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Autonomic impairment in Borderline Personality Disorder: A laboratory investigation

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ABSTRACT

Recent research suggests that emotional dysfunction in psychiatric disorders can be reflected in autonomic abnormalities. The present study examines sympathetic and parasympathetic autonomic nervous system activity in individuals with Borderline Personality Disorder (BPD) before, during, and following a social stressor task. Data were obtained from an analogue sample of participants screening positive for BPD ($n = 12$) and healthy controls ($n = 28$). In general, BPD participants exhibited increased sympathetic activity (indexed by Cardiac Sympathetic Index, CSI; Toichi et al., 1997) and decreased parasympathetic activity (indexed by Respiratory Sinus Arrhythmia, RSA) compared to controls. During the stressful task, BPD and control participants exhibited different trajectories of sympathetic activation: estimates of sympathetic activity increased for BPD participants and decreased for controls. Furthermore, BPD participants reported the task (but not baseline or recovery phases) to be more frustrating than controls. Findings are interpreted in the context of Polyvagal theory.

1. Introduction

Borderline Personality Disorder (BPD) is a complex and debilitating psychiatric disorder. According to the DSM-IV-TR, "the essential feature of [BPD] is a pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity that begins by early adulthood and is present in a variety of contexts" (APA, 2000, p. 706). Other indicators of BPD include extreme difficulties with emotion regulation, self-injurious behaviors, intense fears of abandonment, and occasionally the presentation of "psychotic-like" symptoms during times of stress (APA, 2000). BPD affects approximately 2–6% of the general population (APA, 2000; Grant et al., 2008; Torgersen, Kringlen, & Cramer, 2001; Widiger & Weissman, 1991) and as many as 20% of the psychiatric inpatient population (APA, 2001; Zanarini & Grankenbrug, 2001). Seventy-five percent of individuals diagnosed with BPD are female (APA, 2000). Studies indicate that BPD is frequently comorbid with other psychiatric disorders, particularly mood disorders (Skodol et al., 1999; Zimmerman & Mattia, 1999) and eating disorders (APA, 2000). Between 70% and 75% of BPD individuals have a history of suicide attempts (Clarkin, Hull, & Hurt, 1993; Cowdry, Pickar, & Davies, 1985), and as many as one in 10 will eventually complete suicide (McGlashan, 1986; Paris, 2002; Stone, 1993), a rate 50 times higher than the general population (Skodol et al., 2002). In light of the disorder's prevalence and pernicious course, research on factors that may cause and maintain BPD has increased in recent years.

Research suggests that emotion dysregulation is a central, possibly core characteristic of BPD (Conklin, Bradley, & Westen, 2006; Glenn & Klonsky, 2009; Linehan, 1993). Several BPD symptoms, including affective instability, inappropriate anger, and chronic emptiness, appear to reflect emotional dysregulation. Other BPD symptoms, such as self-injury, result from emotion dysregulation (Klonsky, 2007, 2009). Moreover, research suggests that affective instability is the BPD symptom that best predicts the course of the disorder over time (Tragesser, Solhan, Schwartz-Mette, & Trull, 2007). Therefore, clarifying the nature of emotion dysregulation in BPD could enhance knowledge about the disorder's etiology and treatment.

Recently, research has examined how emotion dysregulation in BPD is reflected in central nervous system functioning. Deficits in frontolimbic circuitry, often broadly associated with difficulties inhibiting automatic emotional and behavioral response, have been identified in BPD (see Brendel, Stern, & Silbersweig, 2005 for a review), as has reduced cingulate gray matter, thought to contribute to decreased impulse control and difficulties with emotional processing (Hazlett et al., 2005). A number of studies have also reported increased activation of the amygdala and areas of visual cortex in response to emotional stimuli in BPD. Herpertz et al. (2001) reported increased bilateral activation in the amygdala and fusiform gyri in response to unpleasant compared to neutral images in individuals with BPD compared to controls. Donegan et al. (2003) found similar amygdala hyperactivity in individuals

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with BPD in response to emotional faces. These data have been interpreted as reflecting the emotional symptoms of BPD (e.g., affective instability, difficulty controlling anger).

In contrast, other psychophysiological studies have not always found consistent patterns in how individuals with BPD respond to emotional stimuli. Findings for the eye-blink startle response have been mixed: some groups have found no differences between BPD and Non-BPD individuals in affective modulation of the startle response (Ebner-Priemer et al., 2005; Herpertz, Kunert, Schwenger, Eng, & Sass, 1999; Herpertz et al., 2002), while others have demonstrated a greater increase in startle response following negative emotional stimuli in BPD (Hazlett et al., 2007). Additionally, as compared to healthy controls, BPD patients have comparable responses to emotional stimuli in terms of skin conductance and self-report ratings (Herpertz et al., 1999), and comparable responses to idiographic stressors in terms of heart rate, skin conductance, and blood pressure (Schmahl et al., 2004). These null results are somewhat surprising given the evidence for differences in central nervous system functioning in individuals with BPD (Donegan et al., 2003; Herpertz et al., 2001), and the fact that the central and peripheral nervous systems are intertwined (Hagemann, Waldstein, & Thayer, 2003; Thayer & Lane, 2000).

