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The Relationship between Social Anxiety and Heartbeat Evoked Potential Amplitude

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Highlights

- Social anxiety relates with HEP during false heartrate acceleration feedback
- Concerns about the social consequences of anxiety explained this relationship
- Social anxiety involves early cortical abnormalities in processing heartbeats

Abstract

Past research suggests that social anxiety is associated with increased processing of cardiac activity. Cognitive theories propose that this is one aspect of self-focus, which is driven by concerns that features of the self, such as anxiety symptoms, will elicit evaluation from others. We investigated the relationship of social anxiety to the cortical processing of heartbeats as reflected in the heartbeat evoked potential (HEP) during false feedback of accelerated heart rate. Thirty-eight participants with high social anxiety (HSA; n = 19) and non-elevated social anxiety (NSA; n = 19) completed a cognitive task during which false feedback of accelerated heart rate was randomly provided on 50% of trials. HEP amplitude was larger in HSAs, but not NSAs, during false heartbeat acceleration cues compared to standard cues. HEP amplitude also was larger in HSAs compared to NSAs during acceleration cues. HEP amplitude during acceleration cues, but not standard cues correlated with social anxiety. Within the first second after the R-peak, social anxiety correlated with voltage at *Fz* from 223-305 ms. Social concerns about the consequences of anxiety symptoms accounted for an indirect relationship between social anxiety and the HEP. These data extend prior evidence of increased processing of cardiac activity in socially anxious individuals, providing support for cognitive theories.

Keywords: social anxiety, heartbeat evoked potential, event-related potential, anxiety sensitivity

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The Relationship between Social Anxiety and Heartbeat Evoked Potential Amplitude

Self-focus plays a prominent role in cognitive theories of social anxiety disorder (SAD; Clark & McManus, 2001; Heimberg, Brozovich, & Rapee, 2010; Hofmann, 2007). Self-focus is characterized by increased attention to features of the self that are believed to elicit rejection by others (see Spurr & Stopa, 2002 for a review). These features include somatic symptoms of anxious arousal, such as increased heart rate (Pineles & Mineka, 2005). Although somatic sensitivity has been noted across anxiety disorders (see Domschke, Stevens, Pfleiderer, & Gerlach, 2010), individuals with social anxiety disorder report greater concerns about the social consequences of somatic symptoms compared to individuals with other anxiety disorders (Taylor et al., 2007). Thus, theory and data point toward a specific relationship between social anxiety and self-focus on somatic symptoms.

Research has investigated the processing of cardiac activity as one aspect of self-focus. Socially anxious individuals are more accurate at perceiving heart rate and heart rate changes (Johansson & Öst, 1982; Stevens et al., 2011). They report increased perception of heart palpitations during both social conversation and guided imagination of social situations, despite comparable heart rate with non-anxious controls (Edelmann & Baker, 2002). Socially anxious individuals also exhibit biased attention to ostensible snapshots of their electrocardiogram (ECG) compared to individuals without elevated social anxiety (Mills, Grant, Judah, & White, 2014; Pineles & Mineka, 2005).

Studies using false heart rate feedback further support the case for abnormalities in the processing of cardiac activity in socially anxious individuals. In response to false feedback of accelerated heart rate, individuals with SAD report increased anxiety and self-focus (Wells &

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Papageorgiou, 2001). During a social interaction, false feedback of accelerated heart rate compared to decreased heart rate results in greater self-report of anxiety and an overestimation of the visibility of somatic anxiety symptoms to observers (Wild, Clark, Ehlers, & McManus, 2008). Similar effects have been found in studies using impromptu speech tasks, with false feedback of accelerated heart rate resulting in increased self-focus and negative affect (Makkar & Grisham, 2013). Socially anxious individuals report more anxiety when their heartbeats are played back on a loudspeaker compared to headphones, suggesting their anxiety may be related to concerns about the social consequences of their heart rate (Gerlach, Mourlane, & Rist, 2004). These findings support cognitive theories, which propose that social anxiety is related to concerns about potential social consequences of noticeable somatic symptoms. But little is known about the neurophysiological processing of heartbeats in socially anxious individuals.

