

# The Worried Mind: Autonomic and Prefrontal Activation During Worrying

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To study the psychophysiological correlates of worrying, the authors recorded heart rate, respiratory sinus arrhythmia (RSA), skin conductance level, and alpha electroencephalographic asymmetry in healthy males during baseline, relaxation, worry induction, and anticipation of an impromptu speech task. Compared with baseline, relaxation, and anticipation, worrying was associated with greater heart rate and lower RSA. Worrying was further characterized by higher skin conductance levels compared with baseline but lower levels than during anticipation. Finally, worrying was associated with relatively greater left frontal activity compared with anticipation. Trait public speaking anxiety was positively correlated with left frontal activity during worrying. These results support the notion that worrying is a unique emotional state that is different from fearful anticipation.

*Keywords:* EEG, hemispheric asymmetry, psychophysiology, worry, heart rate variability

In recent years, the investigation of biological correlates of normative and pathological responses to anxiety-provoking cues has attracted increased attention (for reviews, see Nitschke & Heller, 2002; Nitschke, Heller, & Miller, 2000). Several studies suggest that anxiety is associated with cortical hemisphere asymmetries (e.g., Dien, 1998; Heller, Nitschke, Etienne, & Miller, 1997), although the evidence is mixed (Giordani et al., 1990). In addition, anxiety is often associated with cardiovascular and electrodermal symptoms that reflect autonomic nervous system activity (e.g., Lang, Davis, & Ohman, 2000). However, there is very little integration between the neuroimaging and electroencephalographic (EEG) literature and studies on autonomic indicators of anxiety (for a review, see Keller, Hicks, & Miller, 2000; Zahn, 1986). Furthermore, very few studies have examined both cortical correlates and autonomic indicators during experimentally induced anxiety states (Davidson, Marshall, Tomarken, & Henriques, 2000).

Early investigations of hemispheric laterality point to left-hemisphere hyperactivity in anxiety (Tucker, Antes, Stenslie, & Barnhardt, 1978; Tyler & Tucker, 1982). Consistent with these behavioral findings, EEG (Carter, Johnson, & Borkovec, 1986) and hemodynamic (Baxter et al., 1988; Fredrikson et al., 1993; Swedo et al., 1989; Wu et al., 1991) studies report greater left-hemisphere activity during anxious states. In contrast, other re-

search suggests that anxiety is more likely associated with greater right-hemisphere activity. This finding has been reported for patients with panic disorder (Reiman, Raichle, Butler, Herscovitch, & Robins, 1984), posttraumatic stress disorder (PTSD; Bremner et al., 1999; Pissioti et al., 2002), social phobia (Davidson et al., 2000; Tillfors et al., 2001), and spider phobia (Paquette et al., 2003) as well as in nonclinical populations of high trait-anxious individuals (Nitschke, Heller, Palmieri, & Miller, 1999; Reivich, Gur, & Alavi, 1983).

In light of these inconsistent findings, some authors have proposed the existence of different subtypes of anxiety (Gruzelić, 1989; Heller, Etienne, & Miller, 1995; Heller et al., 1997; Nitschke & Heller, 2002). More specifically, it has been hypothesized that the left hemisphere is more involved when there are strong verbal and cognitive components associated with the anxious emotional state, such as in worrying (Heller et al., 1997). In contrast, the right hemisphere is assumed to be more involved during anticipation of imminent threat. Specifically, Heller et al. (1997) distinguish between anxious apprehension and anxious arousal. *Anxious apprehension* is defined as a state of anxiety characterized predominantly by verbal and cognitive components and directed toward future negative events. In contrast, *anxious arousal* is characterized primarily by a somatic fear response and heightened physiological arousal. Supporting evidence for this distinction comes from Heller et al.'s (1997) experiment. The authors found relatively greater right parietal activation during anxious arousal and larger frontal asymmetry in favor of the left hemisphere during a task designed to induce anxious apprehension. Similarly, in generalized anxiety disorder (GAD), a condition prominently characterized by worry and verbal rumination, evidence of increased left frontal activation has been reported (Wu et al., 1991). Conversely, additional support for the role of the right parietal region in anxious arousal comes from a study by Nitschke et al. (1999), who were, however, unable to replicate Heller et al.'s (1997) finding

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that anxious apprehension led to increased left-hemisphere activation.

Worrying is the underlying pathological cognitive process of anxious apprehension and has been associated with reduced autonomic flexibility as a result of low cardiac vagal tone (Borkovec & Hu, 1990; Hoehn-Saric & McLeod, 2000; Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, & Borkovec, 1996). For example, Thayer et al. (1996) showed that, relative to baseline and relaxation conditions, experimentally induced worrying was associated with higher heart rate but lower high-frequency spectral power, which is an indicator of cardiac vagal tone. Other studies, however, were unable to demonstrate the effect of worrying on cardiac activity (Davis, Montgomery, & Wilson, 2002; Hazlett-Stevens & Borkovec, 2001). Other authors have further argued that electrodermal activity might be a better psychophysiological correlate of worrying than cardiac activity (e.g., Fowles, 1980).

A cognitive process that is related to worrying is rumination. Both rumination and worrying are repetitive thought styles that are closely correlated (Watkins, 2004). However, worrying has been primarily examined in relation to anxiety (e.g., Borkovec, Ray, & Stöber, 1998), whereas rumination has been most closely studied in the context of depression (Nolen-Hoeksema & Davis, 1999). Rumination refers to the tendency to focus on the causes and consequences of problems without moving into active problem solving (e.g., Nolen-Hoeksema, 2000). In contrast, worrying appears to be an attempt to prevent or minimize future problems and might act as a cognitive avoidance strategy to reduce negative emotions associated with intrusive catastrophic images (Borkovec et al., 1998). A number of empirical studies support this distinction (Fresco, Frankel, Mennin, Turk, & Heimberg, 2002; Segerstrom, Stanton, Alden, & Shortridge, 2003). Therefore, despite the similarities between worrying and rumination, we limit our inquiry to the psychophysiological correlates of worrying.

The specific purpose of the current study was to investigate the cortical and autonomic indicators of anxiety. In doing so, we conceptualize worrying as a state of anxious apprehension that is distinguishable from anxious arousal (Heller et al., 1997). We adopted a public speaking task and hypothesized that worrying about the speech is associated with greater left-frontal hemispheric activity, lower cardiac vagal tone, and greater electrodermal activity compared with baseline, relaxation, and fearful anticipation of imminent threat. The latter task was included to induce anxious arousal. On the basis of the findings reviewed previously, we predict that fearful anticipation of imminent threat is associated with the greatest level of subjective distress and heart rate, and greater right-hemispheric activity. In addition, we predicted that the effects of the experimental manipulations on these psychophysiological variables are moderated by trait public speaking anxiety.