1.1. Polyvagal theory

Porges' polyvagal theory provides a coherent framework for generating hypotheses about the psychophysiological abnormalities likely to characterize emotional disorders such as BPD. Polyvagal theory (1995, 2001, 2003, 2007) is a phylogenetic approach relating autonomic system function to behavior. Polyvagal theory explores parasympathetic control over heart period via the vagus nerve, and specifies two sources of vagal efference to the heart terminating on the Sino-Atrial (SA) node, or the cardiac pacemaker. One emanates from the nucleus ambiguus (NA), which also regulates cranial and facial muscles related to social engagement. The other, vegetative vagus, originates in the dorsal motor nucleus (DMNX) and mediates reflexive cardiac activity. The unmyelinated vegetative vagus is responsible for primitive threat responses like orienting, feigning death, and immobilization (for reviews, see Porges, 1995 or Beauchaine, 2001). In contrast, the myelinated vagus is distinctly mammalian and allows for sustained attention and engagement with the environment via suppression of sympathetic influence on the heart.

In his model, Porges also discusses neuroception, the unconscious neural processes by which individuals distinguish friend from foe, safety from danger. In non-threatening situations, the NA path of the vagus sends constant inhibitory signals to the heart, acting as a brake on the heart rate and allowing individuals to engage in tasks that require minimal mobilization of the sympathetic nervous system. Porges places special emphasis on the reliance of social behaviors on the activity of the vagus, in part because of its involvement in coordinating vocalizations, facial expressions, and the communication of emotional states to others (Porges, 1995; Thayer & Lane, 2000). In threatening or distressing situations, vagal influence rapidly decreases and individuals revert to phylogenetically-older responses like freezing, or the sympathetically-activated fight-or-flight response. This decrease in vagal influence is referred to as vagal withdrawal, while any activity away from baseline, including increased vagal control, is referred to as vagal reactivity. Combined with the sympathetic-adrenal system, the two sources of vagal efference offer a broader array of behavioral options, allowing mammals to respond rapidly and flexibly to environmental challenges. The mammalian parasympathetic system is thus conceptualized as a higher-order response to the unique demands of mammalian life, one that allows for transitory mobilization and expression of sympathetic tone without the costly metabolic demands of full sympathetic or adrenal activation. In humans, vagal withdrawal has been shown to occur in response to a variety of emotional stimuli, including sad film clips (Rottenberg, Salomon, Gross, & Gotlib, 2005), anxiety-provoking public speaking tasks (Rottenberg, Clift, Bolden, & Salomon, 2007), and mental arithmetic tasks designed to cause frustration (Mezzacappa, Kelsey, Katkin, & Sloan, 2001).

1.2. Polyvagal theory and BPD

From the perspective of polyvagal theory, emotional impairment in many psychopathologies may be explained by an increased sensitivity to threat information. This increased sensitivity is reflected in decreased vagal control and results in an inability to appropriately engage or disengage defense systems (Porges, 2004). Indeed, atypical vagal influence has been related to multiple emotional disorders and emotional states, including depression (Carney et al., 1995; Rechlin, Weis, Spitzer, & Kaschka, 1994; Rottenberg, Wilhelm, Gross, & Gotlib, 2003), anxiety (Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, & Borkovec, 1996), worry (Hofmann et al., 2005), self-injury (Crowell et al., 2005), trait hostility (Sloan et al., 1994), and stress (Allen & Crowell, 1989). High vagal tone, on the other hand, has been associated with enhanced ability to cope with life stressors (Fabes & Eiseinberg, 1997).

Given that emotional instability and dysregulation may represent a core feature of BPD (Conklin et al., 2006; Glenn & Klonsky, 2009; Linehan, 1993), abnormal patterns of vagal activity should be evident in individuals with this disorder as well. To date, only one study has examined BPD from the perspective of polyvagal theory. Austin, Riniolo, and Porges (2007) compared a BPD group to a control group during emotional film clip viewing. Respiratory Sinus Arrhythmia (RSA) was assessed to index vagal activity; RSA refers to the oscillation in heart period that results from the respiratory cycle, and reliably approximates the effect of the parasympathetic nervous system on the heart (Berntson, Cacioppo, & Quigley, 1993; Berntson et al., 1997; Friedman, Allen, Christie, & Santucci, 2002). Austin and colleagues found RSA to be moderately lower in individuals with BPD, although the difference was not statistically significant, perhaps due to the small sample size (9 BPD v. 11 Controls). They also observed contrasting trajectories of parasympathetic activity over the course of three 10-min film presentations. Specifically, RSA decreased during the viewing task in the BPD group, but increased over the same period in the control subjects. From the perspective of Porges' polyvagal theory, this suggests that the BPD group ended the viewing task in a physiological state of preparedness for defensive behaviors, while the control group ended in a state that would support social engagement behaviors. Austin et al. suggested that the study be replicated in light of the small sample size, and also speculated that the heightened physiological preparedness for defense behaviors observed in their BPD participants might have a sympathetic as well as a parasympathetic component. However, sympathetic activity was not quantified in Austin et al., perhaps because evidence for sympathetic hyperarousal in BPD has been mixed, (e.g., Hazlett et al., 2007; Herpertz et al., 1999; Schmahl et al., 2004). Because other disorders characterized by high negative affect and emotional reactivity have been associated with hyperreactivity of the sympathetic nervous system (SNS) quantified by other physiological measures such as skin conductance response (e.g., Cook, Hawk, Davis, & Stevenson, 1991; Cook, Melamed, Cuthbert, McNeil, & Lang, 1988; Cuthbert, Drobes, Patrick, & Lang, 1994; Hoehn-Saric & McLeod, 1993; Kelly, Brown, & Shaffer, 1970; Öhman & Soares, 1994), and because the influences of the sympathetic and parasympathetic branches of the autonomic nervous system (ANS) are generally thought to be antagonistic, we hypothesized that