Research has begun to examine event-related potential (ERP) components to understand how socially anxious individuals process cardiac activity. Judah, Grant, and Carlisle (2016) found that social anxiety correlated with enhancement of the P2 elicited by false feedback cues of accelerated heart rate, suggesting increased attention to heart rate acceleration cues. Although this finding is consistent with cognitive models, it suggests externally-directed attention toward a physiologically significant stimulus rather than interoceptive processing of the heartbeats. Research using a direct measure of the cortical processing of cardiac activity is needed. Studies have not examined the possibility that abnormal cognitive responses to cardiac activity in socially anxious individuals may include early cortical processing of heartbeats. Addressing this gap may clarify whether self-focus in social anxiety involves abnormalities in the early neurophysiological processing of heartbeats.

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Understanding the cortical processing of cardiac activity in social anxiety may be advanced through studies using the heartbeat evoked potential (HEP). During states of attention toward heartbeats, the HEP is enhanced, reflecting the allocation of attention to cardiac activity (Schandry, Sparrer, & Weitkunat, 1986; Pollatos & Schandry, 2004; Yoris et al., 2017). Source estimation of the HEP is consistent with fMRI and electrocorticography data in suggesting that attention to heartbeats is associated with increased activity in brain areas linked to interoception, namely the insula, anterior cingulate, and somatosensory cortex (Couto et al., 2015; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Kern, Aertsen, Schlze-Bonhage, & Ball, 2013; Park et al., 2017; Pollatos, Kirsch, & Schandry, 2005), which receive sensory signals from mechanoreceptors in the heart walls (Shepherd, 1985) and arteries (i.e., baroreceptors; Weisz et al., 2001), as well as somatosensory afferents in the chest (Khalsa et al., 2009). Early HEP activity is evident as a prominent negative deflection peaking 200-300 ms after the R-peak at fronto-central electrodes (e.g., Canales-Johnson et al., 2015; Schandry et al., 1986). In an early study, the HEP was enhanced in a heartbeat counting task relative to baseline (Schandry et al., 1986). These results were replicated in subsequent studies, which found that focusing attention on heartbeats (e.g., counting heartbeats compared to counting tones) increased HEP amplitude (Montoya, Schandry, & Müller, 1993). These studies suggest that the HEP may be a useful tool to investigate early cortical processing of heartbeats, an approach that could advance the understanding of social anxiety. As HEP amplitude is increased by attentiveness to cardiac activity, and false feedback of heart rate acceleration increases self-focus in socially anxious individuals, it may be expected that socially anxious individuals may show an increase in HEP amplitude during false feedback of accelerated heart rate.

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The primary purpose of this study was to examine how social anxiety and HEP amplitude are related during false feedback of accelerated heart rate. We hypothesized that social anxiety would be associated with increased HEP amplitude during false heart rate acceleration feedback. A secondary goal was to examine whether concerns about the social consequences of somatic symptoms explained the relationship between social anxiety and HEP amplitude.

Method

Participants

This study involved secondary analysis of data from a sample used in a published study (Judah, Grant, & Carlisle, 2016). Participants were recruited using an online research participation system at a large Mid-western university from a pool of 3,574 students (SIAS skew = 1.01; kurtosis = .71). Out of the 42 participants reported by Judah, Grant, and Carlisle (2016), four were excluded from this study due to missing or poor-quality ECG. This resulted in a final sample of 38 participants (25 female) with a mean age of 19.3 (*SD* = 4.05). Participants identified as white (*n* = 31), black or African-American (*n* = 3), Native American (*n* = 1), or preferred not to report their racial identity (*n* = 3). Participants were divided into groups based on social anxiety cutoffs used in prior studies (Judah et al., 2013; Judah et al., 2016; Judah, Grant, and Carlisle, 2016). Participants scoring one standard deviation (i.e., 30) or more above the undergraduate student mean reported by Mattick and Clarke (1998) for the Social Interaction Anxiety Scale (were considered the "high social anxiety" group. Participants scoring at or below the undergraduate mean (i.e., 19) reported by Mattick and Clark (1998) were considered the "non-elevated social anxiety" group (see Judah, Grant, & Carlisle, 2016). The selection of participants at the mean and below was meant to constitute a comparison group that does not

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have elevated social anxiety, in keeping with the goal of understanding excessive social anxiety as the core feature of social anxiety disorder.

Measures

Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998). This 20-item self-report measure asks participants to choose how much they identify with fears related to social interactions. On this five-point Likert-type scale, higher scores indicate higher levels of social anxiety. Internal consistency for this study was excellent (Cronbach's alpha = .94).

