Gender Differences in Psychophysiological Responses to Speech Stress Among Older Social Phobics: Congruence and Incongruence Between Self-Evaluative and Cardiovascular Reactions

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Objective: Evidence suggests increased cardiovascular risk and autonomic impairment among individuals with chronic anxiety. Little attention, however, has been paid to the anxiety disorder of social phobia despite its high prevalence. Additionally, gender- and age-related cardiovascular profiles have not been examined in relation to social phobia. This study investigated cardiovascular responses to a socially threatening situation among older men and women with social phobia and control subjects. **Methods:** Thirty subjects with social phobia and 30 control subjects (mean age = 65 years) were assessed during baseline, paced breathing, speech preparation, and speech presentation. Electrocardiographic variables, blood pressure, respiration, and emotional state (self-reported) were monitored. Hemodynamic variables included heart rate, blood pressure, cardiac output, and systemic vascular resistance; autonomic measures were respiratory sinus arrhythmia and baroreflex sensitivity, both markers of cardiac vagal control, and 0.10-Hz systolic blood pressure variability, an index of sympathetic vasomotor tone. **Results:** Subjects with social phobia, in contrast to nonanxious control subjects, manifested more anxiety, embarrassment, and somatic complaints in response to stress; however, physiological measures generally did not distinguish groups. Interaction effects indicated that socially phobic women were hyperresponsive to the stressor with respect to self-reported, hemodynamic, and autonomic parameters. Socially phobic men manifested no physiological differences in comparison with control subjects, but they reported more psychological and somatic complaints. **Conclusions:** Gender differences in subjective and physiological responses to a socially threatening situation indicate congruence between perceived social anxiety and physiological responses in older women but not men. We found no evidence of impaired cardiovascular autonomic regulation among socially phobic men despite other reports that phobically anxious men are at greater cardiovascular risk. **Key words:** anxiety disorders, social phobia, cardiovascular reactivity, heart rate variability, autonomic control, mental stress.

DBP = diastolic blood pressure; MANOVA = multiple analysis of variance; RSA = respiratory sinus arrhythmia; SBP = systolic blood pressure.

INTRODUCTION

Social phobia, or social anxiety disorder (1), is a frequently occurring anxiety disorder characterized by excessive and persistent fear of social or performance situations in which negative or humiliating scrutiny or evaluation by others is possible (2, 3). This condition interferes with normal work- and socially related functioning, and it is often marked by extreme distress and avoidance of the feared situations. Social anxiety has been reported to have a 1-month prevalence of up to 8% (4) and a lifetime prevalence of up to 15% (5). According to the National Comorbidity Survey, social phobia is the third most frequent psychiatric disorder in the United States and the most prevalent of all anxiety disorders. Results from this survey indicate that the lifetime prevalence of social phobia is 13.3%, ranking behind only major depressive episode (17.1%) and alcohol dependence (14.1%) (6, 7).

A number of investigations indicate that social anxiety has an earlier age of onset than depression and all other anxiety disorders except simple phobia (8–13). Social anxiety is apparently stable over many decades, with patients continuing to show persistent functional impairment and exaggerated interpersonal sensitivity (13, 14). The early onset of social phobia is of special interest because of the extremely high comorbidity of the disorder with other psychological conditions (8). Comorbidity with major depression and other anxiety disorders (eg, panic disorder) is particularly marked (4). These other disorders generally have a later age of onset (9, 15–17), and there is considerable evidence that social phobia typically precedes these disorders when it occurs with them (5, 11, 17–19). Additionally, social phobia shares many common features with other anxiety disorders (including specific somatic symptoms and discrete panic attacks) and may share a common pathophysiology (20–25).

Other research has indicated that phobic anxiety is a risk factor for death due to coronary heart disease among men (26–29) and that patients with other anxiety disorders, particularly panic disorder, may have impaired autonomic regulation of cardiovascular func-

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Received for publication May 22, 2000; revision received January 22, 2001.

0033-3174/01/6305-0765

tioning (30–32). These finding suggest that altered autonomic and hemodynamic activity among the chronically anxious may moderate adverse coronary events. However, no attempt has yet been made jointly to characterize cardiovascular autonomic and hemodynamic responses to a feared social performance situation. Nor has there been an attempt to examine gender effects in relation to cardiovascular reactivity and social phobia, although this may yield important insights illuminating possible mechanisms that mediate the elevated cardiovascular risk among chronically anxious middle-aged and elderly men, because virtually all the associations between anxiety and coronary risk have been found only in men. Given the high comorbidity between social phobia and other anxiety disorders, as well as the apparent overlap of symptoms and possibly shared pathophysiology between social phobia and panic disorder, an in-depth investigation of cardiovascular and autonomic regulation of social phobics seems warranted.

The cardiovascular research published thus far has documented only exaggerated heart rate responses during social performance activities among subgroups of social phobics characterized by fears of one or a few specific situations (eg, public speaking) (33–35). Social phobics whose fears generalize to a broad range of social and performance conditions do not seem to manifest abnormal cardiac reactions to personally relevant performance situations, although in comparison with social phobics with more limited, specific fears, those with generalized social phobia, paradoxically, do report higher levels of subjective anxiety and discomfort during laboratory social task performance. Nevertheless, there is no evidence that persons with specific or generalized social phobia show atypical cardiovascular *autonomic* responses, either sympathetic or vagal, to feared performance situations, although there is some evidence that social phobics demonstrate elevated sympathetic responses to physical challenges (36, 37). No physiological investigations have gone beyond simple heart rate analysis to include a multiparameter study of hemodynamic regulation during phobic stress among social phobics, including such biologically and medically significant measures as cardiac output and systemic vascular resistance. Additionally, to the best of our knowledge, all studies of social phobia have examined younger to middle-aged subjects, and none has explored cardiovascular and autonomic reactivity among clinically anxious patients in later years of life, when cardiovascular disease risk is highest.

We examined the cardiovascular autonomic and hemodynamic profiles of 30 older socially phobic men and women and 30 nonanxious control subjects matched for age, gender, body mass index, presence of hypertension, and use of cardioactive drugs. Subjects were studied during resting phases and during a speech performance task. A public-speaking task was chosen because speaking in public is the most commonly and almost universally feared of all performance situations among both generalized and discrete social phobics (38, 39). We hypothesized that older men and women with social phobia would display greater cardiovascular reactivity and more pronounced negative affect than matched, nonanxious control subjects.