## Method

### Participants

Forty undergraduate students from introductory psychology classes at Boston University, a large private university, participated in this study. One of the participants had missing questionnaire data. Two had missing data of all autonomic measures because of equipment failure. Therefore, autonomic measures were available from 38 participants. The majority of these

participants were Caucasian (82.1%); however, a proportion of participants identified themselves as African American (10.3%), Asian (2.6%), or other (5.2%). The mean age of participants was 19 years (range = 18–23;  $SD = 1.29$ ). All participants were right-handed as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971). Furthermore, to control for potential gender differences in emotional responses (Brody & Hall, 2000), all participants were male. Participants received 3 hr of course credit for the study. Because of technical problems, EEG data were not available for 13 of the 40 participants. Specifically, these individuals had to be excluded because the EEG was incorrectly referenced and yielded unusable data. Therefore, the EEG analyses are based on 27 individuals.<sup>1</sup>

### Procedure

On arrival at the laboratory, participants were briefed about the procedures, and written consent was obtained to a protocol approved by the Institutional Review Board of Boston University. The consent form mentioned that participants would be “asked to give an impromptu speech as part of this experiment.” No further information was provided about the nature of this task. After participants signed the consent form, they were asked to complete a number of self-report instruments.

Participants’ level of public speaking anxiety was assessed with the Personal Report of Confidence as a Speaker Scale (PRCS; Paul, 1966). The PRCS was first developed by Gilkinson (1942) and later shortened by Paul (1966) to assess degree of public speaking confidence. The shortened version by Paul (1966) consists of 30 yes–no items. The scale shows satisfactory internal consistency (Klorman, Weerts, Hastings, Melamed, & Lang, 1974) and validity (Lombardo, 1988) and is frequently used to assess public speaking anxiety (e.g., Hofmann & DiBartolo, 2001). PRCS scores in the current sample ranged from 7 to 30. The sample mean of the scale was 20.33 ( $SD = 5.92$ ;  $Mdn = 22$ ).<sup>2</sup> The internal consistency of the scale in the current sample was  $\alpha = .86$ . The scores were normally distributed and similar to scores in the normal population (e.g., McNeil, 2001; Phillips, Jones, Rieger, & Snell, 1997).

General anxiety and depression were measured with the 20-item State-Trait Anxiety Inventory (STAI–trait version; Spielberger, Gorsuch, & Lushene, 1970), the 16-item Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), and the 21-item Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). These measures are commonly used and psychometrically sound. Negative affect (NA) and positive affect (PA) were measured with the 20-item Positive and Negative Affect Schedule (PANAS–trait form; Watson, Clark, & Tellegen, 1988). The questionnaire is divided into two subscales measuring PA and NA. The PANAS is widely used in experimental studies and has good reliability and validity (Mackinnon et al., 1999; Watson et al., 1988). To measure level of social anxiety, participants were further asked to complete the 20-item Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998), which includes self-statements regarding cognitive, affective, and behavioral responses to situations requiring social interaction. Heimberg, Mueller, Holt, Hope, and Liebowitz (1992) reported that the SIAS had high internal consistency in a group of undergraduate students, social phobics, and a community sample. Furthermore, the scale showed good temporal stability as well as good discriminant and construct validity.

<sup>1</sup> The subsample of individuals with complete EEG data and autonomic measures was not significantly different from the sample with missing EEG data on any demographic variables or questionnaire data (all  $ps > .16$ ). Furthermore, all psychophysiological findings that were based on the larger data set were replicated in the smaller subsample.

<sup>2</sup> The median PRCS score was the same for participants with both complete EEG data and autonomic measures and the participants with only autonomic measures.

After completing these questionnaires, participants were taken to the laboratory, and electrodes were attached to measure various psychophysiological variables, discussed in further detail later. As part of the psychophysiological session, participants underwent an initial baseline phase followed by relaxation, worrying, and anticipation phases. After each of these phases, participants were asked to rate their level of distress on a scale ranging from 0 (*no distress*) to 100 (*very distressed*). During the initial baseline phase, participants sat quietly with their eyes closed for 1 min. This was followed by a relaxation phase. The relaxation phase included a 30-s initial resting period, a 50-s listening period, a 30-s relaxation imagery period, and a 30-s recovery period. During the relaxation imagery period, participants were asked to close their eyes and imagine the script that they had heard during the listening period. Only the 30-s relaxation imagery period was used for further analyses. The script was as follows:

You are sitting at the beach on a Caribbean island. You feel calm and relaxed. You are watching the beautiful sunset. The waves playfully slap the beach, foam, and withdraw. The air is filled with the smell of the ocean water. The sea breeze feels like satin on your skin. You feel at one with the universe. What a perfect moment. You savor the feeling. Please fully experience this scene now.

This phase was followed by worry induction in which participants listened to a number of ruminative self-statements. The self-statements of this script were adapted from the negative subscale of the Self-Statement During Public Speaking Scale (Hofmann & DiBartolo, 2000). Similar to the relaxation phase, the worry phase included a 30-s initial resting period, a 50-s listening period, a 30-s period of worrying (with eyes closed), and a 30-s recovery period. Only the 30-s worry period was used for the subsequent analyses. The script was as follows:

You have to give a presentation in front of a big class. You feel overwhelmed by negative thoughts as you are facing the audience. What you say will probably sound very stupid. People will realize how awkward and dumb you feel. You will probably bomb out anyway. A failure in this situation would be more proof of your incapacity. You feel like such a loser. Please fully experience this scene now.

Participants were then asked to give a 10-min impromptu speech after a 3-min anticipation period. These periods of increasing threat imminence were included to induce fearful anticipation (anxious arousal). The experimenter marked the first, second, and third minutes of this fearful anticipation period for later analyses. For the anticipation phase, participants were given the following instructions:

In a few minutes you will be asked to give an impromptu 10-min speech in front of a video camera. We will videotape your speech, and some members from our research staff will later evaluate the quality of your speech. The experimenter will give you a list of three topics, and you will be asked to give a 10-min speech about any or all of these topics. The experimenter will give you the list of speech topics and more detailed instructions in a few minutes. For now, please just sit quietly with your eyes closed.