BPD, too, would show a pattern of increased SNS activity complementing the expected pattern of decreased parasympathetic nervous system (PNS) activity.

However, there are other reasons that it may be informative to examine the activity of the two branches of the ANS in conjunction, in particular when considering metrics of heart rate variability (HRV), as the heart is dually innervated and the action of each branch is not independent of the other (e.g., Berntson, Cacioppo, & Quigley, 1991; Berntson et al., 1993). As previously discussed, the vagus primarily exerts its direct influence over heart period via efferents terminating on the SA node and the release of fast-acting acetylcholine (Ach) at the neuro-muscular junction, (Talman & Kelkar, 1993). By contrast, the influence of the sympathetic nervous system is more diffuse and slow; although it too has efferents terminating on the SA node, sympathetic regulation relies upon relatively slower-acting adrenaline.

The relative influence of the PNS and SNS in chronometric regulation of the heart can be assessed by examining changes in HRV derived from a continuous tachogram created by quantifying the intervals between *R*-waves. Recent attempts to obtain measures of HRV have used spectral analysis, which utilizes Fourier transformations to decompose the heart rate time series into component frequencies. Spectral analysis of *R–R* intervals has fairly consistently demonstrated that the low-frequency component of the spectral band reflects both vagal and sympathetic influence, while the high-frequency component reflects exclusively vagal influences (e.g., Pagani et al., 1986; Pomeranz et al., 1985). However, the mathematical models for isolating sympathetic influence have often been often unwieldy and have varied from investigator to investigator. The Cardiac Respiratory Index (CSI) was therefore developed by Toichi, Sugiura, Murai, and Sengoku (1997) as a method of assessing the sympathetic influence over heart rate variability. Though CSI has thus far been infrequently used, there are some data to support its validity as an index of SNS influence over heart period. CSI has been shown to be sensitive to sympathetic blockade by a beta-adrenergic blocker (propranolol; Toichi et al., 1997). In addition, changes in CSI have been associated with psychotic symptoms in patients with schizophrenia (Toichi et al., 1999), epileptic discharges in patients with temporal lobe epilepsy (Toichi et al., 1998), and the influence of cocaine on ANS function (Newlin, Wong, Stapleton, & London, 2000). The evidence for CSI changes across resting and stressor tasks has been mixed; some studies have found no change from resting to standing (Toichi et al., 1997) or from resting to mental arithmetic (Toichi & Kamio, 2003). Others have found that CSI can reliably discriminate between resting and stressor tasks (Allen, Chambers, & Towers, 2007). Despite its limited usage, more evidence on CSI's utility as a non-invasive marker of ANS activity may be helpful as our understanding of individual differences in autonomic processing increases.

1.3. Specific hypotheses

The polyvagal theory and Austin et al.'s findings guide the present study, in which impaired parasympathetic and sympathetic activity were expected to characterize individuals with BPD. Because Polyvagal theory suggests that parasympathetic activity in humans facilitates emotion regulation, we examined autonomic activity before, during, and after a stressful task (see Section 2.2 for task details). There were two hypotheses. First, we hypothesized that BPD individuals would exhibit a pattern of attenuated parasympathetic activity (Hypothesis 1a) and heightened sympathetic activity (Hypothesis 1b) compared to controls. It was further hypothesized that this pattern would be associated with an increased heart rate (HR) in BPD individuals compared to controls (Hypothesis 1c). Second, we hypothesized contrasting trajectories of autonomic activity between BPD and control participants. Specifically, we hypothesized that, relative to controls, BPD participants would exhibit a greater decline in parasympathetic activity (Hypothesis 2a) and a greater rise in sympathetic activity (Hypothesis 2b) during the stressor task.

