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BRIEF REPORT

Autonomic recovery and habituation in social anxiety

IRIS B. MAUSS,[a] FRANK H. WILHELM,[b,c] AND JAMES J. GROSS[a]

[a]Department of Psychology, Stanford University, Stanford, California, USA
[b]Department of Psychiatry and Behavioral Sciences, Stanford University, Stanford, California, USA
[c]Palo Alto Health Care System, Palo Alto, California, USA

Abstract

Growing evidence suggests that, contrary to expectation, high trait socially anxious (HTSA) and low trait socially anxious (LTSA) individuals show comparable autonomic reactivity during stressful speech tasks. To test the hypothesis that autonomic differences between groups might emerge during recovery or habituation, 35 HTSA and LTSA participants gave two impromptu speeches. Measures of anxiety experience as well as cardiovascular, electrodermal, respiratory, and vagal activation were obtained. Despite greater reports of anxiety experience in the HTSA versus the LTSA participants, autonomic measures showed comparable reactivity, habituation, and recovery in the two anxiety groups. These results suggest minimal autonomic differences between HTSA and LTSA individuals, thus supporting theories of social anxiety that emphasize cognitive factors.

Descriptors: Social anxiety, Speech stressor, Autonomic reactivity, Habituation, Recovery

Psychophysiological theory—as well as the interoceptive experience of anxious individuals—leads us to expect that social anxiety should be accompanied by robust autonomic responses that give rise to the symptoms reported by socially anxious individuals, such as a racing heart, blushing, or sweaty palms (e.g., Amies, Gelder, & Shaw, 1983; Hazen & Stein, 1995; Schlenker & Leary, 1982). Surprisingly, however, there is growing evidence that socially anxious individuals differ little if at all in physiological responding from non-socially anxious individuals during stressful tasks (e.g., Edelmann & Baker, 2002; Grossman, Wilhelm, Kawachi, & Sparrow, 2001; Mauss, Wilhelm, & Gross, in press; Mulkens, de Jong, Dobbelaar, & Bögels, 1999).

How might this lack of difference in autonomic responding between individuals with high (HTSA) and low (LTSA) trait social anxiety be explained? One possibility is that the magnitude of autonomic reactivity to a single laboratory stressor may not capture more subtle group differences within the full time frame of an anxiety episode. For example, differences might emerge only during repeated stressors, or during recovery and they would have crucial implications for clinical assessment and psychophysiology of social anxiety. This notion dovetails with Davidson's (1998) assertion that individual differences in emotional reactivity could be characterized by the time course of affective responses rather than by response magnitude alone. Consistent with this explanation, Beidel, Turner, and Dancu (1985) found increasing group differences between HTSA and LTSA in systolic blood pressure across three different tasks (same-sex interaction, opposite-sex interaction, impromptu speech). In a similar study, Eckman and Shean (1997) found that although there was no separation between LTSA and HTSA participants in heart rate during an initial impromptu speech, group differences emerged during a second and third speech. Studies on other anxiety disorders such as general anxiety or panic disorder also suggest that there is reason to expect group differences in habituation or during recovery (e.g., Chattopadhyay, Cooke, Toone, & Lader, 1980; Wilhelm, Gerlach, & Roth, 2001; Roth, Wilhelm, & Trabert, 1998).

Studies such as these provide initial support for the notion that HTSA individuals' autonomic responses differ from LTSA individuals' responses principally in terms of their temporal characteristics rather than their magnitude in response to a single stressor. However, prior studies have had important limitations, including: (a) assessment of a relatively small number of autonomic responses, (b) inconsistent findings across different measures of autonomic responding, and (c) use of different types of social stressors in repeated-task designs. These limitations leave some uncertainty as to how social anxiety affects autonomic habituation to and recovery from social stressors.

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Address reprint requests to: James Gross, Department of Psychology, Stanford University, Stanford, CA 94305-2130, USA. E-mail: james@psych.stanford.edu.

To address this issue, the present study assessed anxiety experience and autonomic responses in HTSA and LTSA individuals before, during, and after two highly stressful impromptu speeches. An impromptu speech paradigm was selected because it has been shown to be a reliable and valid method of inducing high levels of anxiety (e.g., Beidel, Turner, Jacob, & Cooley, 1989). Only participants with extreme scores on a measure of trait social anxiety were enrolled. To ensure the relevance of our anxiety induction procedures, we further screened individuals using a specific scale designed to measure anxiety concerning public speaking. To reduce between-group variability that could obscure relations among response components, we enrolled only female participants. To maximize the chances of detecting group differences in physiological responding, we measured a large number of physiological responses across different response systems. Compared to LTSA individuals, we expected HTSA individuals would show *less habituation* (defined as less decline in physiological reactivity from the first to the second speech) and *less recovery* (defined as higher physiological activation during recovery from the speeches).