### *Psychophysiological Data Acquisition*

All measures were recorded with equipment by James Long Company (Caroga Lake, NY) and with the data-acquisition program Snap-Master for Windows. The system allows for continuous collection of the recordings. The physiological measures were digitized at 512 samples per second with a 31-channel A/D converter operating at a resolution of 12 bits and having an input range of  $-2.5$  V to  $+2.5$  V. Autonomic indicators included heart rate, heart rate variability (respiratory sinus arrhythmia

[RSA]), and electrodermal activity (skin conductance level [SCL]). All psychophysiological channels were amplified by individual SA Instrumentation Bioamplifiers. The amplification rates and high-pass filter (HPF) and low-pass filter (LPF) settings were as follows: electrocardiogram (ECG; gain = 500, HPF = 0.1 Hz, LPF = 1000 Hz), respiration (gain = individually adjusted, HPF = none/DC, LPF = 10 Hz), and SCL (gain = 0.1 V/microsiemens, HPF = none/DC, LPF = 10 Hz). The filter settings for the EEG channels were set at 1 Hz (high pass) and 100 Hz (low pass).

During the collection of the data, the onset and termination of experimental phases were defined using an event marker, which was engaged manually by the experimenter at the appropriate times. Average values of the psychophysiological variables level were computed for each period of interest (i.e., 1-min baseline period, 30-s relaxation imagery period, 30-s worry period, and three 1-min anticipation periods). Participants sat quietly with their eyes closed during each of these periods.

### *Recording of ECG*

The plus and minus channels of the grounded ECG were recorded through electrodes attached to both sides of each participant's lowest ribs. Target skin areas were cleaned with alcohol wipes and allowed to dry. Heart rate data were analyzed using a computer program by James Long Company. R waves were automatically detected by the computer program and, subsequently, raw ECG and R-wave identification marks were viewed graphically by the experimenter. The R-wave file was manually corrected to remove R-wave identification marks that were incorrectly specified (e.g., a movement artifact that the computer coded as an R wave) or to score R waves that were missed by the automated detection. Heart rate was computed as number of R waves per minute.

### *Recording of RSA*

A software program provided by James Long Company combining cardiac and respiratory data was used to estimate RSA. RSA refers to the rhythmic variations in heart rate that occur at the frequency of respiration and reflects parasympathetic control over the heart (e.g., Berntson, Cacioppo, & Quigley, 1993). To measure breathing, a flexible respiration band was strapped around each participant's chest. The software program computes RSA using the peak-valley method (Grossman, 1983). This method derives RSA by calculating the difference between the minimum interbeat interval during inspiration and the maximum interbeat interval during expiration. RSA is reported in seconds.

### *Recording of Electrodermal Activity*

SCL was measured using two Ag-AgCl electrodes filled with electroconductive gel that were attached to the palmar surface of the middle phalanges of the third and fourth fingers of the nondominant (left) hand. Participants washed their hands with water before the electrodes were attached. SCL was averaged over 1-s intervals and are reported here in microsiemens.

### *Recording of EEG Activity*

EEGs were recorded with a Lycra stretchcap (Electro-Cap, Inc., Eaton, OH). Electrode placement was based on the International 10–20 Electrode Placement System (Jasper, 1985). The electrodes were placed on the left and right frontal (F3, F4), left and right parietal (P3, P4), midline central (Cz), and right mastoid (A2) sites, each of which was referred to an electrode placed at the left mastoid (A1) site. EEG activity that was recorded from the right mastoid site was used to compute an average mastoid reference (e.g., Miller, Lutzenberger, & Elbert, 1991). Horizontal

and vertical electro-oculogram channels for offline eye movement artifact correction of the EEG data were recorded by placing Beckman miniature Ag-AgCL electrodes at the outer canthus of each eye and above and below the left eye. Electrode impedance was below 5 kΩ, and the impedances for homologous sites were within 500 Ω of each other.

The EEG data were visually inspected for artifacts from eyeblinks and other motor movements, using software developed by James Long Company. This program removes data from all channels if artifacts are present on any one channel. The artifact-free EEG data were analyzed using a discrete Fourier transform, with a Hamming window of 1-s width and 50% overlap. The data were digitally filtered with a 501-weight filter for alpha-band frequencies (8–13 Hz). EEG alpha was quantified by computing a root mean square score for the filtered EEG. To normalize the distribution of the EEG data, alpha values were logarithmically transformed (using the natural log). In the current study, we focused on the alpha frequency band to be consistent with previous research studies in this area (e.g., Heller et al., 1997; Nitschke et al., 1999; see Coan & Allen, 2004, and Harmon-Jones, 2003, for recent reviews on alpha EEG asymmetry and emotion). In accordance with extensive prior literature, alpha activity (8–13 Hz) was used as an inverse proxy of activation (Davidson, 1998; Pfurtscheller, Stancak, & Neuper, 1996; Shagass, 1972).

*General Data Analytic Strategy*

Following the recommendations by Cohen, Cohen, West, and Aiken (2003), we analyzed the data using general linear model procedures for repeated measure design, which is an extension of the regression model, using SPSS for Windows, version 11.0.1. Significant trend analyses were followed up with paired *t* tests to directly compare the worry period with the other experimental conditions.

EEG asymmetry was analyzed in two ways. First, we examined hemisphere (left, right), caudality (anterior, posterior), and changes from baseline to the worry period as the within-subjects variables. The logarithmically transformed EEG alpha (natural log) was the dependent variable. A very similar data analytic strategy was conducted by Heller et al. (1995) and Nitschke et al. (1999). Second, we subtracted the natural log of left hemisphere alpha power from the natural log of right hemisphere alpha power, LN (right alpha, F4) – LN (left alpha, F3) to calculate an asymmetry index (which is a standard procedure; see Davidson et al., 2000, for review). Based on the assumption that alpha activity is inversely correlated with activation (Davidson, 1998; Pfurtscheller et al., 1996; Shagass, 1972), higher scores indicate relatively greater left frontal activity, whereas lower

scores indicate relatively greater right frontal activity. A score of zero represents symmetric activity.

Results

*Questionnaire Data*

Table 1 lists the means, standard deviations, and correlation matrix of the PRCS, BDI, PANAS-PA (trait form), PANAS-NA (trait form), PSWQ, SIAS, and STAI. The PRCS was negatively correlated with the PANAS-PA and positive correlated with the PSWQ, the PANAS-NA, and the SIAS. Similarly, the BDI was positively correlated with the PANAS-NA, PSWQ, SIAS, and the STAI and negatively correlated with the PANAS-PA. All other correlations were also in the expected direction.