METHODS

Subject Selection

Subjects were recruited from 1096 middle-aged to elderly participants of the Boston VA Normative Aging Study (40) and their wives (47–83 years of age) who had volunteered to participate in this population-based investigation. All subjects were first evaluated according to DSM-III-R criteria for anxiety, depressive, and dysthymic disorders using the Composite International Diagnostic Interview (41). Interview data were collected and audiotaped by trained lay interviewers; all data were later verified by a clinical psychologist with special diagnostic training. Subjects were screened for coronary heart disease, abnormal electrocardiographic findings, congestive heart failure, and diabetes; evidence of any of these disorders or current use of psychotropic medications disqualified them from this study. Eighty percent of the eligible Normative Aging Study participants were asked to take part in the study; the remaining 20% were not asked because of scheduling constraints. Of those asked, only 5.7% refused to participate, making systematic bias unlikely.

We studied 30 social phobics and the same number of nonanxious control subjects matched for age, gender, body mass index, hypertension, and the use of cardiovascular drugs for treatment of hypertension. Applying a case-control method, social phobics were individually matched to randomly selected, nonanxious control subjects from the larger group based on similarities of demographic variables. Twelve of the social phobics had generalized social phobia, and 18 had a specific phobia according to recently documented criteria (42). Demographic data are shown in Table 1.

Experimental Protocol

Conditions were presented in the following order: 1) resting baseline (10 minutes), 2) voluntary paced breathing at 15 cpm (5 minutes), 3) speech preparation (4 minutes), and 4) speech presentation (4 minutes).

Mental stress baseline phase. After DSM-IV assessment, subjects were fitted on the same day with electrocardiographic electrodes (leads V_5 and V_1), respiratory bands, and a Finapres finger cuff. They were then seated in a comfortable easy chair in a sound-attenuated room. The hand with the Finapres cuff remained on an armrest at approximately heart level throughout the study. A respiratory calibration procedure was then used to calibrate tidal volume from the Respitrace respiratory bands (43). After administration of questionnaires, calibration of signals, and monitoring the quality of registrations (about 10 minutes), patients were asked to relax quietly (baseline resting period, 10 minutes).

TABLE 1. Demographic and Baseline Characteristics of Subjects With Social Phobia and Nonanxious Control Subjects

Characteristic	Group	
	Social Phobia	Control
Gender, *N*		
Male	21	21
Female	9	9
Age, y (mean ± SD)	64.0 ± 7.9	66.0 ± 6.6
Body mass index, kg/m^2 (mean ± SD)	27.4 ± 4.3	27.5 ± 4.3
Heart rate, beats/min (mean ± SD)	65.5 ± 10.0	69.9 ± 13.0
Resting arm blood pressure, mm Hg (mean ± SD)		
Systolic	117 ± 11	122 ± 13
Diastolic	72 ± 8	75 ± 10
Hypertension, *N*	15 (48.4%)	15 (48.4%)
Cardiovascular medications	11 (35.5%)	11 (35.5%)

Paced breathing. This phase was used to evaluate individual differences in cardiac vagal control under standard and stable respiratory conditions (44, 45). Subjects were fitted with headphones attached to a cassette tape deck. A 5-minute taped acoustic tone pattern was presented to subjects with a periodicity of 15 counts/min. An ascending tone of the cassette tape corresponded to inspiration and a descending tone to expiration. Before data collection, subjects were trained to pace their breathing easily and comfortably to the signal. They were also instructed to maintain a eucapnic tidal volume by breathing comfortably but not too deeply. Once the appropriately paced respiration pattern was stable, recording commenced.

Speech task. Subjects were asked to recall a recent situation that made them feel anxious or angry. Four minutes were given to prepare responses to specific points, including a precise description of the circumstances and the people involved, the subject's physical and emotional responses, and how the subject later felt about the event and his or her reactions. Immediately after preparation, subjects were instructed to address all points while presenting their speech into a video camera and in front of an experimenter. It was indicated that the video recording would later be evaluated for content and style of the speech presentation. Physiological signals were recorded during both phases.

A 22-item self-evaluation scale of emotional state was administered immediately after each condition with the exception of speech preparation.

Physiological Parameters

Two channels of electrocardiographic response, beat-to-beat finger blood pressure (Finapres 2300, Ohmeda, Louisville, CO), and abdominal and thoracic respiration (inductive plethysmography, Respitrace, Ardsley, NY) were monitored. All physiological signals were continuously recorded for the entire length of each experimental phase. Analog data were converted on-line to digital values using a 12-bit A-D board (Dataq Instruments, Akron, OH) attached to a personal computer with a Pentium processor. All data were stored on computer. The electrocardiographic, Finapres blood pressure, and respiratory signals were each digitized at a sampling frequency of 1000 Hz.

Physiological signals were analyzed off-line by computer using customized programs written in the Matlab (Mathworks, Natick, MA) programming environment (46). A variable-threshold peak-detection program identified the R wave from the electrocardiographic signal. Suspect intervals were reviewed using the original electrocardiographic signal as means of verification. When an interval was ectopic or in some other way abnormal, it was deleted from the R-R interval series and replaced by a linearly interpolated value. Records with greater than 10% ectopic beats were not submitted to spectral analysis. A separate routine calculated beat-to-beat SBP and DBP. An artifact-detection and interpolation program for blood pressure was similar to that for the R-R interval. Differences in degrees of freedom reflect subject exclusion due to poor blood pressure or electrocardiographic signals for hemodynamic variables or excessive arrhythmic activity for frequency domain measures.

Time domain measures. Derived parameters included mean heart rate, respiration rate, tidal volume, SBP, and DBP. Finapres estimates of SBP and DBP have been highly correlated with invasive measures of aortic, brachial intra-arterial, and radial intra-arterial pressures (47–53). Additionally, another program (BEATFAST, TNO-Biomedical Instrumentation, Amsterdam, Netherlands) provided analyses of relative changes in left ventricular stroke volume, cardiac output, and systemic vascular resistance (54, 55). These time domain measures were averaged per minute within conditions. Frequencies of supraventricular and ventricular ectopy per condition were also recorded.