2. Method

2.1. Participants

Participants were drawn from a screening completed by college students in lower-level psychology courses for course credit. Twelve participants were chosen because they screened positive for BPD on the McLean Screening Instrument for Borderline Personality Disorder (MSI-BPD; Zanarini et al., 2003). Twenty-eight participants who endorsed either zero or one symptom on the MSI-BPD comprised the control sample.

2.2. Procedure

Those who completed the mass screening and met inclusion criteria either for the BPD group (five or more symptoms of BPD) or the control group (one or zero symptoms of BPD) were recruited to participate in a study on emotion and psychophysiology. Questionnaires assessing demographic characteristics were administered following a psychophysiological protocol. The psychophysiological protocol consisted of three 5-min stages. For Stage 1 (Baseline), participants were asked to sit still and relax. For Stage 2 (stressor), experimenters administered a mental arithmetic task whereby participants had to count down from the number 1258 in increments of 7.

At several points, an experimenter provided feedback meant to frustrate the participant. Specifically, participants were instructed to count down "as fast as you can." Each time an error was made, an experimenter instructed the participants to start over from the beginning (counting down from 1258). If participants achieved eight consecutive correct answers, they were told, "Please try not to pause between responses." If another eight consecutive correct answers were achieved, participants were told, "Try to go faster." For Stage 3 (Recovery), participants were asked to sit still and relax. To verify that the mental arithmetic task and feedback were sufficiently frustrating, 25 participants completed ratings of frustration using a nine-point scale immediately before (baseline) and after the task, as well as after their recovery period.

2.3. Measures

2.3.1. Borderline Personality Disorder (BPD)

BPD symptoms were measured using the McLean Screening Instrument for Borderline Personality Disorder (MSI-BPD; Zanarini et al., 2003). The MSI-BPD is a self-report measure of the DSM-IV BPD criteria. In the present study, a BPD criterion was considered to be present if the corresponding MSI-BPD item was endorsed. Because two items corresponded to the ninth BPD criterion (stress-related paranoid ideation or dissociative symptoms), this criterion was considered to be present if either of the two corresponding MSI-BPD items were present. When compared to a validated structured interview, sensitivity and specificity of the MSI-BPD were both above .90 in a sample of young adults (Zanarini et al., 2003).

2.3.2. Physiological measures

The electrocardiogram (EKG) was monitored via Ag/AgCl electrodes placed on participants' right forearm and both ankles, and connected to a MP100 BIOPAC system running AcKnowledge 3.8 (BIOPAC Systems, Goleta, CA, USA) at a sample rate of 1000 Hz. RSA was utilized as an index of parasympathetic activity. The validity of RSA as a measure of parasympathetic activity has been

established via pharmacologic (cholinergic) blockade (Berntson et al., 1997). RSA was computed from participants' EKG data using the software and procedure described in Allen et al. (2007). Following Allen et al. (2007) raw digitized EKG signals were analyzed offline. Inter-beat Interval (IBI) series were hand-corrected for artifacts and processed by CMetX for measures of HR and HRV. The IBI series was also converted, via CMetX, to a time-series sampled at 10 Hz, and was filtered with a 241-point optimal finite impulse response digital filter designed using FWTGEN V3.8 (Cook & Miller, 1992), with half-amplitude frequencies of .12 and .40 Hz. CMetX estimates RSA as the natural log of the variance of the filtered waveform.

The Cardiac Sympathetic Index (CSI; Toichi et al., 1997) was proposed by Toichi as an index of sympathetic activity. CSI is a metric derived from measures of HRV via CMetX (Allen et al., 2007). CSI is derived via a Lorenz plot of each IBI (IBI_n) plotted against the subsequent IBI (IBI_{n+1}). Beat-to-beat variability is reflected in the length of the transverse axis (T) to the line $IBI_n = IBI_{n+1}$. The range of IBIs is reflected in the length of the longitudinal axis (L). CSI is calculated as the ratio of L/T. In the present study, Pearson's correlations between RSA and CSI at each of the three stages (−.33 at rest, −.33 during stressor task, −.29 at recovery; see Table 1 for complete correlations within and between the indices) indicated an inverse but non-redundant association between the two variables.

2.4. Statistical analyses

A mixed-model repeated-measures analysis of variance (ANOVA) was conducted to examine: (a) differences in RSA, CSI, and mean HR across the three stages (baseline, stress, and recovery), (b) differences in RSA, CSI, and HR between the BPD and control groups, and (c) the group-by-stage interaction for RSA, CSI and HR. A mixed-model repeated-measures ANOVA was also conducted to examine a gender by stage interaction for RSA, CSI and HR. Thus, for all three measures, a 2 (Group) × 3 (experimental stage) repeated-measures ANOVA was conducted, as well as a 2 (Gender) × 3 (experimental stage) repeated-measures ANOVA. The primary analyses were then repeated with gender entered as a covariate. To address Hypotheses 2a and 2b, we examined changes in RSA and CSI over the course of the stressor. The 5-min stressor was divided into two 2.5-min blocks, and RSA and CSI were calculated separately for each block. Two 2 (group) × 2 (stressor half) mixed-model ANOVAs were utilized to determine if changes in RSA and CSI from the first- to second-half of the stressor varied for the control and BPD groups.