Anxiety Sensitivity Inventory (ASI-III; Taylor et al., 2007). On this 18-item self-report measure, participants rate the extent to which they identify with statements describing symptoms and situations of general anxiety. It uses a five-point Likert-type scale, with higher scores reflecting higher levels of general anxiety. For this study, internal consistency was high for both the total scale (α = .89) and the social concerns subscale (α = .81).

Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). On this 21-item self-report measure, participants indicate which statements they find most characteristic of themselves during the previous two weeks. Each statement covers a different depressive symptom, with statements ranging from absence of symptoms to severe. Internal consistency for this study was high (α = .86).

Procedure

All procedures were approved by the Institutional Review Board at the large university where the data were collected. Participants completed a modified change detection task, the details and results of which are described in a previous publication (Judah, Grant, & Carlisle, 2016). Participants viewed a sequence of face stimulus arrays with two neural faces on one side

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and two disgust faces on the other. The faces disappeared, followed by a centrally-located arrow pointing to one side of the screen. A new array of faces then appeared. Participants were tasked with indicating whether a face had changed on the side to which the arrow pointed.

More germane to the aims of the present research question, false heart rate feedback was presented randomly during 50% of the total 1,024 trials, which each lasted 3.000 ms. This feedback was presented in the form of a variation of the standard fixation cross (+), which participants were told would appear as "×" if their heart rate increased. The meaning of the cues was counterbalanced between participants. False feedback of accelerated heart rate has been found to increase attention to heartbeats, as suggested by improved accuracy of heartbeat counting (Brener, Knapp, & Ring, 1995)

After completing the change detection task, participants were asked three questions about the false heart rate feedback to verify the manipulation. These results are reported in another publication (Judah, Grant, & Carlisle, 2016). As noted in this previous study, all participants recalled that the fixation symbol change was due to an increase in their heart rate.

Physiological data processing

ECG data were collected using a modified Lead II electrode placement (electrodes placed on the lower left ribcage and above the right collarbone; Stern, Ray, & Quigley, 2001). Physiological data were sampled at 256 Hz using a 32-channel ActiveTwo BioSemi system. EEG data were filtered online with a band pass filter of .01-100 Hz, then filtered offline from .1-30 Hz and re-referenced to the average of the mastoid electrodes. Independent components analysis (ICA) was used to identify and remove ocular artifacts. Artifact rejection is detailed in Judah, Grant, & Carlisle, (2016). Within each trial (3,000 ms duration) EEG was epoched from -

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200 to 1000 ms relative to all R-peaks occurring within the first 2,000 ms. The average number of epochs per participant was 3,218.50 (*SD* = 702.77) with an average of 324.82 (*SD* = 111.05) epochs rejected due to artifact. Groups did not differ in the total number of epochs, $t = 1.31$, $p = .20$, or epochs rejected, $t = .65$, $p = .52$.

Due to the prominent cardiac field artifact within the first 450 ms after the R-peak, most studies have used artifact correction, such as subtraction of the scaled ECG from each electroencephalogram (EEG) channel (Fukushima, Terasawa, & Umeda, 2011; Montoya et al., 1993; Schandry & Weitkunat, 1990), or measured HEP activity after the T-wave (Baranauskas, Grabauskaite, & Griskova-Bulanova, 2017; Dirlich, Dietl, Vogl, & Strian, 1998; Gray et al., 2007). To reduce cardiac field artifact in the EEG, artifact cleaning procedures used in prior HEP studies were employed (Fukushima et al., 2011; Montoya et al., 1993; Schandry & Weitkunat, 1990). The ECG was scaled to the EEG at each channel according to values at 0 ms with a baseline of -50 to 50 ms on either side (i.e., the R-peak) and subtracted from each EEG channel to correct for ECG contamination. All analyses were conducted using the uncorrected data as well to verify that the results were not incidental to this artifact correction procedure. In all cases, the pattern and statistical significance of the results did not differ between the corrected and uncorrected data. The results reported here detail those using the corrected data.

HEPs have been observed with various timing, scalp topography, and polarity (Gray et al., 2007; Müller et al., 2015; Pollatos & Schandry, 2004; Schulz et al., 2015). These differences might result from the multiple receptor types that underlie heartbeat sensations (e.g., atrial mechanoreceptors are stimulated sooner than baroreceptors), methodological differences (e.g., task conditions, referencing, correction procedures for cardiac field artifacts), and/or the

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existence of multiple ERP components evoked by heartbeats, but this issues has not been resolved in the literature. To characterize the HEP for our study, visual inspection of the waveforms and scalp maps determined the HEP to be a negative deflection maximal at *Fz* from about 200-350 ms after the peak of the R-wave, which is consistent with timing, polarity, and scalp topography in many prior studies (e.g., Canales-Johnson et al., 2015; Fukushima et al., 2011; Schandry et al., 1986). Thus, the HEP was measured as a negative deflection at *Fz* from 200-350 ms. Baseline correction was performed by subtracting the mean voltage in the 200 ms prior to the peak of the R-wave.