Respiratory variables of rate and tidal volume were analyzed and used as control variables to establish adherence to paced-breathing conditions and to ensure that high-frequency R-R interval power was, in fact, RSA (ie, coherent with respiration). Respiration rate and tidal volume were also analyzed during experimental conditions to exclude the possibility that task differences in RSA between groups were due to respiratory variation. Preliminary analyses confirmed that this was not the case.

Frequency domain measures. For these measures we used a spectral estimation procedure based on the Welch algorithm, which averages periodograms (56). Time series of beat-to-beat R-R intervals were linearly interpolated at a frequency of 4 Hz to obtain equally spaced events. For each experimental condition, 60-s data segments, overlapping by half, were then detrended, filtered with a Hanning window, and fast-Fourier-transformed to provide frequency content. Individual periodograms were subsequently averaged for each condition to produce estimates of spectral density. This approach provided a frequency resolution of 0.002 Hz, with the highest frequency evaluated at 0.50 Hz. Preliminary testing with simulated data yielded a flat frequency response characteristic across the frequencies of interest. Identical procedures were used for SBP and for respiration.

Two fixed bandwidths were defined for all power spectral analyses, based on optimal separation of different periodic components. The bandwidths were 0.070 to 0.1298 Hz (low-frequency band) and 0.13 to 0.50 Hz (high-frequency, or RSA, band). Previous research has determined that high-frequency R-R power is almost exclusively vagally mediated (45).

SBP spectra were calculated for low-frequency SBP oscillations. These seem to reflect sympathetic modulation of vasomotor activity (57–60). All R-R interval and SBP spectral data were transformed to natural logarithms to normalize distributions. To estimate baroreflex sensitivity, cross-spectral analysis was performed. The magnitude component of the transfer function provided this index, using SBP as input and R-R interval as output (61). Baroreflex sensitivity was the magnitude of R-R interval change per millimeter of mercury within the low-frequency band, where the coherence was greater than 0.5.

Self-report measures of emotion. A specially constructed 22-item self-evaluation scale assessed different emotions and somatic symp-

toms on a scale of 0 to 10 (not at all to very much). Emotional items had face validity and included such general categories of negative emotions as embarrassment (embarrassed, ashamed, flustered), anxiety (anxious, nervous, tense, worried), and hostility (angry, unfriendly, annoyed, resentful), and positive affects of well-being (happy, calm, relaxed) and confidence (confident, enthusiastic, proud, eager); somatic items included racing heart, chest pain, and shortness of breath. The general categories were decided on an a priori basis, and scores of individual items were summed (Cronbach's α values during baseline = 0.84, 0.80, 0.85, 0.73, 0.78, and 0.71, respectively). In addition, we used the two explicitly cardiac symptoms summed together as an index of specific heart-associated complaints. One item, excited, was excluded from analyses because of its ambiguous positive and negative valence. Most of the above items have been previously categorized into similar subscales yielding equivalent reliabilities (eg, Ref. 62).

Statistical Analyses

Analyses of state self-evaluation measures of emotion were performed using the Mann-Whitney *U* test. For analyses of physiological data, we used repeated-measures MANOVAs with anxiety diagnosis (social phobic vs. nonanxious) and gender as grouping factors. In separate analyses, subtypes of social phobics (specific vs. generalized) constituted the grouping factor with the same dependent measures. The Tukey Honestly Significant Difference test and α error–adjusted paired *t* tests, where appropriate, were used for post hoc analyses to examine specific effects ($p < .05$). The Tukey test was used for group comparisons within conditions, whereas paired *t* tests were used to examine within-group effects across two conditions.

RESULTS

Self-Evaluation Scales

Mann-Whitney *U* tests indicated that social phobics reported more anxiety during baseline than nonanxious control subjects (mean = 3.5 vs. 1.1, $p < .007$) and more somatic complaints (mean = 0.8 vs. 0.4, $p < .04$; Table 2). Analysis of change scores between baseline and speech presentation showed greater increases among social phobics in total anxiety score (mean = 15. 2 vs. 5.3, $p < .0009$), total somatic complaints (mean = 3.2 vs. 0.3, $p < .004$), and specific cardiac-associated symptoms (mean = 2.2 vs. 0.2, $p < .04$). Within-gender comparisons also revealed significant differences in speech-related anxiety and somatic responses between social phobics and control subjects of each gender.

Comparisons of socially phobic men and women, nevertheless, indicated that women demonstrated somewhat higher baseline levels of somatic symptoms (mean = 1.3 vs. 0.7, $p < .04$) and substantially more exaggerated responses from baseline to speech: During speech presentation, socially phobic women, compared with their male counterparts, exhibited greater increases in anxiety (mean = 26.0 vs. 10.6, $p < .005$), hostility (mean = 22.4 vs. 5.9, $p < .007$), embarrassment (mean = 12.3 vs. 6.1, $p = .05$), total somatic complaints (mean = 6.8 vs. 1.9, $p = .05$), and cardiac-associated complaints (mean = 4.6 vs. 1.2, $p < .04$). These responses of socially phobic women were also greater than those of any other subgroup (ie, nonanxious women or men).

Time Domain Physiological Parameters

Heart rate. A 2 × 2 × 3 repeated-measures MANOVA (Diagnosis [socially anxious, nonanxious] by Gender [male, female] by Condition [baseline, preparation, speech]) indicated that heart rate increased from baseline to both preparation and speech presentation (Rao $R(2,53) = 74.87$, $p < .0001$). There was also a significant Gender by Condition interaction (Rao $R(2,53) = 8.05$, $p < .0009$), indicating that women had greater heart rate increases in response to both tasks

TABLE 2. Baseline Mean Levels and Baseline-to-Speech Changes of Reported Emotions and Somatic Complaints for Subjects with Social Phobia and Nonanxious Control (±SD) Subjects[a]