*Distress Ratings*

Participants were asked to indicate their maximum level of distress after the 30-s baseline period, the 30-s relaxation period, the 30-s worry period, at the end of the 3-min anticipation period, and after the impromptu speech task, which had a maximum duration of 10 min. The within-subjects factor was statistically significant,  $F(4, 156) = 38.71, p = .000, \eta^2 = .50$ , and associated with a significant linear trend,  $F(1, 39) = 87.05, p = .000, \eta^2 = .69$ . As shown in Figure 1, the distress ratings were higher after the worry period than after baseline,  $t(39) = 5.43, p = .000$ , and relaxation,  $t(39) = 6.84, p = .000$ , but lower than after the speech,  $t(39) = 2.74, p = .009$  (other *ts* < 1.8, *ps* > .08). The analyses further revealed a significant main effect of the PRCS,  $F(1, 37) = 1.81, p = .008, \eta^2 = .17$ . Follow-up tests revealed that the PRCS scores were positively correlated with distress ratings after the anticipation period,  $r(39) = .56, p = .000$ , and the speech task,  $r(39) = .43, p = .000$  (all other correlations between the PRCS and the distress ratings showed *rs* < .19, *ps* > .2). The interaction effect between the PRCS and the experimental tasks did not reach the level of statistical significance,  $F(4, 148) = 3.24, p = .08, \eta^2 = .08$ . However, it should be noted that the statistical test only had sufficient power (.85) to detect a large effect ( $f^2 = .35$ ) at  $p <$

Table 1  
*Correlations, Means, and Standard Deviations of Self-Report Measures*

Variable	1	2	3	4	5	6	7
1. PRCS	—						
2. BDI	.09	—					
3. PANAS-PA	-.37	-.20	—				
4. PANAS-NA	.38*	.57*	0.003	—			
5. PSWQ	.36*	.46*	-0.17	0.63*	—		
6. SIAS	.48*	.51*	-0.23	0.57*	0.50*	—	
7. STAI	.37	.80*	-0.39*	0.66*	0.81*	0.67*	—
<i>M</i>	20.33	9.41	23.95	14.77	31.64	38.95	44.96
<i>SD</i>	5.92	8.82	6.01	8.59	14.68	13.69	11.78

*Note.* PRCS = Personal Report and Confidence as a Speaker; BDI = Beck Depression Inventory; PANAS-PA = Positive and Negative Affect Schedule, Positive Subscale (trait form); PANAS-NA = Positive and Negative Affect Schedule, Negative Subscale (trait form); PSWQ = Penn State Worry Questionnaire; SIAS = Social Interaction Anxiety Scale; STAI = State-Trait Anxiety Inventory (trait form).

\*  $p < .05$ .



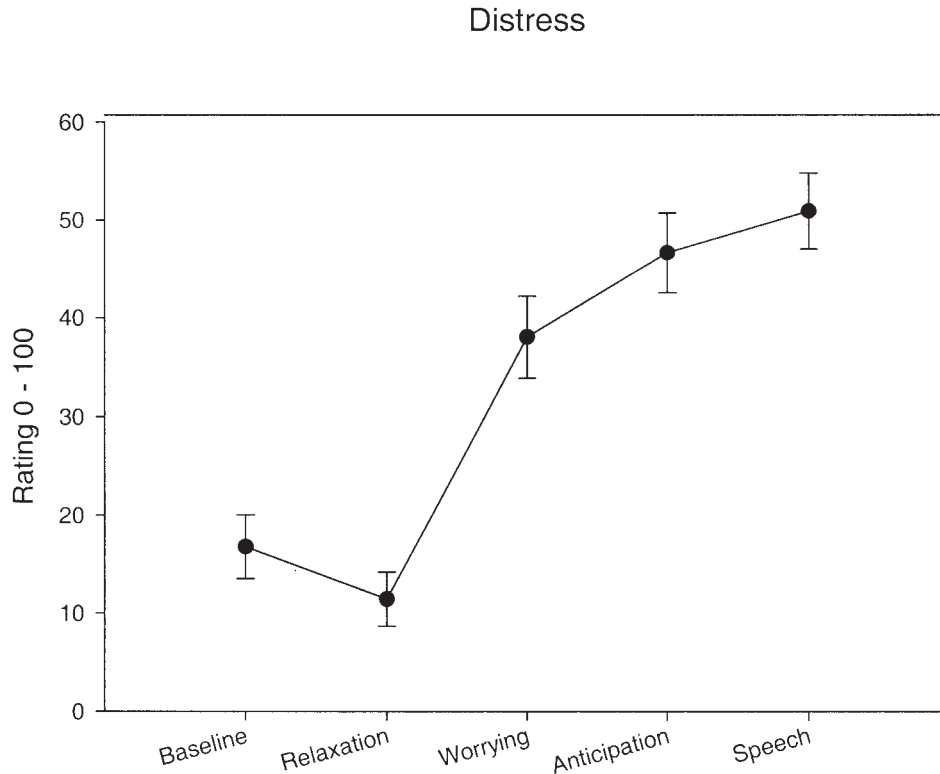


Figure 1. Distress ratings after completing the experimental tasks. Values represent means, and vertical lines represent standard errors of the mean.

.05. The power was insufficient (.46) to detect a medium-sized effect ( $f^2 = .15$ ) at  $p < .05$ .

#### Autonomic Indicators

Heart rate, SCL, and RSA were measured during baseline, relaxation, worrying, and anticipation after 1, 2, and 3 min. Using general linear modeling procedures, we examined changes in these indicators during the six experimental conditions.

**Heart rate.** The within-subjects factor was statistically significant,  $F(5, 185) = 26.65, p = .000, \eta^2 = .42$ , and associated with a significant linear,  $F(1, 37) = 47.60, p = .000, \eta^2 = .56$ , and quadratic trend,  $F(1, 37) = 29.41, p = .000, \eta^2 = .44$  (see Figure 2). Heart rate was higher during the worry period than during baseline,  $t(37) = 5.23, p = .000$ , relaxation,  $t(37) = 7.00, p = .000$ , and Minute 2 of the anticipation phase,  $t(37) = 3.13, p = .003$ . No difference was found between the worry period and the first and second minutes of the anticipation phase ( $ts < 1, ps > .1$ ). The PRCS between-subjects effect was not significant,  $F(1, 35) = 1.87, p = .18, \eta^2 = .05$ . Furthermore, the interaction effect between PRCS and experimental conditions was not significant,  $F(5, 175) = 0.21, p = .96, \eta^2 = .00$ . The correlation between the PRCS scores and heart rate during the worry period was  $r = .01, p = .95$ .

**RSA.** The within-subjects factor was statistically significant,  $F(5, 185) = 10.86, p = .000, \eta^2 = .23$ , and associated with significant linear,  $F(1, 37) = 9.48, p = .004, \eta^2 = .20$ , quadratic,

$F(1, 37) = 22.83, p = .000, \eta^2 = .38$ , and cubic,  $F(1, 37) = 16.13, p = .000, \eta^2 = .30$ , trends (see Figure 3). RSA was lower during the worry period than during baseline,  $t(38) = 4.68, p = .000$ , relaxation,  $t(37) = 2.19, p = .04$ , and the second,  $t(38) = 2.43, p = .02$ , and third,  $t(38) = 2.75, p = .01$ , minutes of the anticipation phase. RSA did not differ between the worry period and the first anticipation period ( $t < 1.7, p > 1$ ).<sup>3</sup> The analysis of the PRCS showed no significant between-subjects main effect,  $F(1, 35) = 0.07, p = .79, \eta^2 = .00$ , and no significant interaction effect between PRCS scores and experimental conditions,  $F(1, 175) = 0.74, p = .60, \eta^2 = .02$ . The correlation between the PRCS scores and RSA during the worry period was  $r = -.12, p = .49$ .