METHOD

Participants

Participants were drawn from a larger study of social anxiety (Mauss et al., in press). In the larger study, 96 participants were asked to give a single speech. The aim of this study was to test whether high versus low trait social anxiety individuals would differ in reactivity to a single speech. In the present study, we focused on a subset of 36 participants who gave a second speech. The aim of the present study was to test whether group differences in habituation or recovery would emerge over the course of two speeches. Participants were selected to be low versus high anxiety based on their scores on the Social Phobia and Anxiety Inventory (SPAI), a questionnaire validated for identifying individuals with a trait of social phobia or social anxiety (Beidel, Turner, Stanley, & Dancu, 1989). Only participants with extreme scores (from the top 25% and the bottom 25% of the distribution of 840 female undergraduates) were selected. In addition, participants had to be above (for the HTSA group) or below (for the LTSA group) the mean of a subscale comprised of the six SPAI items relating to fear of speaking in front of others (items 5, 6, and 22a–d). One participant was excluded because of technical problems. This left 35 participants: 18 HTSA (average age = 19.3 years, $SD = 1.2$) and 17 LTSA (average age = 19.0 years, $SD = 1.2$). Mean SPAI scores were 104.3 for HTSA ($SD = 23.2$) and 30.4 ($SD = 17.4$) for LTSA, $t\ (34) = 10.5$, $p < .001$.

Procedure

Participants underwent testing in five stages within a single experimental session: baseline (3 min), speech 1 (3 min), recovery 1 (2 min), speech 2 (3 min), and recovery 2 (2 min). The session began with a neutral videotape of seascapes (baseline). At the end of the baseline, participants filled out a set of emotion ratings and underwent several short procedures not relevant to this report. The experimenter then informed the participant that for the next 2 min she would be presented with short bursts of white noise through a headset in order to get accustomed to the tones she would be hearing during later parts of the study. These white noise bursts were designed to increase task apprehension and task anxiety, and were presented every 15 s during the preparation and the speech. After this, participants rested for a minute.

Participants were informed that they would be asked to give a speech during which they would be watched closely and videotaped. To increase social anxiety, the experimenter told participants that the videotape would be shown to a panel of expert judges, who would rate the quality of their performance. The experimenter then entered the subject room to ostentatiously position the video camera directly in front of the participant. Next, the participants were informed that the topic of the speech was "Is it wrong for the government to execute people?" Participants were given 3 min to prepare their speech. After the speech, they were given 2 min to recover, during which time they were instructed to just sit quietly. After this, they were given 1 min to watch the seascapes film again. They were then asked to give another speech, this time on the topic "Is it right to assist people who want to commit suicide?," followed by the second recovery period.

Measures

Anxiety experience. Participants rated their anxiety experience on an 11-point Likert scale, ranging from 0 (*none at all*) to 10 (*extremely*) for baseline, speech 1, recovery 1, speech 2, and recovery 2.

Physiological measures. During the session, physiological channels were sampled at 400 Hz. Later, customized analysis software (Wilhelm, Grossman, & Roth, 1999) was applied to physiological data reduction, artifact control, and computation of average physiological scores for each participant for the baseline, speech, and recovery periods.

Measures were chosen to sample response systems most relevant to anxiety, including the cardiovascular, electrodermal, and respiratory systems. *Heart rate* was calculated from R-R intervals in the electrocardiogram. *Blood pressure* was obtained from the third finger of the nondominant hand by means of the Finapres™ 2300 (Ohmeda, Madison, WI) system. *Finger pulse amplitude* (FPA) was measured with a plethysmograph transducer (Model 1020 photoplethysmograph; UFI, Morro Bay, CA) attached to the tip of the participant's second finger. *Facial blush* was measured with the same type of plethysmograph transducer, attached with surgical tape to the participant's left cheek right below the cheekbone. *Skin conductance level* (SCL) was indexed by the mean level derived from a signal using a constant-voltage device to pass 0.5 V between Beckman electrodes (using an electrolyte of sodium chloride in Unibase) attached to the palmar surface of the middle phalanges of the first and second fingers of the nondominant hand. *Skin conductance fluctuations* (SCF) were detected as changes in SCL from a zero-slope baseline exceeding 0.2 μS. Fluctuations likely to stem from electrode contact artifacts were identified and excluded. *Respiratory rate* (RR) was measured using an inductive plethysmography device (Respitrace Corporation, Ardsley, NY) connected to bands containing coils of insulated wires placed around the abdomen and chest. Signals were calibrated for each individual to milliliter lung volume change using a fixed volume bag. Respiratory rate was calculated breath by breath using customized programs.