Analytic Approach

Zero-order correlations between the HEP, ECG, and self-report measures were examined. Repeated-measures ANOVA was used to investigate the effect of Group (HSA, NSA) and the within-participants effect of Cue (Heart Rate Acceleration, Standard) on HEP amplitude. To confirm the timing of the HEP relationship with social anxiety, SIAS scores were correlated with each sample of the ERP in the first second after heart rate acceleration cues. Simple effects analysis was used in follow-up tests. To ensure that the effects were due to the psychological meaning of the cues (i.e., accelerated heart rate) and not their physical characteristics, the ANOVA was repeated with the physical characteristics of Cue (+, ×). To ensure that effects were not explainable by cardiac artifact or activity, the main ANOVA was repeated with ECG and heart rate as dependent variables. To test the hypothesis that ASI Social Concerns would explain the relationship between SIAS and HEP amplitude during acceleration cues, a mediation model with bias-corrected bootstrapping (5,000 samples) was examined using the PROCESS macro in

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SPSS (Hayes, 2015). Heart rate during acceleration cues was included as a covariate due to its moderate correlation with HEP amplitude.

Results[1]

HEP and ECG correlations

As expected, all HEP measures were highly correlated with one another, r = .68 to .91, p < .001, and all ECG measures were highly correlated with each other, r = .93 to .99, p < .001. Notably, the HEP and ECG measures were not correlated, rs = .02 to .16, ps = .33 to .89.

HEP and self-report correlations

HEP amplitude (which is negative) during trials with heart rate acceleration cues was significantly correlated with SIAS score, r = -.39. p = .017 (see Figure 1), and with both the ASI total score, r = -.39. p = .015, and the Social Concerns subscale, r = -.38. p = .018 (see Figure 2). The HEP during standard cues was not correlated with any self-report measures. All self-report measures (SIAS, BDI, and ASI and its subscales) were positively correlated with one another, rs = .35 to .46, ps < .05, except that the BDI was not related with ASI total score or ASI Social Concerns, and SIAS was not related with ASI Physical Concerns. Heart rates during acceleration and standard cues were correlated with one another, r = .99, p < .001, and with HEP amplitude, rs = .38 to .42, ps < .05. Supporting the specificity of the symptom correlations with HEP rather than the cardiac field, ECG peak and mean amplitude within the HEP measurement window were not correlated with scores on the SIAS, the BDI, or the ASI and its subscales (ps = .25 to .99).

[1] To enable comparison with other studies, all analyses were repeated using a SIAS cutoff score of 34 or higher (Heimberg, Mueller, Holt, Hope, & Liebowitz, 1992), which resulted in the removal of six subjects. Notably, all statistically significant findings remained significant, including correlations, ANOVA, and mediation findings. No new significant findings were observed.

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To characterize the time course of the relationship between SIAS scores and HEP amplitude during acceleration cues, correlations were examined between SIAS score and each EEG sample after the R-peak (see Figure 3). A significant negative correlation was observed between the SIAS and each sample from 223 ms to 305 ms, confirming the measurement window that had been selected based on prior studies. No other samples in the epoch were significantly correlated with SIAS scores.

Group and cue comparisons

There was a significant interaction between Group and Cue, $F(1, 36) = 7.13$, $p = .011$, $\eta_p^2 = .17$ (see Figure 4). Simple effects analysis revealed that HEP amplitude was larger (i.e., more negative) for HSA participants during acceleration cues ($M = -1.22$, $SD = 1.12$) compared to standard cues ($M = -.96$, $SD = 1.29$); $t = -2.37$, $p = .029$, $d = .22$). HEP did not differ between cues for NSA participants, $p = .21$. HEP during acceleration cues was also significantly greater (i.e., more negative) for HSA participants ($M = -1.22$, $SD = 1.12$) compared to NSA participants ($M = -.42$, $SD = .90$; $t = -2.44$, $p = .020$, $d = .79$), but HEP did not differ between groups during standard cues, $p = .29$. Neither the main effect of Group, $F(1, 36) = 3.04$, $p = .09$, $\eta_p^2 = .08$, nor that of Cue, $F(1, 36) = .73$, $p = .40$, $\eta_p^2 = .02$, were significant.