**SCL.** The within-subjects factor was statistically significant,  $F(5, 185) = 12.47, p = .000, \eta^2 = .25$ , and associated with a quadratic trend,  $F(1, 37) = 28.17, p = .000, \eta^2 = .43$

<sup>3</sup> Some authors recommend controlling for respiration rate and tidal volume when quantifying RSA (e.g., Berntson et al., 1997; Grossman & Kollai, 1993; Ritz, Thöns, & Dahme, 2001; but also see Porges & Byrne, 1992). We did not measure tidal volume but did record respiration rate. Respiration rate did not change significantly over the course of the six experimental conditions,  $F(5, 185) = .45, p = .81, \eta^2 = .01$ . Furthermore, the PRCS  $\times$  Task interaction effect,  $F(5, 180) = 1.06, p = .38, \eta^2 = .03$ , and the PRCS main effect,  $F(1, 36) = .29, p = .59, \eta^2 = .01$ , was not statistically significant. These findings suggest that changes in respiration rate are unlikely to account for changes in RSA.

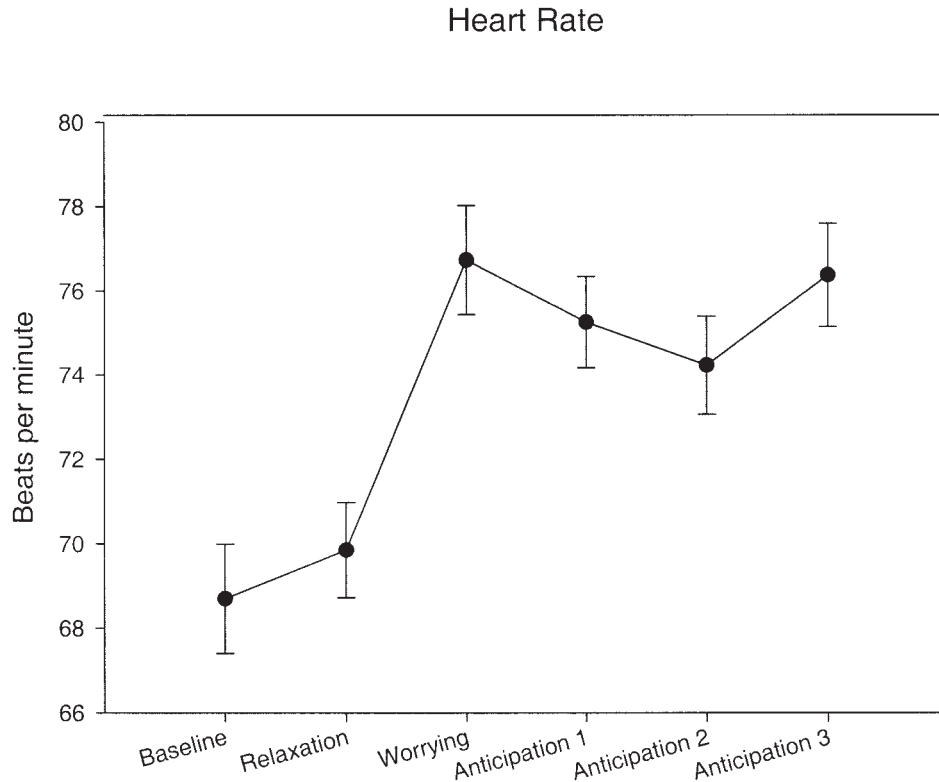


Figure 2. Heart rate during the experimental procedures. Anticipation 1, 2, and 3 denote the first, second, and third minutes, respectively, of the speech anticipation period. Values represent means, and vertical lines represent standard errors of the mean.

(see Figure 4). SCL was higher during the worry period than during baseline,  $t(37) = 3.49, p = .001$ , but lower than during the first minute,  $t(37) = 3.82, p = .001$ , and third minute,  $t(37) = 3.40, p = .002$ , of the anticipation phase. No difference was found between the worry period and the relaxation period ( $t < .08, p > .9$ ) or the second minute of the anticipation period ( $t < .05, p > .9$ ). The PRCS was not significant between subjects,  $F(5, 175) = 1.32, p = .26, \eta^2 = .04$ . Furthermore, the interaction effect between PRCS scores and experimental conditions was not significant,  $F(5, 175) = 0.11, p = .74, \eta^2 = .00$ . The correlation between the PRCS scores and SCL during the worry period was  $r = -.15, p = .36$ .

#### EEG Asymmetry

The results of a general linear modeling procedure with hemisphere (left, right), caudality (anterior, posterior), and task (baseline to worry) as the within-subjects variables revealed significant hemisphere,  $F(1, 26) = 24.69, p = .000, \eta^2 = .49$ , and caudality,  $F(1, 26) = 67.24, p = .000, \eta^2 = .72$ , effects. None of the other effects were significant, including the task effect,  $F(1, 26) = .37, p = .55, \eta^2 = .01$ , Task  $\times$  Caudality effect,  $F(1, 26) = 2.31, p = .14, \eta^2 = .08$ , Hemisphere  $\times$  Caudality effect,  $F(1, 26) = .26, p = .62, \eta^2 = .01$ , and Task  $\times$  Hemisphere  $\times$  Caudality effect,  $F(1, 26) = 1.09, p = .31, \eta^2 = .04$ . The PRCS only revealed a trend for a main between-

subjects effect,  $F(1, 24) = 3.67, p = .067, \eta^2 = .13$ . None of the interaction effects involving PRCS were statistically significant ( $F_s < 1.7, p > .20, \eta^2 < .07$ ). As shown in Figure 5, participants showed relatively greater left-hemispheric activity and relatively greater activity in the frontal region.