3. Results

Mean age of the sample was 19.9 (sd = 5.0). Twenty-nine participants were female, and eleven were male. Nineteen participants were Asian, fifteen Caucasian, two African American, and four Hispanic/Latino. There were no significant age, gender, or ethnic differences between participants in the BPD and control groups.

However, there were some physiological differences between the genders. Specifically, for RSA, there was a main effect of gender [$F(1, 38) = 4.00$, $p < .05$, $\eta_p^2 = .09$], such that female participants exhibited significantly lower parasympathetic activity overall ($M = 6.11$, $s_M = 0.13$) than males ($M = 6.63$, $s_M = 0.22$). There was no significant effect of stage [$F(2, 76) < 1$ $\eta_p^2 = .01$], and no interaction between stage and group [$F(2, 76) < 1$, $\eta_p^2 = .00$]. Additionally, for CSI, there was a main effect of gender [$F(1, 38) = 4.34$, $p < .05$, $\eta_p^2 = .10$], such that female participants exhibited significantly higher sympathetic activity overall ($M = 2.70$, $s_M = 0.1$) than males ($M = 2.33$, $s_M = 0.2$). There was a significant effect of stage [$F(2, 76) = 25.03$, $p < .001$ $\eta_p^2 = .40$], but no interaction between stage and group [$F(2, 76) < 1$, $\eta_p^2 = .00$]. Finally, for HR, there was no main effect of gender [$F(1, 38) = 2.46$, $p = .13$, $\eta_p^2 = .06$] and no interaction between stage and group [$F(2, 76) < 1$, $\eta_p^2 = .02$], though there was a significant effect of stage [$F(2, 76) = 42.32$, $p < .001$, $\eta_p^2 = .53$]. For this reason, we conducted all primary study analyses both with and without gender as a covariate.

3.1. Frustration ratings

Self-reports of frustration before and after the stressor were available for a subset of participants (16 controls, nine BPD subjects). As expected, there was a main effect of stage [$F(1, 23) = 52.3$, $p < .001$, $\eta_p^2 = .62$], such that frustration was significantly higher following the stressor ($M = 5.0$, $s = 2.4$) than at baseline ($M = 1.8$, $s = 1.5$). There was no main effect of group [$F(1, 23) < 1$, $\eta_p^2 = .04$]. However, there was a significant group-by-stage interaction [$F(1, 23) = 3.65$, $p = .05$, $\eta_p^2 = .12$]. Specifically, BPD participants became substantially more frustrated as a result of the stressor ($M = 6.0$, $s = 2.5$) compared to controls ($M = 4.5$, $s = 2.3$), even though BPD and controls reported comparable frustration for baseline and recovery.

3.2. Autonomic activity

3.2.1. Parasympathetic activation

Results for RSA for BPD and Non-BPD participants as a function of experimental stage are displayed in Fig. 1. Consistent with Hypothesis 1a, there was a main effect of group [$F(1, 38) = 14.28$, $p < .01$, $\eta_p^2 = .22$], such that participants with BPD exhibited significantly lower parasympathetic activity overall ($M = 5.7$, $s_M = 0.2$) than controls ($M = 6.5$, $s_M = 0.1$). There was no effect of stage [$F(2, 76) < 1$, $\eta_p^2 = .01$], and no interaction between stage and group [$F(2, 76) < 1$, $\eta_p^2 = .01$]. When gender was included as a covariate, there was a main effect of group [$F(1, 37) = 10.26$, $p < .01$, $\eta_p^2 = .22$], such that participants with BPD exhibited significantly lower parasympathetic activity overall than controls. There was

Table 1
Intercorrelations within and between RSA and CSI at each stage of the experiment.

	RSA rest	RSA stressor	RSA recovery	CSI rest	CSI stressor	CSI recovery	HR rest	HR stressor
RSA stressor	.66**	–						
RSA recovery	.81**	.69**	–					
CSI rest	−.33*	−.33*	−.38*	–				
CSI stressor	−.22	−.42**	−.18	.26	–			
CSI recovery	−.49**	−.41**	−.29	.60**	.27	–		
HR rest	−.17	−.44**	−.11	.21	.20	.13	–	
HR stressor	−.03	−.52**	.02	.10	.47**	.07	.76**	–
HR recovery	−.17	−.49**	−.09	.16	.10	.15	.96**	.80**

* Indicates the correlation is significant at $p < .05$.
** Indicates the correlation is significant at $p < .01$.

