



Heart rate and heart rate variability in panic, social anxiety, obsessive–compulsive, and generalized anxiety disorders at baseline and in response to relaxation and hyperventilation

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ABSTRACT

It remains unclear if diminished high frequency heart rate variability (HF-HRV) can be found across anxiety disorders. HF-HRV and heart rate (HR) were examined in panic (PD), generalized anxiety (GAD), social anxiety (SAD), and obsessive–compulsive disorder (OCD) relative to healthy controls at baseline and during anxiety stressors. All disorders evidenced diminished baseline HF-HRV relative to controls. Baseline HRV differences were maintained throughout relaxation. For hyperventilation, PD and GAD demonstrated greater HR than controls. Psychotropic medication did not account for HF-HRV differences except in OCD. Age and sex evidenced multiple main effects. Findings suggest that low baseline HF-HRV represents a common index for inhibitory deficits across PD, GAD, and SAD, which is consistent with the notion of autonomic inflexibility in anxiety disorders. Elevated HR responses to hyperventilation, however, are specific to PD and GAD.

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1. Introduction

A robust literature examined cardiac psychophysiology at rest and in response to provocation among individuals with anxiety disorders. Most studies focused on panic disorder (PD), and to a lesser extent, on generalized anxiety disorder (GAD) and social anxiety disorder (SAD; see Friedman, 2007). Within these studies, heart rate variability (HRV) and heart rate (HR) were the most common measures of cardiovascular activity. HRV in the high frequency spectrum (HF-HRV) represents an index of respiratory sinus arrhythmia (RSA), which involves regular patterns of HR fluctuations that are linked to the breathing cycle and modulated by the parasympathetic nervous system (Thayer and Lane, 2000). Extensive research demonstrated associations between HRV and various physical diseases, psychopathology, and emotion regulation (e.g., Camm et al., 1996; Beauchaine, 2001; Friedman, 2007). Most important, theories have proposed close links between low RSA and psychopathology, especially anxiety disorders.

The Polyvagal theory (e.g., Porges, 2007) links autonomic regulation and RSA to a variety of (psycho-) pathological states and behaviors. Adaptive behavior and autonomic responses emerge from the hierarchical organization of different phylogenetic subsystems of the autonomic

nervous system with phylogenetically newer systems inhibiting older ones. These inhibition processes are essential for adaptive behavior with (autonomic) variability being associated with healthy responses. Deficits in these inhibitory processes are seen as a risk for emotion dysregulation and psychopathology more generally (Beauchaine, 2001). The related neurovisceral model of cardiac and emotion regulation proposes specific links between cardiovascular variability and anxiety disorders (Thayer and Lane, 2000; Friedman, 2007). It outlines a central and peripheral network that integrates autonomic, attentional, and affective systems involved in emotion self-regulation. Within this homeodynamic view, healthy physiological variability involves the ability to adaptively react to environmental demands to maintain the stability of an organism. Anxiety disorders (and other affective disorders) are characterized by a rigid emotional response style with responses not reflecting environmental demands. More specifically, this rigid response style expresses as an inability to inhibit inappropriate anxious responses in non-threatening situations. HF-HRV predominantly represents the parasympathetic nervous system output of this integrated network and, thus, serves as an important index of the adaptability and regulatory ability of an organism, with decreased HF-HRV associated with less flexible responding to the environment. Thus, this model predicts that the rigid responding in anxiety disorders is associated with lower HRV.

In support, numerous studies demonstrated that relative to non-anxious controls, individuals with anxiety disorders evidence lower HF-HRV at rest or in response to anxiety stressors (Klein et al., 1995; Friedman and Thayer, 1998a, 1998b). This lower parasympathetic

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cardiac control has been especially found in PD (see Friedman, 2007). The smaller number of GAD and SAD studies demonstrate more mixed results, with some showing lower HF-HRV at rest or in response to anxiety-related provocation relative to non-anxious controls (Thayer et al., 1996, 2000; Grossman et al., 2001) and others showing no differences, particularly for SAD (Kollai and Kollai, 1992; Mauss et al., 2003). The sole published study examining HRV in obsessive-compulsive disorder (OCD) (Slaap et al., 2004) found no HRV differences between OCD and control patients. This study, however, also reported null findings on HRV differences between PD and control patients, contradicting much of the literature and suggesting that sample or other study-specific variables may account for the findings. Overall, findings regarding lower HRV at rest across anxiety disorders remained mixed.

Recent studies also demonstrated stronger HRV reactivity to certain stimuli or tasks in anxiety disorders. PD patients, for example, showed a larger decrease in HRV in response to panicogenic substances like sodium lactate or yohimbine (Yeragani et al., 1992, 1994), or a stronger increase in sympathetic-parasympathetic ratios after isoproterenol (Pohl and Yeragani, 2001). These elevated task-specific effects on HRV are also evident in other anxiety disorders. For example, similar effects were found for individuals with dental phobia when confronted with phobia-relevant stimuli (Johnsen et al., 2003), for PTSD patients during speech or mental arithmetics (Keary et al., 2009), or for general measures of anxiety (Shinba et al., 2008). Particularly within homeostatic approaches, differences between individuals with and without anxiety disorders in tonic and phasic HR have been investigated. Despite mixed findings, many studies demonstrate higher HR at baseline and in response to anxiety-related stressors among individuals with anxiety disorders controls (see Aikins and Craske, 2010). Thus, the rigid response style with little variability in anxiety disorders is not only observable as low tonic HRV (e.g., at rest or during relaxation), but also as an inadequate over-reactivity in HR and HRV to certain tasks and stimuli.

