Age-Related Differences in Cardiovascular Reactivity During Acute Psychological Stress in Men and Women

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We examined potential age and gender differences in cardiovascular reactivity during acute psychosocial stress in 133 normotensive participants using a cross-sectional design. Results revealed that age predicted increased systolic blood pressure (SBP) reactivity during stress (p < .001). The greater SBP reactivity found in older individuals appeared due to an age-associated increase in both cardiac output and total peripheral resistance during stress as statistically controlling for these changes rendered the age and SBP reactivity effect nonsignificant. Similar analyses revealed that the age-related increase in cardiac output reactivity appeared to be driven by increased cardiac sympathetic control of myocardial contractility as measured by pre-ejection period. Older individuals also had greater vagal withdrawal during stress compared to younger individuals as indexed by respiratory sinus arrhythmia (p < .01). These results were comparable for men and women, and could not be explained by task-specific affective responses, task performance, or demographic factors. Implications for the study of age, cardiovascular reactivity, and health are discussed.

GERING is associated with changes in physiological function that confer increased vulnerability to physical health problems (Kart, Metress, & Metress, 1992; Timiras, 1994). There are well-documented age-related differences in resting cardiovascular function (e.g., increased blood pressure) that may influence cardiovascular disease risk (Timiras, 1994). However, much less is known about the influence of age on cardiovascular reactivity during psychosocial stress (Boutcher & Stocker, 1996; Jennings et al., 1997). According to the reactivity hypothesis, stress-induced changes in cardiovascular function may be independent predictors of the development and expression of cardiovascular disease (Kamarck et al., 1997; Krantz et al., 1996; Jennings et al., 1997). A study of age, cardiovascular reactivity, and health are discussed.

It is important to note that much less is known about the association between age and cardiovascular reactivity during psychosocial stress. The existing studies have mostly examined age-related differences in blood pressure and heart rate reactivity during stress (Faucheux, Dupuis, Baulon, Lille, & Bourliere, 1983; Garwood et al., 1982; Ginter, Hollandsworth, & Intrieri, 1986; Steptoe et al., 1990). These prior studies suggest that older adults have greater SBP reactivity than their younger counterparts, with no apparent difference in DBP reactivity (Faucheux, Bourliere, Baulon, & Dupuis, 1981; Garwood et al., 1982; Johansson & Hjalmarson, 1988; Steptoe et al., 1990; but see Ginter et al., 1986). The data for heart rate are more equivocal, but some studies suggest an age-related decrease in heart rate reactivity during stress (e.g., Barnes, Raskind, Gumbrecht, & Halter, 1982; Ginter et al., 1986; but see Steptoe et al., 1990).

An important issue for the prior research on age and reactivity concerns the mechanisms responsible for these multiply determined cardiovascular endpoints. For instance, blood pressure alterations are driven by changes in cardiac output and/or total peripheral resistance. An examination of these underlying determinants of blood pressure (as well as factors that contribute to each) may elucidate more specific mechanisms underlying these age-associated cardiovascular differences. To this point, impedance cardiography might be useful to model these underlying mechanisms as it provides noninvasive, reliable estimates of cardiac output and total peripheral resistance (Kamarck et al., 1992; Sherwood et al., 1990). Impedance cardiography can also be used to measure the systolic time intervals reliably and thus provide information on the contractile properties of the heart (e.g., pre-ejection period) potentially responsible for changes in cardiac output.

There appear to be only two studies that have examined age-related difference in cardiovascular reactivity during psychological stress utilizing impedance cardiography (Boutcher & Stocker, 1996; Jennings et al., 1997). These studies have produced conflicting results. Boutcher and Stocker (1996) exam-
vided cardiovascular reactivity during a 2-minute Stroop task in a sample of 15 young (mean age = 21) and 15 older (mean age = 59) men. Results revealed that increasing age was associated with decreases in total peripheral resistance reactivity and no differences in CO reactivity. Inconsistent with prior research, these authors also found no age-related difference in SBP reactivity. However, the relatively small sample size and short task may have limited the ability of this study to detect some of these age-related differences.