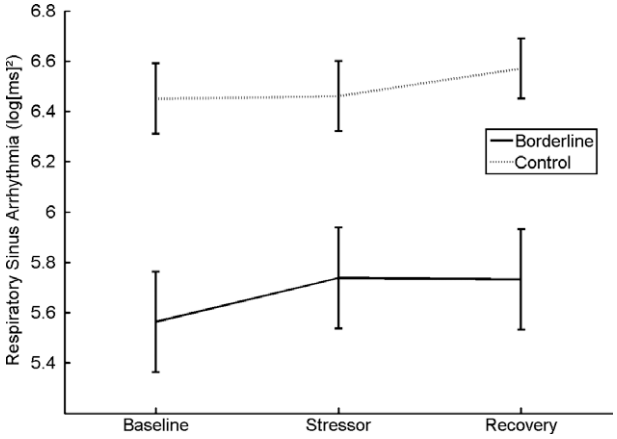

Fig. 1. Parasympathetic nervous system activity indexed by Respiratory Sinus Arrhythmia (RSA) in participants with and without BPD.

also a small main effect of gender [$F(1, 37) = 4.07$, $p < .05$, $\eta_p^2 = .09$], such that males exhibited slightly higher parasympathetic activity overall ($M = 6.4$, $s_M = 0.2$) than females ($M = 6.1$, $s_M = 0.1$). There was no effect of stage [$F(2, 74) < 1$, $\eta_p^2 = .01$], and no interaction between stage and group [$F(2, 74) < 1$, $\eta_p^2 = .01$] or stage and gender [$F(2, 74) < 1$, $\eta_p^2 = .01$].

Contrary to Hypothesis 2a, for RSA there was no interaction between stressor half and group[$F(1, 38) < 1$, $\eta_p^2 = .01$]; controls showed a non-significant increase in RSA from the first- ($M = 6.2$, $s_M = 0.2$) to second-half ($M = 6.4$, $s_M = 0.3$) of the stressor, whereas the means for the BPD group remained virtually identical for both blocks ($M_1 = 5.7$, $s_M = 0.3$, $M_2 = 5.7$, $s_M = .3$). When gender was entered as a covariate, there was again no interaction between stressor half and group [$F(1, 34) < 1$, $\eta_p^2 = .01$] or between stressor half and gender [$F(1, 34) < 1$, $\eta_p^2 = .01$].

3.2.2. Sympathetic activation

Results for CSI for BPD and Non-BPD participants as a function of experimental stage are displayed in Fig. 2. Consistent with Hypothesis 1b, there was a main effect of group [$F(1, 38) = 4.0$, $p = .05$, $\eta_p^2 = .10$], such that participants with BPD exhibited more sympathetic activity ($M = 2.9$, $s_M = 0.2$) than controls ($M = 2.5$, $s_M = 0.1$). In addition, CSI differed significantly by stage [$F(2, 76) = 26.8$, $p < .001$, $\eta_p^2 = .41$], with CSI being higher during the stressor ($M = 3.2$, $s = 0.8$) than during baseline ($M = 2.3$, $s = 0.6$) and recovery ($M = 2.3$, $s = 0.7$). There was no interaction between stage and group [$F(2, 76) < 1$, $\eta_p^2 = .00$]. When gender was entered as a covariate, there was no longer a main effect of group [$F(1, 37) = 2.1$, $p = .16$, $\eta_p^2 = .05$]. However, there was a trend towards a main effect of gender [$F(1, 37) = 3.9$, $p = .06$, $\eta_p^2 = .09$] such that males exhibited less sympathetic activity ($M = 2.3$, $s_M = 0.1$) than females ($M = 2.7$, $s_M = 0.1$). In addition, the effect of stage was no longer significant [$F(2, 74) = 2.17$, $p = .12$, $\eta_p^2 = .06$]. There was no interaction between stage and group [$F(2, 74) < 1$, $\eta_p^2 = .00$], or between stage and gender [$F(2, 74) < 1$, $\eta_p^2 = .00$].

However, consistent with Hypothesis 2b, for CSI there was an interaction between stressor half and group [$F(1, 38) = 7.19$, $p < .01$, $\eta_p^2 = .17$]. Specifically, for the BPD group, CSI increased from the first- ($M = 3.2$, $s_M = 0.3$) to second-half ($M = 3.5$, $s_M = 0.2$) of the stressor, whereas for the control group CSI decreased from the first- ($M = 3.2$, $s_M = 0.2$) to second-half ($M = 2.9$, $s_M = 0.2$) of the stressor. Results for CSI for BPD and Non-BPD participants as a function of stressor half are displayed in Fig. 3. When gender was included as a covariate, the interaction between stressor half and group remained [$F(1, 34) = 6.14$, $p < .01$, $\eta_p^2 = .15$]. There was no interaction between stressor half and gender [$F(1, 34) < 1$, $\eta_p^2 = .00$].