We further examined changes in F4/3 asymmetry index associated with the experimental phases (baseline, relaxation, worrying, and anticipation after 1, 2, and 3 min). The results revealed a significant task effect,  $F(4, 130) = 3.45, p = .01, \eta^2 = .12$ . As shown in Figure 6, the asymmetry index was greater during the worry phase than during the first,  $t(26) = 4.25, p = .000$ , and second,  $t(26) = 3.01, p = .006$ , minutes of the anticipation phase. The difference between the worry period and baseline approached statistical significance,  $t(26) = 1.84, p = .08$ . No other differences were found between the worry period and the other experimental conditions (all  $t_s < .11, p_s > .2$ ). Furthermore, the interaction effect between the PRCS and the experimental conditions was not significant,  $F(5, 120) = 1.12, p = .35, \eta^2 = .05$ . The results only showed a trend for a between-subjects main effect of the PRCS,  $F(1, 24) = 2.17, p = .15, \eta^2 = .08$ .

To specifically examine the relationship between the level of social anxiety and frontal asymmetry during worrying, we correlated the PRCS scores and the F4/3 asymmetry index during the worry period. This association was significant ( $r = .47, p = .02$ ) and is depicted in Figure 7.

## Respiratory Sinus Arrhythmia

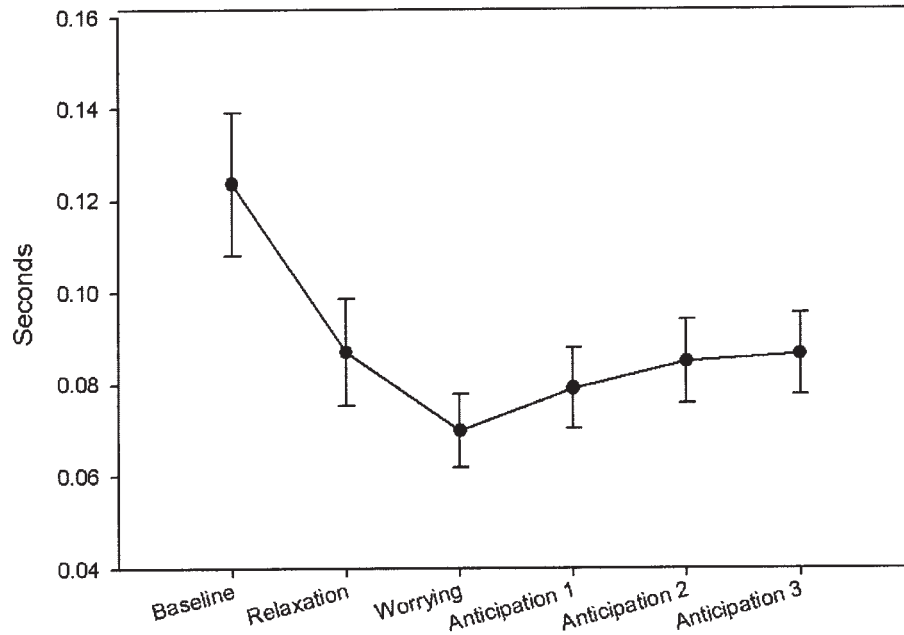


Figure 3. Respiratory sinus arrhythmia during the experimental procedures. Values represent means, and vertical lines represent standard errors of the mean.

Finally, we examined changes in the P4/3 asymmetry index during the experiment. The results only revealed a trend for the task effect,  $F(5, 130) = 2.18, p = .09, \eta^2 = .08$ . No PRCS main or interaction effects were found (all  $F_s < .9, p_s > .4, \eta^2 < .20$ ). The correlation between the PRCS scores and the P4/3 asymmetry index during the worry period was not significant ( $r = .19, p > .3$ ). Table 2 presents a summary of the results.

### Discussion

To our knowledge, this study is the first to simultaneously measure EEG asymmetry and autonomic measures during experimental induction of worrying. The experimental procedure to induce worrying was similar to the methodology used by Borkovec and Hu (1990); Lyonfields et al. (1995), and others. We measured frontal EEG asymmetry, heart rate, RSA, and SCL during a relaxation phase, worrying, and varying degrees of threat imminence, which began with a worry induction phase followed by three 1-min episodes of fearful anticipation of an upcoming speech task, an experimental procedure similar to that of Heller et al. (1997) and Davidson et al. (2000). Based on prior psychophysiological and EEG findings, we hypothesized that worrying, compared with relaxation or fearful anticipation, would be associated with less cardiac vagal tone, greater electrodermal activity, and frontal asymmetry in favor of the left hemisphere. We further predicted that fearful anticipation of imminent threat is associated with the greatest level of subjective distress and heart rate and greater right-hemispheric activity. We expected these effects to be greater

among public speaking anxious than among nonanxious individuals.

A summary of our findings is shown in Table 2. Consistent with our initial prediction, worrying was associated with relatively greater left-frontal activation compared with the speech anticipation period (anxious arousal). We also found a statistical trend for greater left-frontal activation during worrying than during baseline. These results are in line with the notion that worrying involves a predominance of negatively valenced verbal thoughts (Borkovec & Inz, 1990; Heller et al., 1997; Nitschke et al., 2000). Public speaking anxiety, which was measured with the PRCS, further significantly moderated this relationship. The PRCS scores and the F4/3 asymmetry index during the worry period showed a correlation of  $r = .47 (p = .02)$ . It should be noted that the internal consistency of the PRCS in the current sample was satisfactory. The mean score of the PRCS in the current sample (mean = 20.33;  $SD = 5.92$ ) was very similar to the mean score of a clinical sample reported by Paul (1966), who found a mean score of 20.6 ( $SD = 3.31$ ). In comparison, the mean PRCS score in a normative sample of 1,109 college students (Phillips et al., 1997) was 14.24 ( $SD = 7.76$ ). Therefore, the PRCS mean score of the current sample was slightly higher but still within 1  $SD$  of a normative sample of college students.

Relatively increased left-frontal activity during worrying is in line with prior findings in generalized anxiety disorder (GAD), in which worry and verbal rumination are cardinal symptoms. In an early positron emission tomography study of GAD patients, Wu et