Follow-up tests

As expected, there was no main effect for the physical characteristics of the cues, $F(1, 36) = .63$, $p = .43$, nor did this interaction with Group, $F(1, 36) = .47$, $p = .50$. There was no significant main effect of Group, $F(1, 36) = .34$, $p = .56$, nor Cue, $F(1, 36) = .09$, $p = .77$, on mean amplitude of the ECG within the HEP time window. The interaction between Group and Cue was not significant, $F(1, 36) = .33$, $p = .57$. Thus, the interaction effect observed in the HEP

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was not evident in the ECG. Similarly, all effects were not significant on the peak ECG amplitude within the measurement window, *F*s < 1.0. Heart rate did not differ between cues, $F(1, 36) = 1.24$, $p = .27$. Similarly, the social anxiety groups did not differ significantly, nor was there an interaction between Group and Cue, *F*s < 1.

Indirect effect through ASI Social Concerns

Skewness values (all < 1.4) and kurtosis values (all < 2.3) suggested a normal distribution for each variable (Kline, 2011). The homoscedasticity assumption was supported by the non-significance of the Breusch-Pagan, $\chi^2 = .82$, $p = .66$, and Koenker test, $\chi^2 = .58$, $p = .75$. Both the direct effect, $\beta = -.02$, LLCI = -.04, ULCI = -.002, and the indirect effect, $\beta = -.01$, LLCI = -.02, ULCI = -.001, were significant with 95% confidence intervals that did not include zero. Together, the direct and indirect effects accounted for approximately 32% of the variance in HEP amplitude.

Discussion

Our findings indicate that social anxiety is associated with increased HEP amplitude during the presentation of false feedback that heart rate has accelerated. Our results are not explainable as a global increase in the cortical representation of heartbeats (HEP) in HSAs because the effects were dependent on the presence of false heart rate feedback. Social anxiety was correlated with mean HEP amplitude at *Fz* from 200-350 ms during false feedback of accelerated heart rate. To better characterize the timing of this effect, correlations between social anxiety and each sample in the HEP epoch were examined. Social anxiety was correlated with each sample in the HEP from 223-305 ms. Taking an extreme groups approach, socially anxious individuals showed increased HEP amplitude when cued that heart rate was elevated compared

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to trials without these cues, but no difference was observed in participants with non-elevated social anxiety. Socially anxious participants also showed greater HEP amplitude compared to participants with non-elevated social anxiety during cues that heart rate was elevated.

Our findings contribute to cognitive frameworks that view the processing of somatic symptoms as a core feature of social anxiety (e.g., Clark & Wells, 1995; Spence & Rapee, 2016). Identifying an early difference in the cortical representation of heartbeats in socially anxious individuals builds upon past findings of heightened attention toward external stimuli related to cardiac activity, such as attentional bias toward ECG snapshots (Mills et al., 2014) and attention to false heart rate feedback cues (Judah, Grant, & Carlisle, 2016; Judah, Grant, Mills, & Lechner, 2013;). Our findings also complement evidence that socially anxious individuals are better perceivers of their heartbeats than individuals low in social anxiety (Stevens et al., 2011). Similar findings for other anxiety conditions have led to the proposal that interoceptive sensitivity to cardiac sensations may be an intermediate phenotype of anxiety disorders (Domschke et al., 2010). In the case of social anxiety, attention to cardiac sensations relates to worries about the visibility of somatic symptoms to others (Gerlach et al., 2004). Our data suggest that abnormal processing of heartbeats in socially anxious individuals is present early in the cortical representation of heartbeat sensations. Our findings also complement neuroimaging evidence that socially anxious individuals exhibit heighted activity in the insula, an area identified as an HEP source (Park et al., 2017), while evaluating somatic statements (e.g., “I have a fast pulse”) (Terasawa, Shibata, Moriguchi, & Umeda, 2013).