Jennings and colleagues (1997) utilized a large sample of 902 men (aged 46 to 64) in the Kuopio Ischemic Heart Disease Risk Factor Study. Multiple stressors were utilized in this study as part of the Pittsburgh reactivity battery. These tasks consisted of a target shooting task, memory scanning task, maze tracking task, and Stroop task. Cardiovascular responses to these diverse (but correlated) tasks were aggregated to increase the reliability and generalizability of the cardiovascular reactivity assessment (Kamarck et al., 1992). Jennings and colleagues then computed a composite measure of cardiac reactivity based on measures of SBP, pre-ejection period, and heart rate changes during stress, as well as a composite measure of vascular reactivity based on changes in DBP and stroke volume during stress. Results revealed that older men had greater stress-induced reactivity on both indices of cardiac and vascular reactivity. Consistent with Boutcher and Stocker (1996), Jennings and colleagues did not find any age related differences in parasympathetic reactivity to stress, at least as indexed by a task known to elicit a vagal-mediated heart rate deceleration (i.e., anticipatory response to shooting task). These authors interpreted these data as primarily reflecting an age-related increase in sympathetic control of the cardiovascular system. This interpretation is consistent with Esler and colleagues (1995) who found an age-related increase in cardiac sympathetic spillover during mental stress.

There are several important limitations of the aforementioned studies examining age-related differences in impedance-derived measures of cardiovascular reactivity. One limitation is that these studies have not utilized the full range of ages spanning young to older adulthood. A better characterization of these age differences might be obtained with data across broader age ranges (Lakatta, 1993). A second limitation is that both of these studies only examined men. In general, most of the prior studies on age and blood pressure reactivity to stress have not contrasted men and women in the same study. However, of the few studies examining both men and women, none have reported gender differences in age-associated blood pressure and heart rate reactivity during stress (e.g., Johansson & Hjalmarson, 1988). Based on these data it is possible that there might not be any gender difference in age-associated impedance-derived reactivity measures as these indices in theory model the mechanisms responsible for blood pressure changes. This prediction assumes that the same blood pressure change is not being achieved by different mechanisms in men and women.

In the present study, we examined age-related differences in blood pressure and impedance-derived measures of cardiovascular reactivity during stress utilizing both men and women between the ages of 30 and 70. Based on prior research, we predicted that older individuals would have greater SBP reactivity during stress compared to their younger counterparts. Furthermore, these age-associated differences in cardiovascular reactivity are predicted to be comparable in men and women. As reviewed above, the data on mechanisms responsible for the above differences in predictions are limited and conflicting (Boutcher & Stocker, 1996; Jennings et al., 1997). Therefore, we further investigate the underlying mechanisms responsible for these hypothesized effects using impedance cardiography.

**METHOD**

**Participants**

Sixty-four men and 69 women between the ages of 30 and 70 participated in this study. Approximately equal numbers of men and women were recruited from each decade group (e.g., 30 to 39; see Table 1) through advertisements placed in local newspapers. Individuals were paid $35.00 for approximately 2.5 hours of participation. Consistent with our prior research, the following self-reported inclusion criteria were used to select healthy participants: no existing hypertension; no cardiovascular prescription medication use; no past history of chronic disease with a cardiovascular component (e.g., diabetes); no recent history of psychological disorder (e.g., major depressive disorder); no tobacco use; and no consumption of more than 10 alcoholic beverages per week. Demographic characteristics of our sample are detailed in Table 1.