3.2.3. Heart rate

There was a main effect of stage [$F(2, 76) = 37.9$, $p < .01$, $\eta_p^2 = .50$], such that HR was significantly higher during the stressor task ($M = 88.7$, $s_M = 2.6$) than at baseline ($M = 79.9$, $s_M = 2.3$) or recovery ($M = 77.1$, $s_M = 2.3$). However, contrary to Hypothesis 1c, there was no effect of group [$F(1, 38) < 1$, $\eta_p^2 = .01$], and no interaction between stage and group [$F(2, 76) = 1.1$, $p = .3$, $\eta_p^2 = .01$]. When gender was included as a covariate, there was a main effect of stage [$F(2, 74) = 3.1$, $p < .05$, $\eta_p^2 = .08$]. There was no effect of group [$F(1, 37) < 1$, $\eta_p^2 = .00$], no effect of gender [$F(1, 37) = 2.7$, $p = .11$, $\eta_p^2 = .07$], no interaction between stage and group [$F(2, 74) < 1$, $\eta_p^2 = .02$], and no interaction between stage and gender [$F(2, 74) < 1$, $\eta_p^2 = .00$].

A Spearman's correlation indicated there was no significant relationship between age and RSA [$r(38) = -.08$, $p = .68$] or CSI [$r(38) = -.27$, $p = .09$].

4. Discussion

The present study examined autonomic activity associated with BPD symptomatology from the perspective of polyvagal theory.

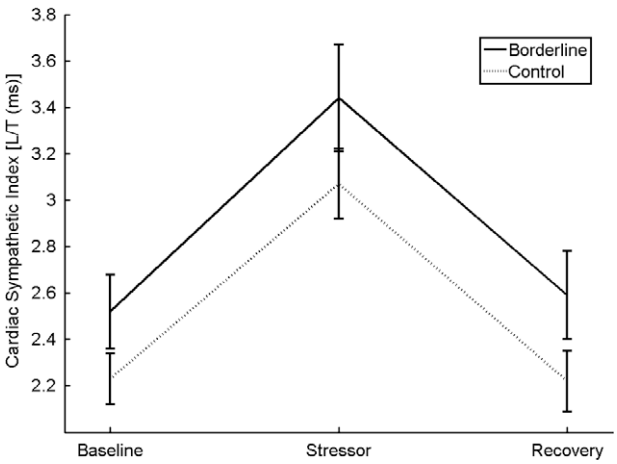

Fig. 2. Sympathetic nervous system activity indexed by Cardiac Sympathetic Index (CSI) in participants with and without BPD.

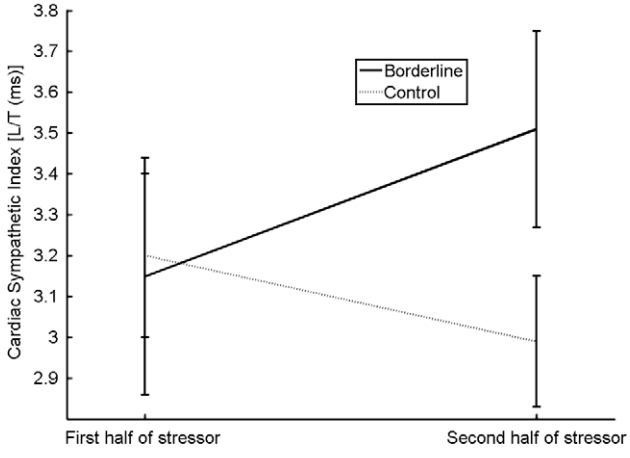

Fig. 3. Trajectories of sympathetic nervous system activity indexed by Cardiac Sympathetic Index (CSI) during a stressor task in participants with and without BPD.

Findings regarding autonomic functioning were largely consistent with previous research and theory on psychiatric disorders characterized by emotional dysfunction (Beauchaine, 2001; Porges, 1995, 2001, 2003, 2007). More specifically, three of the four study hypotheses were supported. BPD participants exhibited decreased parasympathetic and increased sympathetic activity during a 15-min laboratory study. In addition, the groups exhibited contrasting sympathetic trajectories. Whereas sympathetic activity decreased over the course of the 5-min stressor for healthy controls (perhaps indicating partial habituation to the stressor), sympathetic activity increased for BPD participants. This pattern suggests that during the stressful task BPD participants were becoming increasingly aroused and were more inclined than controls to revert to the phylogenetically-older 'fight-or-flight' response. Consistent with their sympathetic response to the stressful task, BPD participants reported feeling more frustrated by the task than controls (but did not report more frustration at baseline or during recovery).

However, contrary to expectations, the two groups did not show diverging trajectories of RSA over the course of the 5-min emotional stressor task. Austin et al. (2007), the first study to apply polyvagal theory to BPD, found contrasting trajectories of RSA for BPD versus control participants watching emotional film clips. In the present study RSA trajectories for BPD participants and controls diverged in a similar manner, but the pattern (e.g., stage by group interaction) did not approach statistical significance. In Austin et al. the trend was *not* detectable after participants watched an emotional film clip for 10 min, and only became apparent after 20 min of watching film clips and more so after 30 min. Perhaps the trend in the present study would have become significant if our emotional stressor had continued for longer than 5 min. However, there were several other differences between the present study and Austin et al. (2007). For example, Austin, et al. used only female participants, their sample was a community sample, and the HRV data was recorded from different sites on the participants. Any of these factors could also have contributed to the discrepancies between the two studies.

