments

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