Theories of social anxiety propose that the processing of somatic symptoms of anxiety, such as cardiac activity, may affect attention to stimuli in the environment, either reducing the

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ability to attend to external stimuli (Clark & Wells, 1995) or enhancing the salience of stimuli that signal possible rejection (Rapee & Heimberg, 1997). Studies have shown that stimulus processing is partially gated by cardiac phases. During the contraction phase of the heart (i.e., systole), fearful faces are detected more easily, and both disgust (Gray et al., 2012) and fear faces are rated to be more intense compared to the period between contractions (i.e., diastole); Garfinkel et al., 2014). Similarly, HEP activity predicts the detection of visual stimuli presented just after the heart beats (Park, Correia, Ducorps, & Tallon-Baudry, 2014). Future studies informed by such research and our findings are needed to test and advance theories about how attention to heartbeats affects the processing of stimuli in the context of social anxiety.

We found that the association between social anxiety and the HEP was accounted for by a particular component of anxiety sensitivity, namely social concerns. Several studies have found that fears of experiencing publicly observable symptoms are uniquely associated with social anxiety symptoms and SAD (Deacon & Abramowitz, 2006; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012). Our results expand on previous studies by identifying specific consequences of anxiety sensitivity on cognitive processing. Anxiety about somatic symptoms evoking negative evaluation may direct neural resources towards or away from the processing of cardiac activity. Based on prior studies evaluating social anxiety and cognitive processing (e.g., Judah, Grant, & Carlisle, 2016; Judah et al., 2013), these data contribute to the growing literature documenting the association of social anxiety with self-focus, suggesting that physiological indicators of interoception are worthy of further investigation.

Beyond social anxiety, abnormal processing of cardiac activity has been noted in other anxiety disorders. Although heartbeat detection paradigms have been used to investigate anxiety

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disorders (e.g., Dunn et al., 2010), neural mechanisms remain understudied. The HEP only recently has been investigated in the context of obsessive-compulsive disorder (OCD), revealing enhanced HEP in OCD patients compared to patients with panic disorder and to healthy controls (Yoris et al., 2017). Notably, reduced HEP amplitude has been observed in depression (Terhaar, Viola, Bar, & Debener, 2012) and borderline personality disorder (Müller et al., 2015). Though the investigation of HEP activity in the context of other anxiety disorders lies beyond the scope of this study, such research is needed given the preponderance of studies suggesting the role of anxiety sensitivity and related constructs across anxiety disorders (Boswell et al., 2013). Indeed, our findings suggested a correlation between HEP amplitude and anxiety sensitivity, specifically the sub-domain comprised of concerns about the social consequences of anxiety symptoms. The lack of a significant relationship with the ASI Physical Concerns subscale may be due to the manipulation used in this study, which did not propose any health consequences of accelerated heart rate to the participants. Future research is needed to clarify how HEP components can be used to increase understanding of cardioception in anxiety disorders.

Several alternative explanations of the HEP effects were considered. In addition to artifact correction to remove cardiac field artifact, analyses were included to test the possibility of effects on the ECG. Neither the mean nor peak amplitudes of the HEP were correlated with corresponding ECG measures. Conducting the analyses on the ECG rather than the HEP revealed no significant effects on mean or peak amplitude. Heart rate was correlated with HEP amplitude, but high and non-elevated social anxiety groups did not differ in heart rate in either cue condition, nor did heart rate differ between the cue conditions. When considering previous studies (e.g., Edelmann & Baker, 2002), these analyses offer further support to the hypothesis

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that socially anxious individuals display evidence of early cortical sensitivity to cardiac information, irrespective of actual changes in heart rate. The correlation between heart rate and HEP that we found has not been reported in prior studies to our knowledge. This correlation could be due to a modulation of both sympathetic response (e.g., heart rate) and interoceptive sensitivity (e.g., HEP) by certain stimuli. The finding also suggests that researchers should take into account the relationship between heart rate and HEPs.

Several limitations apply to our conclusions. The study relied on a small sample of undergraduate students recruited to facilitate an extreme groups comparison based on social anxiety. We did not assess relationships with broader measures of anxiety; therefore, the extent to which the effects noted in this study are specific to social anxiety remains unclear. As noted previously, elevated HEP has been observed in OCD, though not in panic disorder (Yoris et al., 2017), and is reduced in depression (Terhaar, et al., 2012), suggesting that the HEP may show some differences across emotional disorders. Artifacts of the cardiac field, particularly the T wave, and vascular dynamics can offer alternative explanations of HEP activity. However, this concern is reduced by our artifact correction approach and the lack of correlations between the ECG with the HEP and with the symptom measures. In addition, the timing and condition specificity of the HEP relationship with social anxiety partially mitigates these limitations. The various temporal and topographical characteristics of HEPs limit the conclusions that can be drawn from our data. HEPs have been observed with positive polarity in a number of studies (e.g., Leopold and Schandry, 2001; Montoya et al., 1993; Pollatos and Schandry, 2004); therefore, it is not known whether our HEP effects indicate increased or reduced processing of heartbeats. The conclusion that our effects indicate increased attention to heartbeats cannot be