Skin Conductance Level

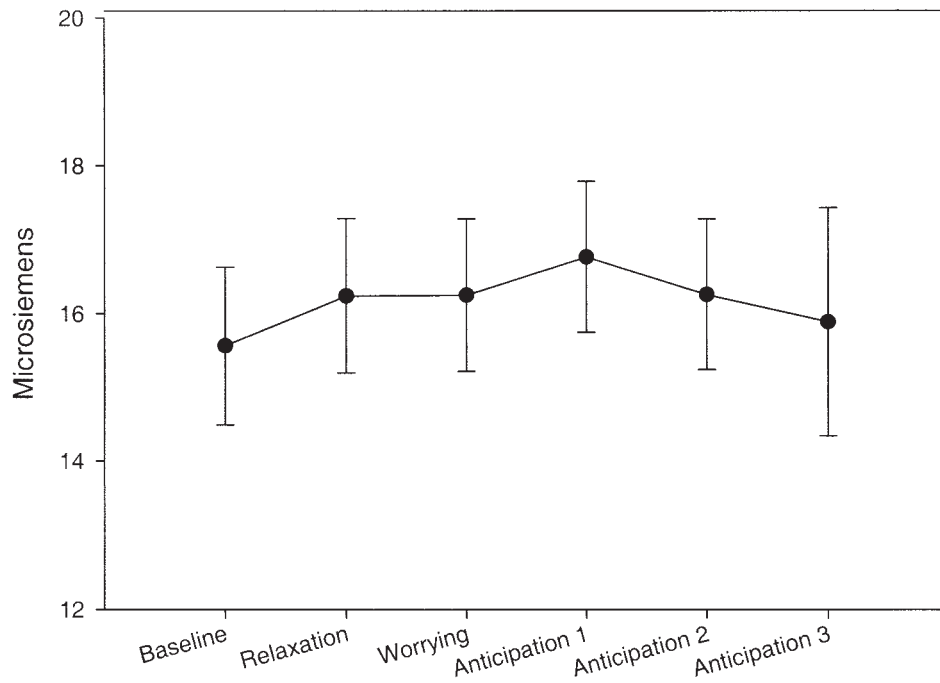


Figure 4. Skin conductance level during the experimental procedures. Values represent means, and vertical lines represent standard errors of the mean.

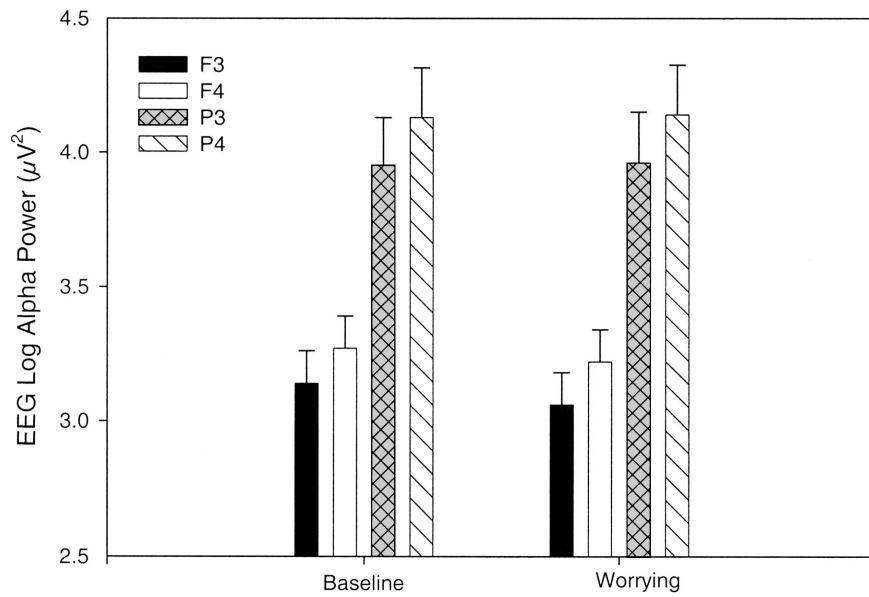


Figure 5. Means and standard errors of logarithmically transformed (natural log) electroencephalographic (EEG) alpha power density of the left and right frontal (F3 and F4) and left and right parietal (P3 and P4) regions during baseline and worrying. Lower alpha power density indicates greater activity. Vertical lines represent standard errors of the mean



## Frontal Asymmetry

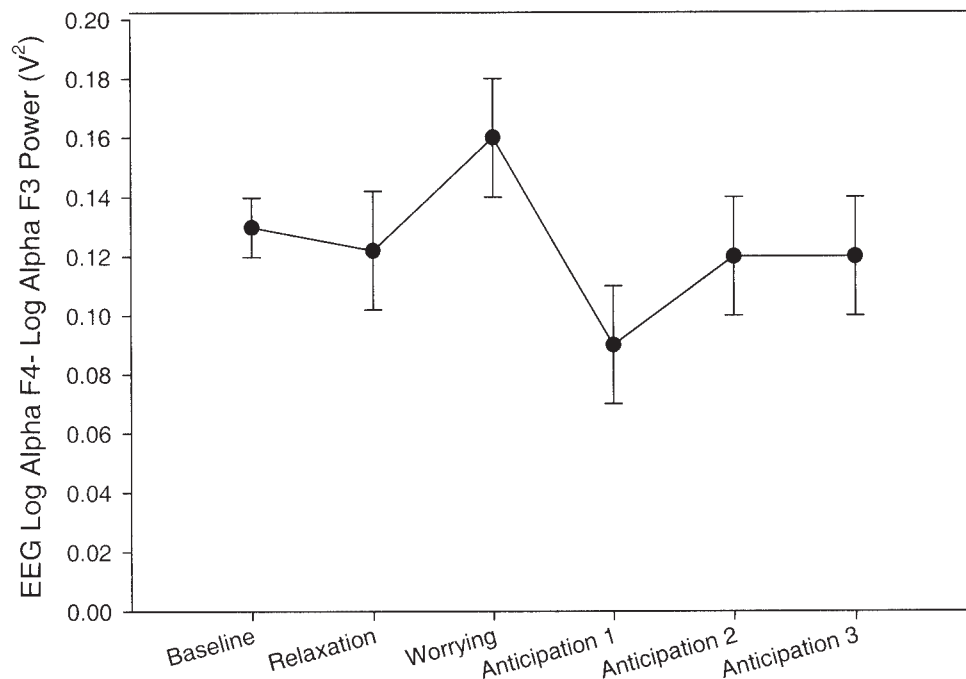


Figure 6. Frontal asymmetry index (natural log of right frontal, F3, minus natural log of left frontal, F4, region) during the experimental procedures. Values represent means, and vertical lines represent standard errors of the mean. EEG = electroencephalographic.

al. (1991) reported increased glucose metabolism in left inferior frontal regions in close proximity to Broca's language areas. In a behavioral study (Otto, McNally, Pollack, Chen, & Rosenbaum, 1994), auditory perceptual asymmetry in favor of the left hemisphere was associated with increased memory for threat-related words in a GAD sample. Finally, an EEG study found that clinical improvement after benzodiazepine treatment was accompanied by increased left-frontal alpha power (i.e., decreased activity; Buchsbaum et al., 1985). Collectively, the current as well as prior findings in GAD samples converge in highlighting a prominent role of left-frontal involvement in worrying.

