



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.

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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, Kringle, & Cramer, 2001; Widiger & Weissman, 1991) and as many as 20% of the psychiatric inpatient population (APA, 2001; Zanarini & Grankenburg, 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 (Lyons-Borkovec, 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 & Eisenberg, 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, Drobos, 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

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