We also assessed vagal activation, which is especially relevant to recovery (e.g., Rottenberg, Wilhelm, Gross, & Gotlib, 2003), by measuring *respiratory sinus arrhythmia* (RSA). To index RSA, the R-R interval series from the ECG was converted into a

time series of instantaneous R-R intervals with a resolution of 4 Hz. Vagal control of heart rate was estimated as the magnitude of the transfer function relating RR interval oscillations to lung volume oscillations (resampled to 4 Hz) at the peak respiratory frequency (Saul et al., 1991), thus adjusting RSA for tidal volume changes. The peak respiratory frequency was automatically detected as the greatest local maximum in the lung volume power spectral density. Spectral coherence at this frequency was required to be at least 0.5, which led to the exclusion of 1 participant's RSA data for speech 2. All of the epochs met inclusion criteria for peak respiratory frequency being above 0.15 Hz.

Data Analysis

For the analyses of self-reported anxiety and physiological activation, we used baseline, speech 1, recovery 1, speech 2, and recovery 2 scores.

To test whether the speech task was effective in inducing experiential anxiety, we compared anxiety experience reports during speech 1 to the baseline employing a 2×2 ANOVA with speech versus baseline as a repeated measure (Task) and trait anxiety group as a between-participants factor (Anxiety Group). To test whether the speech task was effective in inducing physiological activation, we conducted a 2×2 MANOVA using all physiological measures with speech versus baseline as a repeated measure (Task) and trait anxiety group as a between-participants factor (Anxiety Group). Univariate tests were used to follow up on a significant multivariate effect of Task. Effects of Task indicate that the speech had a significant effect on anxiety experience or physiological reactivity.

To test whether there was differential reactivity or habituation between the two anxiety groups, we used a 2×2 MANCOVA with the speeches as a repeated measure (Task) and trait anxiety group as a between-participants factor (Anxiety Group). Baseline activation was used as a covariate to control for individual variation of resting activation.[1] Additionally, respiratory rate during the speeches was used as a changing covariate for analyses of RSA. Because measures of peripheral physiological activation are not independent, we simultaneously entered all physiological measures in the MANCOVA. Univariate tests were used for self-reported anxiety and to follow up on significant multivariate tests. Main effects of Anxiety Group indicate differential activity; main effects of Task indicate habituation; interactions between Task and Anxiety Group indicate differential habituation.

To test whether there was differential recovery between the two anxiety groups, we performed the same set of analyses on dependent measures during recovery from speeches 1 and 2, using a 2×2 MANCOVA with the recovery periods as a repeated measure (Task) and trait anxiety group (Anxiety Group) as a between-participants factor. To control for individual variation of resting and speech activation, the baseline was used as a fixed covariate and the speeches were used as a changing covariate. Additionally, respiratory rate during the recovery periods was used as a changing covariate for analyses of RSA. Main effects of Anxiety Group indicate differential recovery.

[1]Groupwise *t* tests confirmed that during the baseline, the two anxiety groups reported equal levels of anxiety and exhibited equal levels of physiological activation. However, we used the baseline as a covariate to control for individual differences in physiological activation.

Results

Reactivity to the Speech

Raw scores of anxiety experience and physiological responding for baseline, speech 1, recovery 1, speech 2, and recovery 2 are summarized in Figures 1 and 2. Main effects of Task indicated that participants showed significant increases in self-reported anxiety, $F(1,33) = 170.78$, $p < .001$, $\eta^2 = .84$, from the baseline to speech 1. Univariate tests to follow up on a significant effect of Task for the physiological measures, $F(8,24) = 123.00$, $p < .001$, $\eta^2 = .98$, revealed significant increases in heart rate, $F(1,33) = 766.79$, $p < .001$, $\eta^2 = .96$, blood pressure, $F(1,33) = 91.80$, $p < .001$, $\eta^2 = .75$, finger pulse amplitude, $F(1,33) = 35.08$, $p < .001$, $\eta^2 = .53$, skin conductance level, $F(1,33) = 77.90$, $p < .001$, $\eta^2 = .70$, skin conductance fluctuation rate, $F(1,33) = 73.40$, $p < .001$, $\eta^2 = .70$, and significant decreases in RSA, $F(1,31) = 5.56$, $p = .025$, $\eta^2 = .15$, from the baseline to speech 1. There was no significant effect of Task for repiratory rate and blushing. Adjustment for familywise errors with the Bonferroni method (at a critical alpha level of .05) did not change the significance of these results.