In one other respect our findings diverged from Austin et al. (2007). The tendency for BPD participants to exhibit lower vagal tone was statistically significant in the present study but not in Austin et al. The discrepancy may have been due to a small sample size in Austin et al. (9 BPD subjects versus 11 controls), because the effect in their study was non-trivial and in the predicted direction. In addition, previous research has consistently found low vagal tone in participants with emotional disorders (Beauchaine, 2001; Crowell et al., 2005), and in our study vagal tone was significantly lower in BPD participants than in controls, as would be expected.

It is also possible that inconsistencies in the current body of research on physiological correlates of BPD stem from the use of different stressors. Stimuli and stressors used to elicit emotional responses from BPD subjects have ranged from acoustic startle probes (Ebner-Priemer et al., 2005), to startle probes combined with BPD-relevant words (Hazlett et al., 2007), to standardized photographic images (Herpertz et al., 1999, 2001), to emotional and neutral faces (Donegan et al., 2003), to film clips (Austin et al., 2007). As the body of research on psychophysiological responding in BPD grows, a more cohesive pattern of results may emerge suggesting that some types of stressors differentiate BPD better than others. In addition, it will be important to evaluate BPD individuals in social stress as well as other domains of functioning.

Nonetheless, results from the present study and previous research shed light on emotion dysregulation and BPD. Specifically, it appears that BPD individuals can be considered vulnerable in two ways. First, our study would indicate that parasympathetic activity may be lower and sympathetic reactivity higher in BPD individuals compared to controls, perhaps effecting a constant state of readiness in BPD individuals, allowing for rapid response to threat information. Second, vagal strength in BPD individuals is attenuated to such an extent that, in the face of a relatively non-threatening stressor, sympathetic activity increases, perhaps facilitating a more acute experience of negative events. This evidence for enhanced emotional reactivity is further supported by the increased self-reports of frustration as a result of the stressor demonstrated in the BPD participants.

Findings suggest several future directions for research. First, an open question remains as to whether attenuated vagal tone, vagal hyporeactivity, and potentiated sympathetic response cause the chronic and often debilitating emotional and interpersonal difficulties frequently displayed by BPD patients (e.g., Daley, Burge, & Hammen, 2000). This issue could be addressed both by examining the prospective link between compromised autonomic functioning and interpersonal dysfunction and by ambulatory monitoring of autonomic activity while emotional and interpersonal events are recorded using daily–diary methods (e.g., Ebner-Priemer et al., 2007). Second, in addition to the effects of stressful tasks on autonomic activity and mood, research should examine the effects of autonomic and emotional dysfunction on performance-based stressful tasks. Such research could help capture the extent to which autonomic and emotional impairment contributes to behavioral impairment.

One limitation of the present study is the use of Toichi's (1997) measure of CSI as our only index of sympathetic activity. Although CSI has been shown to react to stressors and sympathetic blockade in a manner supporting its validity as an index of sympathetic activity (Allen et al., 2007; Toichi et al., 1997) and the index behaved as expected in the present study, replications of the blockade study have not yet been conducted, and there is limited convergent data in support of CSI as a reliable index of sympathetic nervous system influence. In addition, correlations in the present study between CSI, and RSA and HR are somewhat lower than those reported in Allen et al. (2007). This may be a result of differences in sample size between the two studies ($n = 96$ in Allen et al.), or the fact that HRV was assessed in different sites; however, future studies should also utilize additional indices of sympathetic activity, such as skin conductance.

Additional limitations of the present study suggest future research directions. These include a small sample size ($n = 40$), reliance on a self-report measure to assess BPD, and use of a college sample. Future studies should examine autonomic functioning in larger samples of BPD participants and controls from community and clinical populations, and utilize structured interviews to diagnose BPD. Additionally, it will be important to incorporate clinical control groups (e.g., depressed and/or anxious patients) into future studies to identify patterns of autonomic responding that differentiate physiological responses in BPD from other psychiatric disorders. Given that the present study did not compare the BPD and control groups on other variables that might also affect physiological responding—such as anxiety, depression, alcohol or drug use, or general difficulties with emotion dysregulation, all of which are frequently comorbid with BPD (e.g., Glenn & Klonsky, 2009)—future work should also consider the contribution of these more specific traits in influencing physiological response to stressors in BPD. Future studies might also include controls with emotional disorders such as anxiety or depression. In addition, though the stressor utilized in the present study had social elements (e.g., deliberate frustration of the participant by an experimenter), it is difficult to isolate the effects of social stress from frustration associated with a cognitively demanding task. Therefore, it will be necessary to further evaluate ANS functioning in BPD utilizing other stressors which can more directly manipulate social or interpersonal stress.

Finally, given that one aim of Dialectical Behavior Therapy (DBT), currently the front-line treatment for BPD, is to improve emotion dysregulation (Linehan, Bohus, & Lynch, 2007), it may be useful to conduct physiological assessments pre- and post-treatment. In this manner, autonomic indices of emotion dysregulation could be examined as potentially valuable predictors and indices of treatment outcome.

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