As expected, the distress ratings were higher after the worry period than after baseline and relaxation but lower than after the speech. PRCS scores were positively correlated with distress ratings after the anticipation period and the speech task. Consistent with previous research (Borkovec & Hu, 1990; Hoehn-Saric & McLeod, 2000; Lyonfields et al., 1995; Thayer et al., 1996), worrying was further associated with decreased heart rate variability (as assessed by RSA), which appears to be the result of decreased cardiac vagal tone (e.g., Berntson et al., 1993). This is consistent with the notion that worrying is associated with decreased autonomic flexibility. In addition, worrying appears to be associated with greater sympathetic activation as indicated by the relative increase in SCL compared with baseline and the fearful anticipation periods. However, we found no difference between

heart rate response to worrying and fearful anticipation. This result is consistent with earlier clinical studies on the effects of worrying on autonomic measures (e.g., Thayer et al., 1996).

Studies investigating cognitive processes often distinguish between verbal thoughts and images. These two cognitive phenomena seem to have very different effects on the psychophysiological response to emotional material. For example, verbalizing a fearful situation typically induces less cardiovascular response than visually imagining the same situation (Vrana, Cuthbert, & Lang, 1986), possibly because verbalizations are used as a strategy for abstraction and disengagement to decrease sympathetic arousal to aversive material (Tucker & Newman, 1981). This suggests that the verbal activity during worrying is less closely connected to the affective, physiological, and behavioral systems than images are and might, therefore, be a poor vehicle for processing emotional information (Borkovec et al., 1998).

In sum, our findings suggest that worrying is not merely a state of fearful anticipation that differs from fearful arousal in its degree of threat imminence. In fact, worrying had very different psychophysiological correlates than the anticipatory phases just before the speech, consistent with growing evidence that different types of anxiety are associated with distinct physiological markers (Lang et al., 2000; Nitschke & Heller, 2002; Nitschke et al., 2000). Accordingly, our findings suggest that worrying is a unique emotional state that is associated with frontal brain asymmetry in favor of the

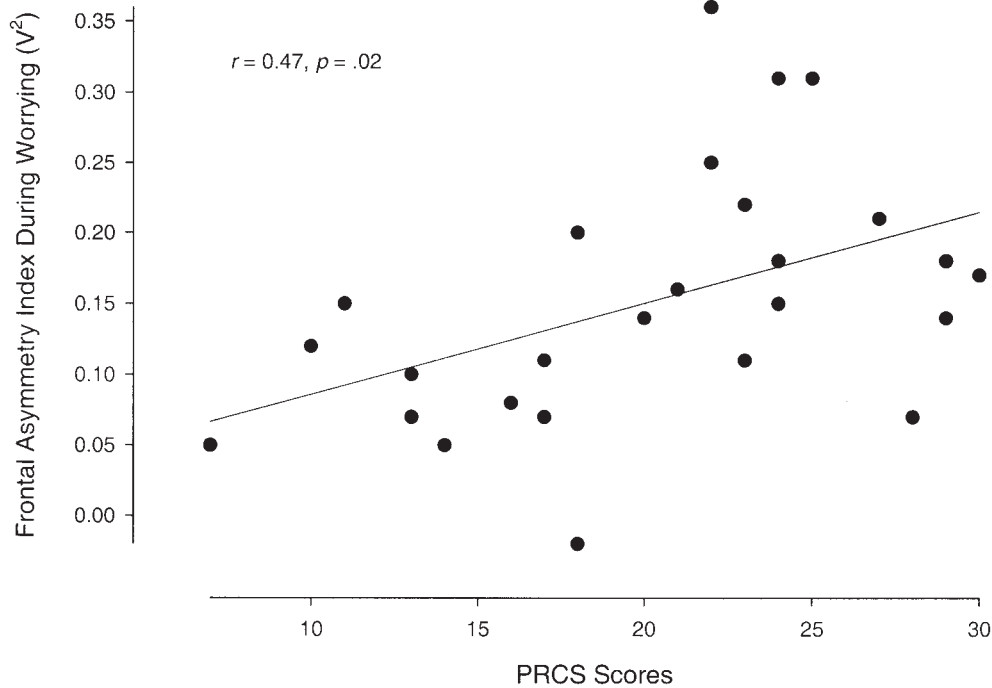


Figure 7. Correlation between frontal asymmetry index and Personal Report of Confidence as a Speaker Scale (PRCS) scores during the worrying period.

left hemisphere, less cardiac vagal tone, and greater electrodermal activity compared with fearful anticipation.

A number of limitations should be mentioned. First, because of the nature of the experimental tasks, we did not vary the order because threat imminence was a defining and distinguishing feature of the experimental tasks. Therefore, it is impossible to rule out any order effects. For example, it is possible that participants already had a heightened level of anxiety during baseline and relaxation because they were informed earlier about the speech when they signed the consent form. Second, the task intervals lasted 30 s. Although this length is typical in psychophysiological experiments on worry, it is possible that a longer time interval

might have led to more pronounced effects of the experimental worry manipulation. However, this might have also resulted in greater carryover effects. Third, the participant sample on which the EEG analyses were based was relatively small ( $n = 27$ ). However, the subsample appeared to be a random subgroup of the total sample ( $n = 40$ ), and the EEG analyses revealed significant effects despite the lower statistical power. Fourth, to minimize within-group variance, we limited our sample to only male participants. We are unaware of any studies that have specifically examined gender differences in worry experiments, but we have no reason to believe that the effects reported in this study are gender specific. Nevertheless, future studies are required to assess whether the current findings will extend to female participants. Finally, the participants of our study were undergraduate students. Although this is not an unusual sample choice and is consistent with other reports in this area (e.g., Borkovec & Hu, 1990; Heller et al., 1997), it is difficult to directly compare the result with other reports that used clinical samples (e.g., Davidson et al., 2000). Therefore, we recommend that future studies examine the psychophysiological differences between worrying and fearful anticipation in clinical samples, especially in individuals with GAD and social phobia. Furthermore, it will be important to examine whether the psychophysiological correlates of worrying are trait markers or whether they change as psychopathology improves.

Table 2  
Summary of Comparison Between Experimental Phases in Autonomic Measures and Frontal Activation

Measure	Baseline	Relaxation	Anticipation phase		
			AP1	AP2	AP3
HR	W > B	W > R	ns	W > AP2	ns
RSA	W < B	W < R	ns	W < AP2	W < AP3
SCL	W > B	ns	W < AP1	ns	W < AP3
FAI	(W > B)	ns	W > AP1	W > AP2	ns

Note. Results of the comparison between worrying (W) and baseline (B), relaxation (R), and anticipation after 1 min (AP1), 2 min (AP2), and 3 min (AP3) in heart rate (HR), respiratory sinus arrhythmia (RSA), skin conductance level (SCL), and frontal asymmetry index (FAI); W > B, measure is greater during worrying than during baseline at  $p < .1$ ; all other comparisons,  $p < .05$ .

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