Variable	Group			
	Social Phobia		Control	
	Baseline	Speech Response	Baseline	Speech Response
Anxiety	3.5 ± 5.6[b]	15.2 ± 12.3[c]	1.1 ± 2.5[b]	5.3 ± 6.0[c]
Embarrassment	1.2 ± 4.4	8.0 ± 7.5[d]	0.6 ± 1.8	4.7 ± 5.0[d]
Hostility	1.0 ± 4.4	11.0 ± 14.1	0.3 ± 1.0	5.3 ± 8.4
Well-being	19.4 ± 7.8	−4.1 ± 8.9	20.3 ± 7.2	−5.8 ± 7.3
Confidence	18.1 ± 10.2	−8.7 ± 9.5	16.1 ± 10.4	−7.5 ± 8.2
Somatic complaints	0.8 ± 1.9[b]	3.2 ± 4.9[c]	0.4 ± 1.4[b]	0.3 ± 1.3[c]
Heart complaints	0.5 ± 1.5	2.2 ± 3.8[c]	0.3 ± 1.1	0.2 ± 1.3[c]

[a] Mann-Whitney *U* test used to determine differences between groups.
[b] Baseline differences, *p* values < .04 to .007.
[c] Speech responses, *p* values < .04 to .0001.
[d] Speech responses, $p < .09$.

than men (Figure 1). Additionally, a Diagnosis by Gender interaction closely approached significance ($F(1,54) = 3.30$, $p = .07$). Post hoc tests revealed that socially phobic women had higher heart rates and greater stress responses than socially phobic men. There were no differences in heart rate level or task-related change between nonanxious men and women.

Systolic blood pressure. Analysis of baseline, speech preparation, and speech presentation revealed a significant effect of Condition (Rao $R(2,51) = 48.91$, $p < .0001$); post hoc tests indicated that SBP increases in response to the two stressors were significant from baseline SBP and from each other (Figure 2). There was also a Diagnosis by Gender interaction effect ($F(1,52) = 10.47$, $p = .002$), whereby socially phobic women had higher levels of SBP across all conditions compared with female control subjects (Figure 2).

Diastolic blood pressure. Similar to SBP, there were significant effects for stress conditions (Rao $R(2,51) = 62.70$, $p < .0001$) and a Diagnosis by Gender interaction effect across the baseline-to-stress analyses ($F(1,52) = 9.72$, $p < .003$; Figure 2). Post hoc tests indicated that female social phobics manifested higher DBP levels than both male social phobics and nonanxious female control subjects.

Stroke volume. A Conditions effect (Rao $R(2,45) = 35.12$, $p < .0001$) and subsequent post hoc tests indicated that baseline, preparation, and speech presentation all differed from each other; stroke volume was lowest during speech and highest during speech preparation (Figure 3). Additionally, there were both two-way (Gender by Conditions: Rao $R(2,45) = 5.34$, $p < .0083$) and three-way (Diagnosis by Gender by Conditions: Rao $R(2,45) = 3.20$, $p = .050$) interaction effects. Post hoc analyses indicated that men maintained similar levels of stroke volume during baseline and speech preparation but decreased levels during actual presentation. Women, on the other hand, had similar levels of stroke volume at baseline and during the speech but increased levels during speech preparation. Finally, the three-way interaction indicated that among women, only social phobics had significantly decreased stroke volume from speech preparation to presentation, whereas among men, only the nonanxious subjects showed reductions during speech presentation.

Cardiac output. Analysis of baseline, speech preparation, and presentation yielded Conditions (Rao $R(2,49) = 30.72$, $p < .0001$), Gender by Conditions (Rao $R(2,49) = 5.93$, $p < .005$), and three-way interaction effects (Rao $R(2,49) = 6.29$, $p < .004$; Figure 4). Post hoc results indicated greater increases in cardiac output among women from baseline in response to both stressors. Furthermore, socially phobic women displayed the greatest cardiac output reactivity in response to speech preparation, whereas socially phobic men did not exhibit differences in cardiac output from baseline to stress.

Systemic vascular resistance. Analysis of baseline-to-stress conditions yielded Conditions (Rao $R(2,48) = 30.12$, $p < .0001$) and Gender by Condition effects (Rao $R(2,48) = 5.02$, $p = .01$; Figure 5). Systemic vascular resistance differed among all conditions, with highest levels during speech presentation and lowest levels

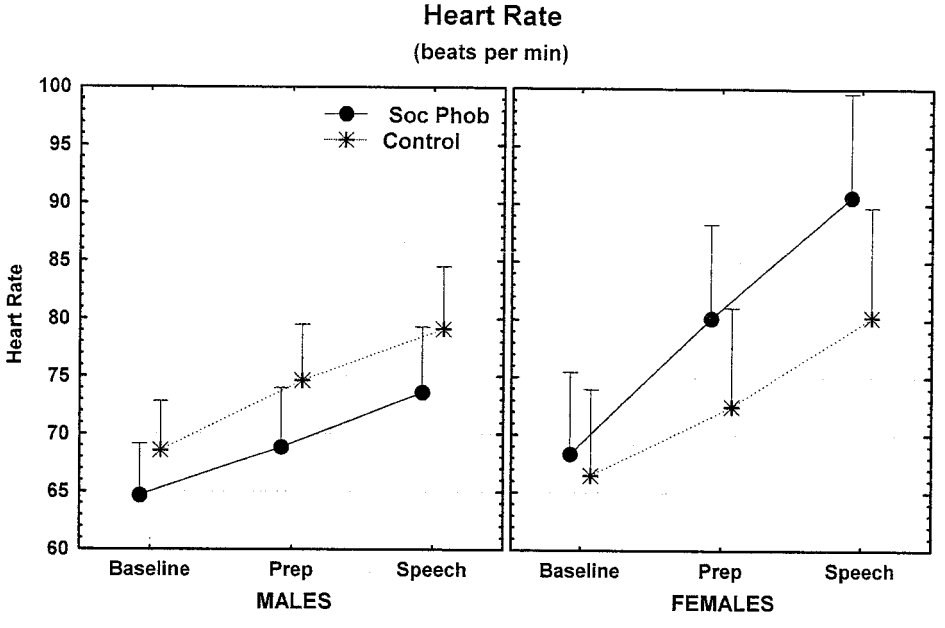

Fig. 1. Heart rate by gender and diagnostic group.