So far, most studies have focused on HF-HRV or HR differences among individuals with one anxiety disorder (versus controls) or have compared two anxiety disorder groups (Asmundson and Stein, 1994; Friedman and Thayer, 1998b). A notable exception investigated more than two anxiety disorders within a single sample, but did not investigate HF-HRV in response to anxiety-related provocation (Licht et al., 2009). While yielding important information, this narrow focus limits the capacity to examine HF-HRV and HR differences across the full spectrum of the anxiety disorders. Including a broader range of anxiety disorders would enable investigation of disorder-specific versus shared features of cardiac psychophysiology. A finding of similar features of cardiac psychophysiology across different nosological categories would favor a functional perspective of anxiety disorders (van Praag et al., 1990).

The present study, therefore, examined HF-HRV and HR differences among individuals with a DSM-IV (American Psychiatric Association, 2000) anxiety disorder at baseline and in response to two distinct anxiety-related experimental tasks, hyperventilation and relaxation. A recent finding might suggest that association between anxiety disorders and low HRV is accounted for by antidepressant medication use (Licht et al., 2009). This finding calls into question the theorized mechanism of HRV and HR abnormalities in anxiety disorders, and at the very least, suggests that analyses should account for different covariates like psychotropic medication use as well as comorbid mood disorders (Rottenberg, 2007). Therefore, we tested and controlled for the effects of psychotropic medication use and comorbid mood disorders, as well as the contributions of age, sex, and respiration, each of which can affect HRV (Grossman et al., 2001; Burriss et al., 2007; Rottenberg, 2007; Licht et al., 2009). In sum, the present study investigated the following questions: (1) Do patients with an anxiety disorder (all anxiety disorder patients combined) show lower HF-HRV or higher HR at resting baseline than a non-anxious control group (CG)?; (2) Are these differences evident across the different subdivided anxiety disorder groups (ADGs; PD, GAD, SAD, OCD)?; (3) Do anxiety disorder patients combined and

the subdivided ADGs show lower HF-HRV responses and/or greater HR responses during the two anxiety-related tasks than the CG?; (4) Is lower HF-HRV and/or higher HR associated with higher symptom severity?; and (5) Are potential differences in HF-HRV and HR solely explained by relevant demographic variables (age, sex), differences in respiration (for HF-HRV) or certain clinical variables (comorbid mood disorder or psychotropic medication use)?

2. Materials and methods

2.1. Participants

The current study included 131 participants, including 82 treatment-seeking patients with a primary DSM-IV anxiety disorder and 39 non-anxious controls. The UCLA Internal Review Board approved all study procedures and all participants gave informed consent. Participants were originally recruited for a clinical trial study comparing cognitive behavioral and acceptance and commitment therapy (see Arch et al., 2012). Data presented herein reflect the pretreatment laboratory assessment. Table 1 shows the clinical and demographic characteristics of the CG and the ADGs. Anxiety disorder patients were diagnosed with the Anxiety Disorders Interview Schedule-IV (ADIS-IV; Brown et al., 1994). They were included if they met DSM-IV criteria for PD with or without agoraphobia, SAD, GAD, or OCD with a clinical severity rating (CSR) of four or higher. The CSR is an interviewer rating made on a 0 to 8 scale based on current symptom severity, distress, and disablement (0 = none, 2 = subclinical, 4 = clinically significant, 6 = moderately severe, 8 = most severe). Patients were stabilized on psychotropic medications for a minimum length of time (1 month for benzodiazepines and beta blockers, 3 months for heterocyclics and SSRIs/SNRIs) or medication free. Exclusion criteria for the ADGs included a history of psychiatric hospitalization in the last five years, active suicidal ideation, severe depression (clinical severity rating > 6 on ADIS-IV, see below), or a history of bipolar disorder, psychosis, mental retardation or organic brain damage. In all groups, individuals with substance abuse or dependence within the last 6 months, or with any severe diseases or pregnancy were excluded. CG participants were medication free and were administered the Mini International Neuropsychiatric Interview (MINI) for DSM-IV (Lecrubier et al., 1997; Sheehan et al., 1998) to rule out any mental disorders, including anxiety disorders (for further details see Arch et al., 2012).

If patients met criteria for multiple anxiety disorders, they were assigned to more than one ADG to increase statistical power. The number of patients with primary diagnosis and specific comorbidities in each subdivided ADG are shown in Table 2. Thus, the subdivided ADGs do not represent pure individual disorders, but rather subgroups of patients with shared symptomatology and different comorbidities. Due to the overlapping ADGs, each ADG was compared to the CG, but subdivided ADGs were not compared to one another. There were no significant differences between the CG and anxiety disorders patients combined, as well as any of the subdivided ADGs in age, all $ps > .16$, years of education, all $ps > .09$, sex ratio, all $ps > .29$, or ethnicity, all $ps > .08$.

2.2. Questionnaire battery

The following questionnaires were used to measure disorder-specific symptom severity: (a) the Anxiety Sensitivity Index (ASI; Reiss et al., 1986; Peterson and Reiss, 1992) was used as a panic-specific measure of severity, because it relates most strongly to panic symptoms; (b) the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) was used as GAD-specific measure; (c) the revised Padua Inventory (Burns et al., 1996) was used to measure severity of obsessive and compulsive symptoms common in OCD; and (d) the Fear Questionnaire-Social Phobia Subscale (FQ-S; Marks and Mathews, 1979) was used as a SAD-specific measure of severity.

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