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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 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.

Keywords: Borderline personality disorder; Polyvagal Theory; Heart rate; Heart rate variability; Respiratory sinus arrhythmia

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)

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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-