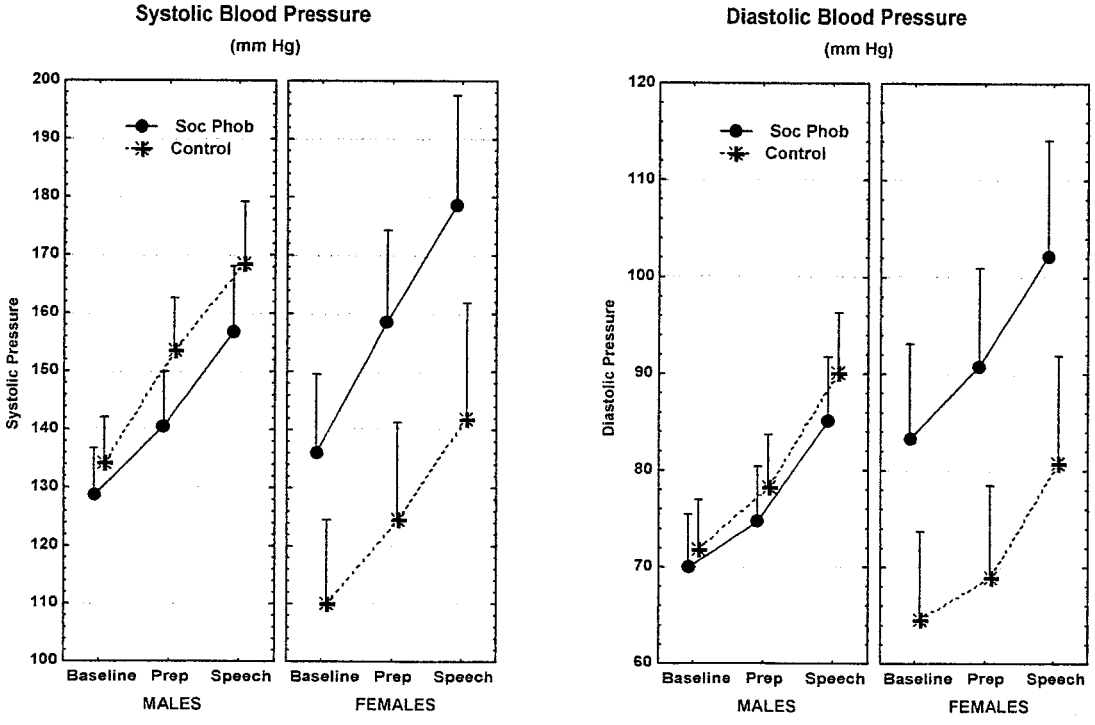

Fig. 2. Systolic and diastolic blood pressures by gender and diagnostic group.

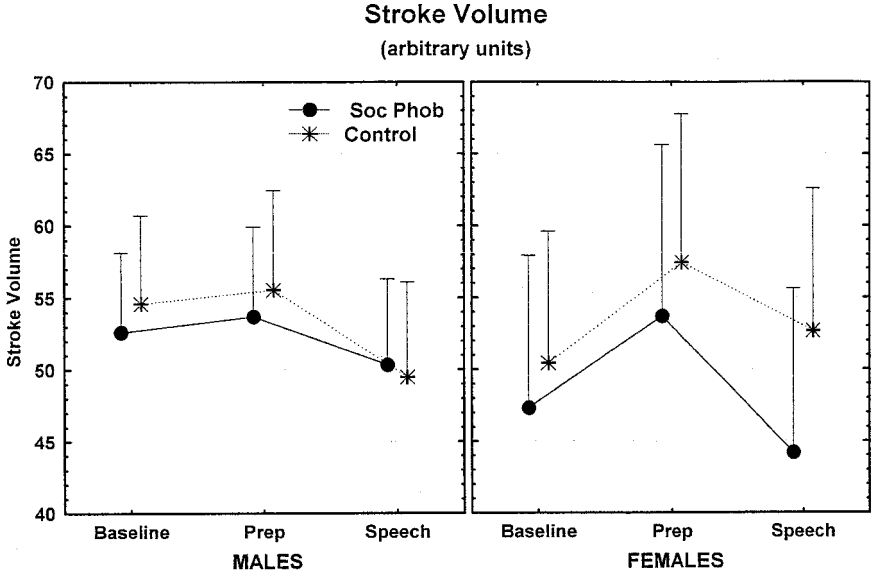

Fig. 3. Left ventricular stroke volume by gender and diagnostic group.

during preparation. Post hoc tests of the two-way interaction indicated that baseline-to-preparation decreases were characteristic only for women, but both genders showed increased resistance from speech preparation to presentation.

Ventricular and atrial ectopic activity. Three subjects (two social phobics and one control subject) manifested very modest ventricular ectopic activity during the study: one social phobic had 16 ectopic beats during baseline, which was reduced to 4 during speech stress, and the other two had just 1 ventricular ectopic beat in a single condition. Atrial ectopic activity was more frequent, occurring in 11 subjects (three social phobics and eight control subjects); the greatest number of atrial ectopic beats occurred during baseline (range, 0–12; mean = 3), followed by speech stress

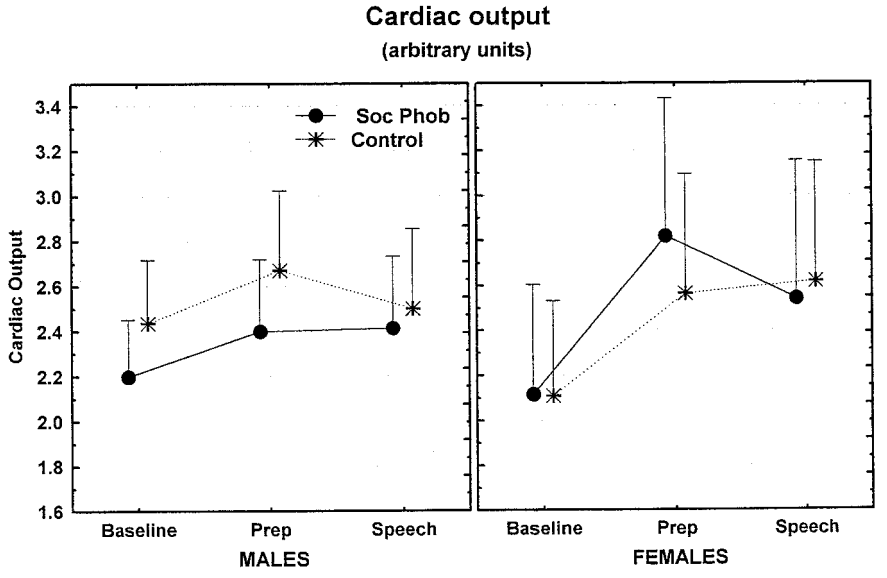

Fig. 4. Cardiac output by gender and diagnostic group.