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drawn from our results because attention to heartbeats was not assessed. However, the possibility that attention to heartbeats modulated the ERP effects we observed is consistent with evidence that false feedback of accelerated heart rate increases attention to heartbeats (Brener et al., 1995). Additional research is needed to clarify how HEP abnormalities in socially anxious individuals correspond with attention to heartbeats.

In summary, these data support and extend cognitive theories of social anxiety disorder. Consistent with theoretical predictions of increased self-focus in social anxiety, we found that social anxiety correlated with enhanced amplitude of the HEP during false feedback of accelerated heart rate. Furthermore, this relationship was partially explained by concerns about the social consequences of anxiety symptoms. Additional research examining the HEP may offer further insight into abnormal processing of cardiac activity in social anxiety and other anxiety disorders.

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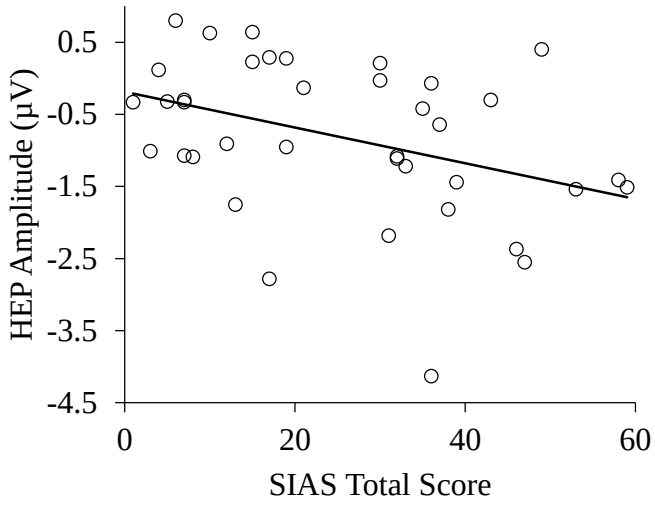

Figure 1. Correlation between HEP during self-focus trials and SIAS scores.

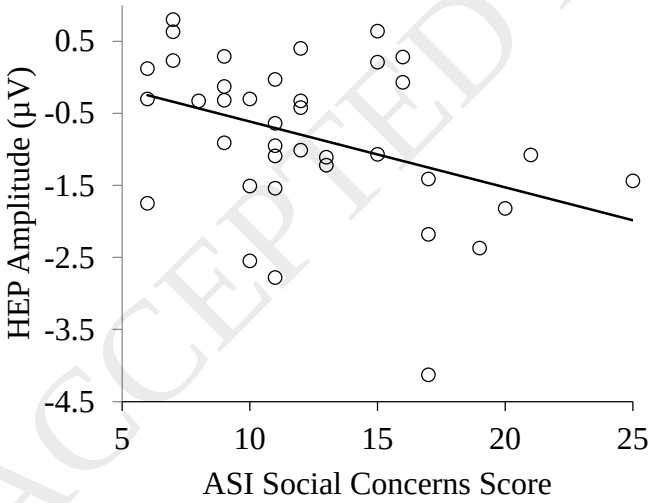

Figure 2. Correlation between HEP during self-focus trials and ASI Social Concerns scores.

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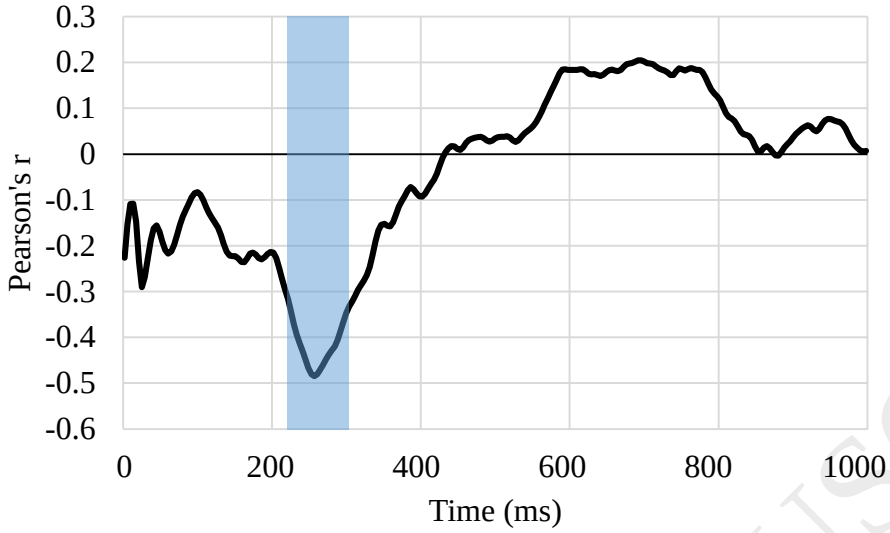