Habituation and Differential Habituation

Main effects of Task indicated that participants showed significant decreases in self-reported anxiety, $F(1,33) = 11.68$, $p = .002$, $\eta^2 = .26$, from speech 1 to speech 2. Univariate tests to follow up on a significant effect of Task for the physiological measures, $F(8,21) = 8.19$, $p < .001$, $\eta^2 = .76$, revealed significant decreases in heart rate, $F(1,31) = 62.49$, $p < .001$, $\eta^2 = .69$, skin conductance level, $F(1,31) = 4.62$, $p < .040$, $\eta^2 = .14$, and skin conductance fluctuation rate, $F(1,31) = 21.67$, $p < .001$, $\eta^2 = .44$, and significant increases in RSA, $F(1,31) = 4.95$, $p = .034$, $\eta^2 = 15$, from speech 1 to speech 2. After adjusting for familywise errors with the Bonferroni method (at a critical alpha level of .05), the Task effects on SCL and RSA are no longer significant.

Effects of Anxiety Group indicated that HTSA participants reported greater anxiety during speeches 1 and 2 than LTSA, $F(1,33) = 20.06$, $p = .001$, $\eta^2 = .38$. There were no main effects of Anxiety Group for measures of physiological reactivity, and none of the Task $\times$ Anxiety Group interactions reached significance. Thus, there was no difference between anxiety groups in physiological responding to the speeches, and there was no differential habituation to the repeated speech tasks.

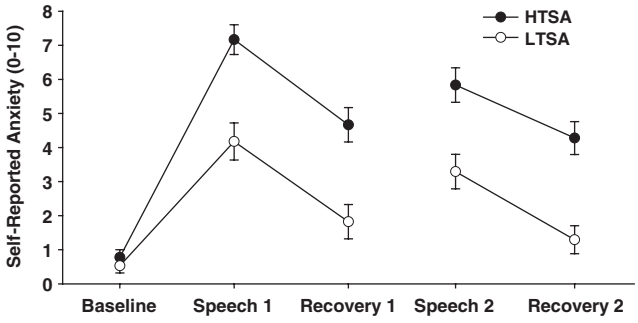

Figure 1. Self-reported anxiety for high trait socially anxious (HTSA) versus low-trait socially anxious (LTSA) participants for baseline, speech 1, recovery 1, speech 2, and recovery 2. Error bars represent standard errors of the mean.