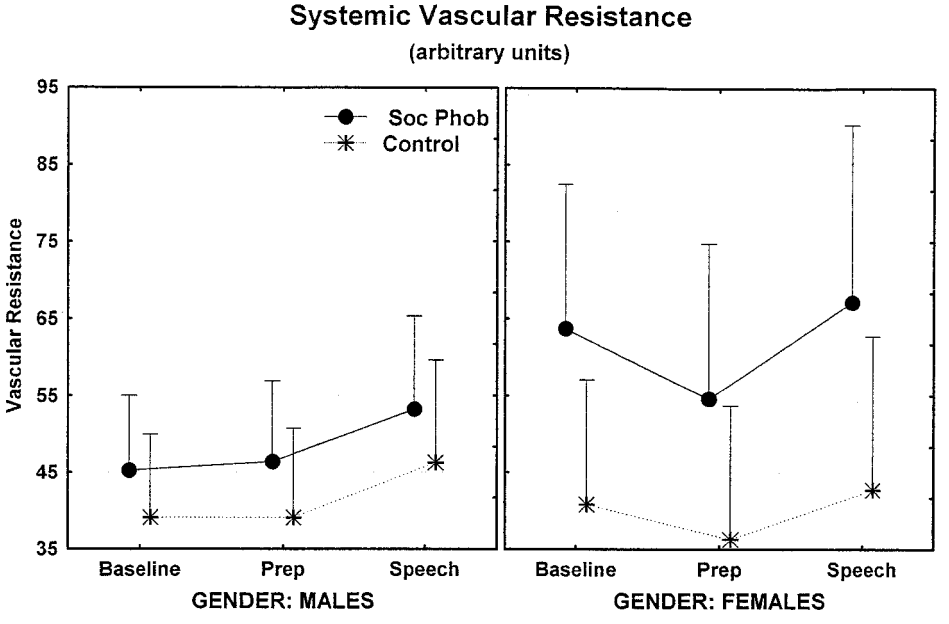

Fig. 5. Systemic vascular resistance by gender and diagnostic group.

(range, 0–5; mean = 1.9). Paired *t* tests comparing conditions among subjects with atrial ectopic activity were not significant. Thus, premature atrial and ventricular beats were rare in our sample, making it unlikely that ectopic activity could have influenced other results (although our findings may not be representative of older populations with greater ectopic activity).

Frequency Analysis Parameters

Cardiac vagal indices. Analysis of RSA (high-frequency R-R interval power) across baseline and stress levels indicated borderline significant interaction effects for both Diagnosis by Condition (Rao $R(2, 51) = 3.03$, $p < .06$) and Gender by Condition (Rao $R(2,51) = 2.81$, $p < .07$; Figure 6). Post hoc analyses suggested that during speech presentation RSA was reduced from both baseline and speech preparation levels only in socially phobic women. No group differences were found for respiratory variables (rate or tidal volume) that could account for variations in RSA, and slower respiration rates during speech, compared with baseline and preparation phases, suggested that true vagal attenuation had occurred during speech presentation

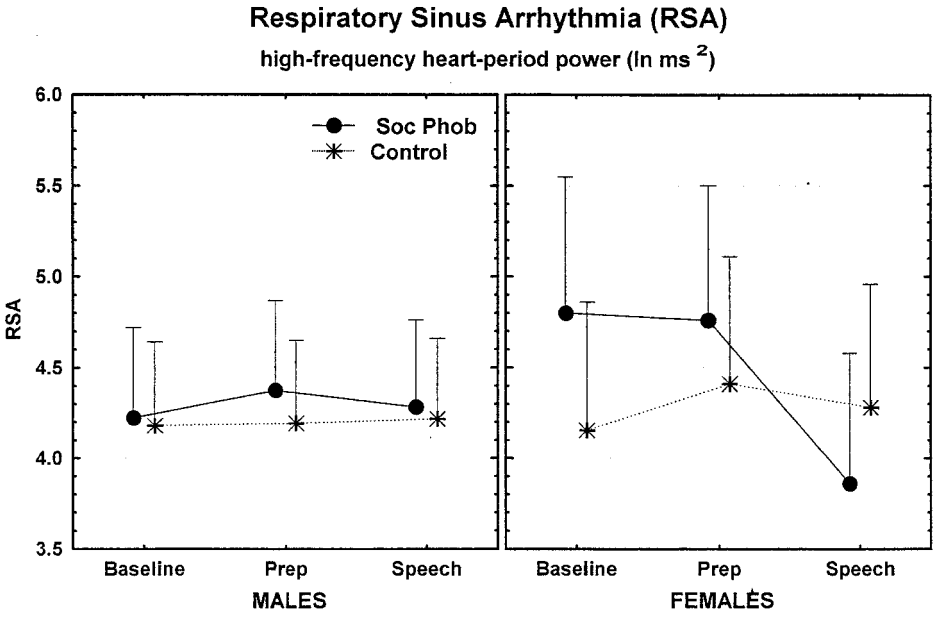

Fig. 6. Respiratory sinus arrhythmia by gender and diagnostic group.

for female social phobics (44, 45). Separate analyses for the paced-breathing condition yielded no group or interaction effects for RSA.

Spontaneous baroreflex sensitivity also paralleled the Diagnosis by Condition effects across baseline and stress conditions (Rao $R(2,42) = 3.22$, $p < .05$), although gender did not further interact (Figure 7). Post hoc tests indicated that only social phobics showed diminished baroreflex gain during speech presentation.

Sympathetic vasomotor index. Low-frequency blood pressure oscillations increased from baseline to stress levels (Rao $R(2,42) = 51.29$, $p < .0001$). Additionally, a three-way interaction effect was significant (Rao $R(2,42) = 3.37$, $p = .04$), and post hoc tests indicated that socially phobic women showed an exaggerated increase in magnitude of these oscillations during stress (Figure 8).