Figure 3. Correlation between HEP during self-focus trials (*Fz*) and SIAS scores. Shading denotes region of significance ($p < .05$) from 223-305 ms.

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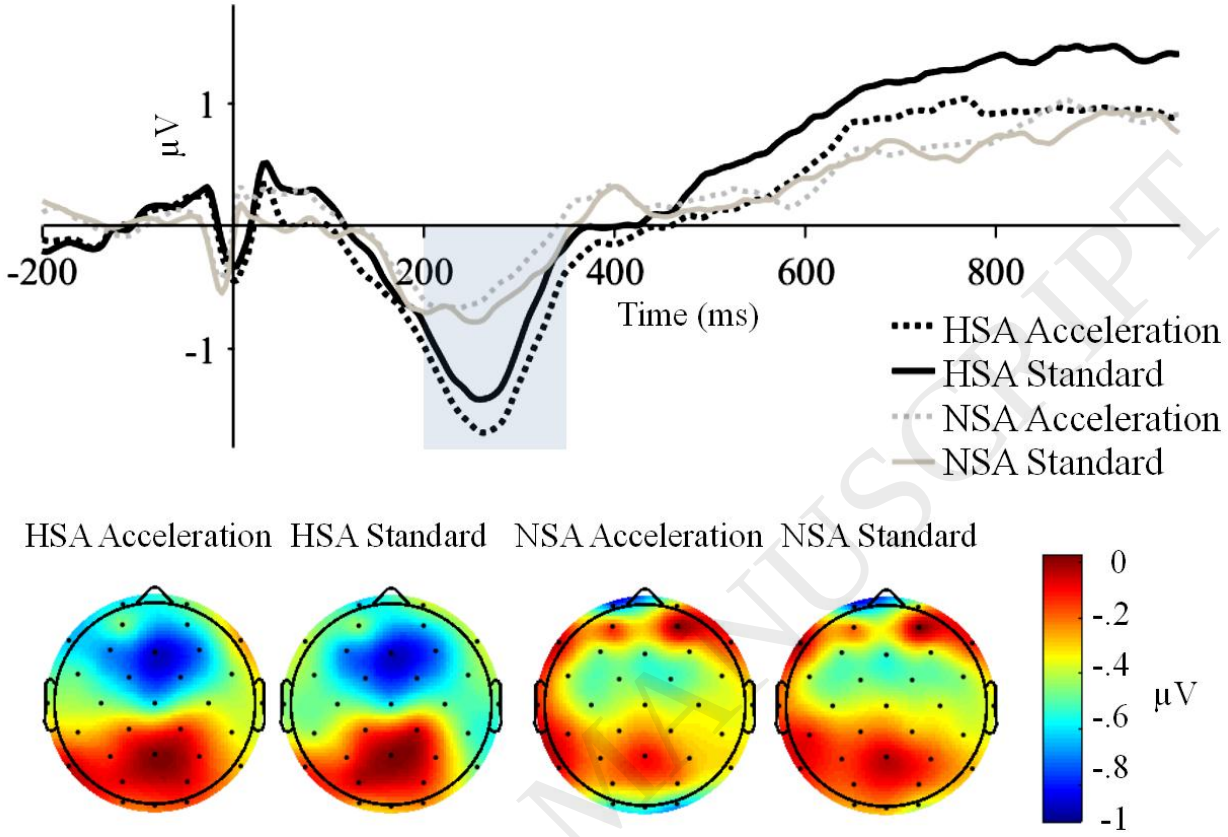

Figure 4. Heartbeat evoked potential (HEP) at *Fz*. Scalp maps of HEP mean amplitude within the measurement window (200-350 ms) denoted by shaded box.

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