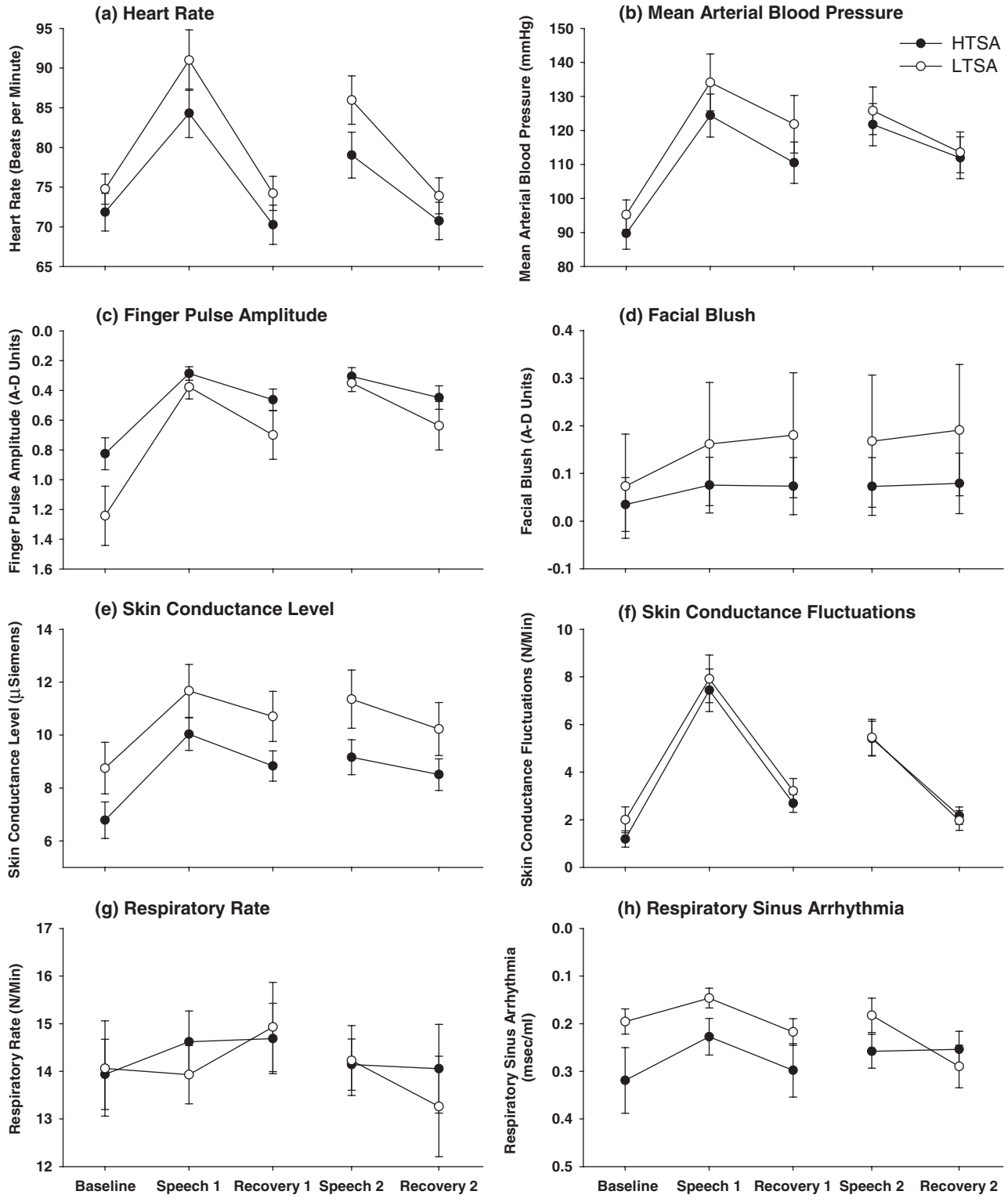

Figure 2. Heart rate, mean arterial blood pressure, finger pulse amplitude, facial blush, skin conductance level, skin conductance fluctuation rate, respiratory rate, and respiratory sinus arrhythmia for high trait socially anxious (HTSA) versus low-trait socially anxious (LTSA) participants for baseline, speech 1, recovery 1, speech 2, and recovery 2. Error bars represent standard errors of the mean.

Recovery and Differential Recovery

There were no significant main effects of Task, indicating that when controlling for speech activation, recovery after speech 2 was equal to recovery after speech 1 for anxiety experience and physiological activation. Effects of Anxiety Group indicated that LTSA exhibited more normalization (return to baseline) in anxiety experience, $F(1,33) = 7.59$, $p = .009$, $\eta^2 = .19$, than HTSA participants. There were no effects of Anxiety Group

for the physiological measures, and there were no significant Task × Anxiety Group interactions.[2] Thus, there was no differential recovery from the repeated speech tasks in the two anxiety groups.

Discussion

Contrary to expectations derived from psychophysiological theory and the interoception of anxious individuals (e.g., Amies et al., 1983; Hazen & Stein, 1995; Schlenker & Leary, 1982), there is growing evidence that socially anxious individuals differ only minimally or not at all in autonomic responding from non-socially anxious individuals during stressful tasks (e.g., Edelmann & Baker, 2002; Grossman et al., 2001; Mauss et al., in press; Mulkens et al., 1999). The present study tested the hypothesis that more subtle temporal features of participants' autonomic responses (recovery and habituation) might reveal important differences between HTSA and LTSA participants. Results indicate that HTSA participants reported greater anxiety during both speeches and recovery periods than did LTSA participants. However, there were no group differences in autonomic physiological reactivity, habituation to, or recovery from the speeches.