Analyses of specific vs. generalized subtypes of so-

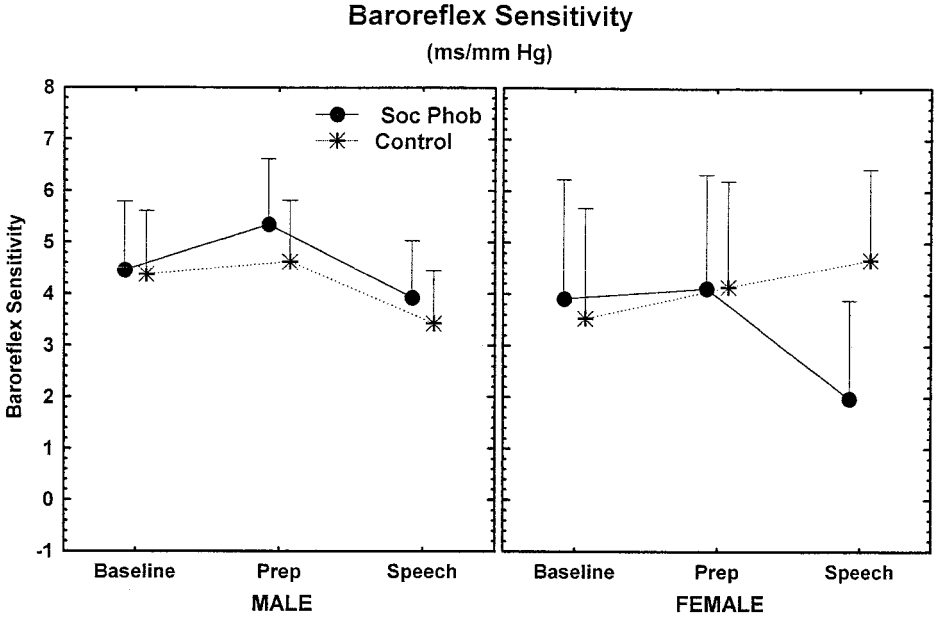

Fig. 7. Cardiac baroreflex sensitivity by gender and diagnostic group.

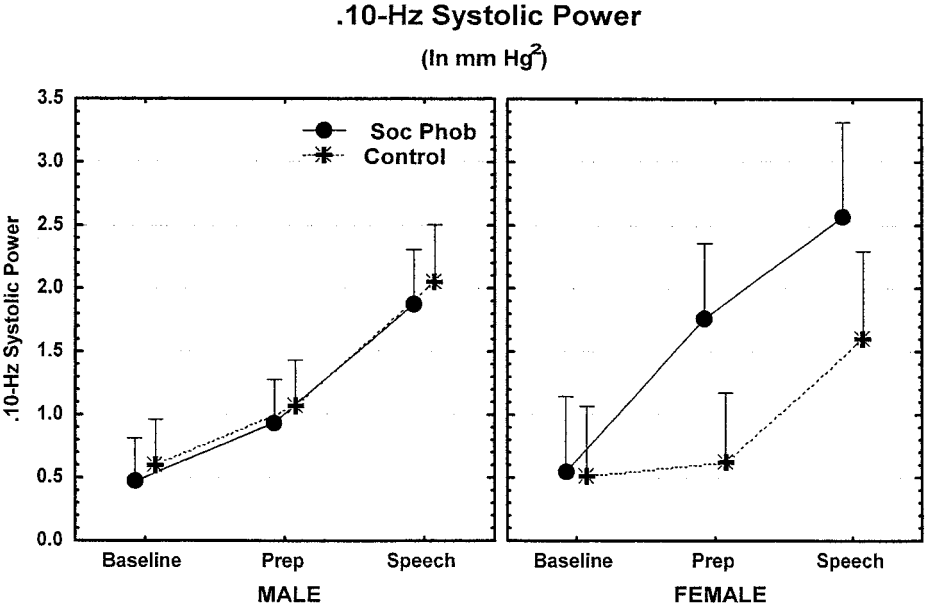

Fig. 8. Systolic blood pressure spectral power (0.10 Hz) by gender and diagnostic group.

cial phobia. We also analyzed all dependent variables comparing subjects with specific social phobia and subjects with generalized social phobia (42), but we found no significant differences on any self-report or physiological measure.

DISCUSSION

Our study focused on social phobia in a late middle-aged and elderly sample. To our knowledge, this is the first examination of social phobia among older individuals, as well as the first to characterize and provide an integrated analysis of cardiovascular, autonomic, and hemodynamic responses to a social performance stressor. In our investigation, subjects with social phobia, in contrast to matched, nonanxious control subjects, reported substantially greater emotional responses and somatic complaints after presentation of a provocative speech. The most pronounced differences between groups occurred for anxiety and somatic symptoms, the latter being chest complaints typically perceived as related to heart or respiratory function. These two categories of symptoms showed higher responses to speech among social phobics both within and across genders. Nevertheless, socially phobic women reported markedly greater anxiety and somatic reactions to speech than either their male counterparts or nonanxious female control subjects. Socially phobic women also responded to speech stress with considerably more embarrassment and hostility than other groups.

Physiological data revealed that speech preparation and speech presentation elicited large cardiovascular responses across groups. However, there was no simple relationship between cardiovascular reactivity and self-reported negative affect or somatic complaints. To the contrary, inspection of the cardiovascular autonomic and hemodynamic results provides evidence of both congruence and incongruence between physiological and self-reported emotional/somatic responses. In support of incongruence, we could find little evidence of exaggerated autonomic or hemodynamic responses among social phobics as a whole. Despite the greater levels and reactions of psychosomatic self-report measures that occurred among social phobics, only the cardiac vagal index of baroreceptor sensitivity showed a two-way Diagnosis by Condition interaction effect, indicating greater vagal withdrawal during speech presentation among the socially anxious. Even with this measure, the data presented in Figure 7 suggest an impact of gender, although no significant interactions effects were found.