These findings suggest that differences in physiological responding between low and high trait social anxiety groups are either nonexistent or very small. This is consistent with models of social anxiety that emphasize the role of cognitive processes such as attentional focus, dysfunctional appraisal of the self and social situations, and negative self-schemata in social anxiety (e.g., Clark & McManus, 2002; Hope, Gansler, & Heimberg, 1989; Leary & Kowalski, 1995). These theories attribute group differences in *self-reported* physiological activation to misperception and overreporting by HTSA rather than to actual group differences in physiological activation (e.g., Edelmann & Baker, 2002; Mulkens et al., 1999; Sarason, 1985). Together, these findings lend support to the notion that cognitive mechanisms might be more important than autonomic physiological activation for explaining individual differences in social anxiety.

Recent brain imaging and startle reactivity studies provide additional support for this interpretation. Malizia, Wilson, Bell, Nutt, and Grasby (2000) reported increased activation of dorsolateral prefrontal cortex areas when social phobics thought about anxiety-provoking situations. Similarly, Davidson, Marshall, Tomarken, and Henriques (2000) showed heightened activation in the right prefrontal cortex associated with anticipating a speech in social phobics relative to controls, which can be interpreted as indicating differences in cognitive processing (e.g., Clark & McManus, 2002; Hofmann, 2000). Conversely, research on startle reactivity indicates that startle is *not amplified*—and perhaps even reduced—in social anxiety (e.g., Blumenthal, Chapman, & Muse, 1995). Startle reactivity is thought to be linked to limbic involvement in the experience of negative emotions (e.g., Davis, 1992). Decreased startle might thus reflect suppression of low-level processing and lead one to expect—in line with current findings—no exaggerated peripheral physiological reactivity in HTSA individuals.

Several limitations of the present study must be acknowledged. First, our conclusions are based on "null findings," raising the concern that our study design was inappropriate or lacked statistical power to detect real differences between HTSA and LTSA participants. One concern is that the 3-min speeches or the 2-min recovery periods might have been too short to detect differences between anxiety groups. However, this seems unlikely for two reasons: First, we did find the expected group difference in self-reported anxiety; second, if we think of recovery as the ability to return to baseline quickly, we would expect group separations to occur earlier during the recovery period rather than later. We also think it unlikely for several reasons that our null findings are due to Type II error. First, we obtained a large number of measures of autonomic physiological responding, thus excluding the explanation that we just did not have sensitive measures. Group differences in autonomic habituation or recovery reported in prior studies occurred only in single autonomic measures and were not consistent across studies (e.g., Beidel et al., 1985; Eckman & Shean, 1997). This fact gives one further confidence in our finding no group differences. Further, we carefully selected participants with extreme (not just above average) scores on a measure of trait social anxiety and induced high levels of anxiety by requiring participants to give two highly stressful impromptu speeches. Lastly, if our null findings were due to a lack of statistical power, there should at least be trends in the means consistent with our hypotheses. However, Figure 2 shows that if there are trends for group separation (e.g., in heart rate, SCL, and RSA), they are in the direction of the LTSA group showing *higher activation* than the HTSA group.

A second limitation is that we used only female college students as participants. Although some studies indicate that the present findings might generalize to other populations (e.g., Panayiotou & Vrana, 1998; Puigcerver, Martinez-Selva, Garcia-Sanchez, & Gomez-Amor, 1989), there might also be important differences in physiological reactivity as a function of participants' age, sex, and culture (e.g., Grossman et al., 2001; Tsai, Levenson, & Carstensen, 2000). Moreover, although the SPAI scores of our high anxiety group were comparable to clinical groups in other studies ($M = 104.3$ in our sample vs. $M = 96.8$ in Beidel, Turner, Stanley, & Dancu, 1989), participants did not have a clinical diagnosis of social anxiety. In future studies, it will be important to assess physiological reactivity accompanying social anxiety in more diverse community samples including clinical groups.

A third important limitation of the present study is that it focused exclusively on social anxiety. It is important to be extremely careful when generalizing to other anxiety disorders. For example, Öhman (1986) showed different patterns of physiological activation in social anxiety as compared to specific phobias. Specifically, he noted that different types of fear might involve different evolutionary evolved "systems," leading to lesser autonomic nervous system activation in social phobia than in specific phobias. Thus, studies of other subtypes of anxiety are needed to investigate the generalizability of the present findings. If the lack of group separation does not generalize to other anxiety disorders or other emotional states, an examination of this effect's specificity could provide interesting insights into the psychophysiology of social anxiety.

[2]Because the MANCOVA is a conservative test of group differences, we conducted univariate tests of the Anxiety Group effect for the speeches as well as for the recovery periods on the eight physiological measures. None of those tests revealed a significant effect involving Anxiety Group.

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