Congruence between physiological parameters and self-reports of emotional/somatic symptoms was approached only when gender was, in fact, considered: Among both self-report and physiological measures, Gender by Diagnosis, Gender by Conditions, and three-way interaction effects were found for almost all physiological measures, both autonomic and hemodynamic. Consistent with their apparent self-reported hyperreactivity, socially phobic women also showed the greatest physiological responses to speech stress. This was true for hemodynamic variables, including heart rate, SBP, DBP, cardiac output, and systemic

vascular resistance. Also, spectral indices of cardiac interval and blood pressure variability indicated greater stress-related cardiac vagal attenuation (RSA) and sympathetic vasomotor activation (low-frequency blood pressure variability). These differences were not accounted for by variations in subtype of social phobia, possibly because our definition of subtype (42), based on the Composite International Diagnostic Interview, by necessity, differed somewhat from criteria used in other investigations (33–35). Nor can these results be readily explained by a greater incidence of hypertension among socially phobic women. In fact, only one of nine socially phobic women had a history of hypertension, and she was taking pressure-lowering medication; two of nine nonanxious female control subjects had a history of hypertension, and both were taking pressure-lowering drugs.

These data, nevertheless, also indicate that a history of hypertension and current cardiovascular drug usage were much more prevalent among men: Fourteen socially phobic men and 13 male control subjects had a history of hypertension (of whom 10 and 9, respectively, were using cardiovascular medication), and both factors were relatively evenly distributed among the two groups of men. Because hypertension status and medication usage was so disproportionate across gender, it is possible that these factors may have importantly influenced our findings. For this reason, we performed post hoc analyses on just the subgroup of 30 nonhypertensive subjects. The pattern of Gender by Diagnosis interactions remained very similar to that from analyses using the full sample, although effects only sometimes remained significant, possibly because of reduced power. Two noteworthy effects, however, that did stay highly significant were Gender by Diagnosis interactions effects, both for SBP (p values $<$ .0004 and 0.008, respectively), suggesting that blood pressure effects were not merely due to gender differences in incidence of hypertension or its treatment. Still it would be important to replicate our findings with gender groups more evenly balanced for these variables.

There are several other possible reasons why socially phobic women in our study displayed the greatest physiological and emotional reactivity. First, a number of reports indicate that clinically anxious and socially phobic women are more severely fearful than their male counterparts (63–65). Alternatively, women may tend to have less experience than men in socially relevant performance situations, and this may predispose socially anxious women to greater reactivity during speech presentation. Our sample of men was drawn from a long-term epidemiological investigation requiring laboratory participation over many years in a number of unrelated studies. This may have served to habituate socially phobic men to the laboratory environment to a greater extent than the socially anxious wives of the male cohorts, who certainly had less general experience in this setting. Finally, our sample of socially phobic women was unfortunately rather small (N = 9), and these results require replication in a large sample.

The lack of any cardiovascular effects of stress for socially phobic men, on the other hand, was very consistent across all autonomic and hemodynamic variables (separate MANOVAs, not presented, were also performed only for men and yielded no significant effects for Diagnosis or Diagnosis by Conditions interactions). This suggests that socially phobic men do not differ from nonanxious men in cardiovascular regulation. Given the elevated coronary risk among phobically anxious men reported in the literature, it would, therefore, seem either that coronary risk is not elevated in this specific anxiety disorder of social phobia or that any such risk may be due to factors other than variations in cardiovascular or autonomic regulation, in agreement with several studies that have found no autonomic impairment in anxiety disorders (66–68).

Because social phobia is such an early occurring and stable disorder (10–13) and one commonly followed by other major anxiety disorders and depression (5, 11, 17–19), the absence of any evidence of atypical cardiovascular control among older socially phobic men (presumably typically afflicted for many years) seems especially relevant: Even such a tenacious and stable disorder does not seem to exert a significant effect on cardiovascular regulation. Also, the fact that earlier laboratory studies of cardiovascular regulation and anxiety have relied heavily on female subjects and have not taken into account gender differences in their results or interpretations (with the exception of Ref. 29) may require us to reassess previously reported associations between anxiety and cardiovascular function (because the epidemiological studies showing cardiovascular risk have been composed almost exclusively of male samples).

Our overall results (ie, with gender pooled) are largely consistent with those of other studies of younger social phobics (33–35): In contrast to control subjects, self-report measures of task-related negative affect were elevated in our older social phobics, but there were almost no physiological differences between groups. This incongruence between heightened self-report measures of distress and normal cardiovascular responses is also similar to what has often been found for other major anxiety and anxiety-related psychosomatic disorders (69–71). Such evidence points to a possible dissociation of physiological (at least car-

diovascular) and psychological mechanisms in the maintenance of social phobia and may support the primacy of cognitive/emotional factors in social phobia.

One must, however, be careful not to exclude physiological contributions prematurely: Laboratory studies of anxiety may present a distorted picture of what happens in the real world, and thus far, there has been insufficient emphasis in anxiety research placed on longer-term ambulatory monitoring of hemodynamic measures and cardiovascular variability parameters in natural settings. Additionally, even well-controlled psychophysiological studies are relatively few and have typically investigated social phobics presumably afflicted for some time. Physiological reactivity may, however, play a varying role depending on the phase of the disorder, with hyperreactivity possibly occurring during onset and then habituating over time toward more typical levels of response, except, perhaps, under extremely threatening conditions. Our results with socially phobic women, who reported the highest levels of distress in response to the speech presentation, indicate, in fact, that exaggerated cardiovascular responses may occur among social phobics when they experience extreme emotional distress.

Alternatively, of course, our findings may point to either constitutional differences between men and women with this disorder or to more situational aspects of how men and women react to individual task demands (eg, due to social factors or degree of prior experience with the task rather than constitution). Such gender differences may require different experimental and analytic models for appropriate investigation, including separate analyses by gender (as we reported above for men). At the very least, relations between physiological and experiential dimensions in social phobia (as well as, perhaps, in other anxiety disorders) require further exploration into gender differences and phase and subtype of disorder. Only then will we begin also to understand the extent to which dispositional and situational aspects of physiological activity are relevant to social phobia.

This work was supported by Grant HL 54098 from the National Heart, Lung, and Blood Institute, National Institutes of Health. The Normative Aging Study is supported by the Cooperative Studies Program/Epidemiology Research and Information Center of the US Department of Veterans Affairs and is a component of the Massachusetts Veterans Epidemiology Research and Information Center. Dr. Sparrow is a Research Career Scientist of the Medical Research Service of the Department of Veterans Affairs.

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