**Table 1. Sample Demographic Characteristics as a Function of Gender**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men (n = 64)</th>
<th>Women (n = 69)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age Frequency Distribution</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–39</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>40–49</td>
<td>18</td>
<td>19</td>
</tr>
<tr>
<td>50–59</td>
<td>17</td>
<td>21</td>
</tr>
<tr>
<td>60–70</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Age (Mean)</td>
<td>46.9</td>
<td>47.5</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partial high school</td>
<td>3.1%</td>
<td>1.4%</td>
</tr>
<tr>
<td>Graduated from high school</td>
<td>4.7%</td>
<td>8.7%</td>
</tr>
<tr>
<td>Partial college</td>
<td>32.8%</td>
<td>30.4%</td>
</tr>
<tr>
<td>Graduated from college</td>
<td>18.8%</td>
<td>34.8%</td>
</tr>
<tr>
<td>Partial graduate/professional school</td>
<td>10.9%</td>
<td>8.7%</td>
</tr>
<tr>
<td>Completed graduate or professional school</td>
<td>29.7%</td>
<td>15.9%</td>
</tr>
<tr>
<td>Yearly Income (Median)</td>
<td>$15,000–19,999</td>
<td>$15,000–19,999</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>84.4%</td>
<td>78.3%</td>
</tr>
<tr>
<td>African American</td>
<td>1.6%</td>
<td>0%</td>
</tr>
<tr>
<td>Hispanic/Latino</td>
<td>3.1%</td>
<td>2.9%</td>
</tr>
<tr>
<td>Asian/Pacific Islander</td>
<td>7.8%</td>
<td>11.6%</td>
</tr>
<tr>
<td>Native American</td>
<td>1.6%</td>
<td>5.8%</td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single/never married</td>
<td>23.4%</td>
<td>20.3%</td>
</tr>
<tr>
<td>Married/living with partner</td>
<td>56.3%</td>
<td>40.6%</td>
</tr>
<tr>
<td>Divorced</td>
<td>14.1%</td>
<td>26.1%</td>
</tr>
<tr>
<td>Separated</td>
<td>4.7%</td>
<td>2.9%</td>
</tr>
<tr>
<td>Widowed</td>
<td>1.6%</td>
<td>8.7%</td>
</tr>
</tbody>
</table>

**Note:** One woman had missing data for ethnicity and marital status.
Procedure

Potential participants were recruited through advertisements in the local papers. The 144 individuals who responded to these advertisements were first screened by telephone according to the inclusion criteria detailed above. Qualifying individuals were scheduled for an appointment, and participants' self-reports were again checked for reliability against the inclusion criteria. The follow-up check against the inclusion criteria revealed that 11 participants were ineligible, mostly due to differences in reported use of depression medication. Thus, the final sample consisted of 133 men and women. These participants completed an informed consent document and a demographic background questionnaire. Following completion of these questionnaires, the participant's height and weight were obtained using a standard medical scale from which body mass index was calculated (i.e., weight in kg/height² in meters).

Participants were then escorted to a separate sound-attenuated room where four mylar bands were placed in the tetrapolar configuration for impedance cardiograph recordings according to published guidelines (Sherwood et al., 1990). An occluding cuff of appropriate size was placed on the upper left arm. Individuals were seated in a comfortable chair and instructed to relax for the next 12 minutes while resting measures of cardiovascular function were obtained. During the final 5 minutes of the resting assessment, cardiovascular assessments of SBP and DBP were obtained once every 90 seconds, whereas impedance cardiograph readings were recorded continuously. Participants also completed a state anxiety scale at the end of the rest period as a baseline measure of state anxiety.

Following the resting assessments, participants performed a speech and mental arithmetic protocol developed by Cacioppo and colleagues (1995). The order of the stressors was counterbalanced and all verbal instructions were standardized. For the speech task, participants were asked to formulate a speech about their reactions to the following scenario: They were shoplifting a belt. The participants were asked to prepare a 3-minute speech in their defense that included the following points: (a) tell their side of the story; (b) tell what the security guard did wrong; (c) tell why the security guard may have suspected them of shoplifting; (d) explain how they can prove that they did not steal the belt; (e) explain what should happen to the guard for his/her mistake; and (f) provide a summary of their points. Any questions from participants about the upcoming task were answered prior to the preparation period. In order to increase the relevance of the task for participants, they were told that it would be corrected by the experimenter with an answer sheet and to resume the serial subtraction from the corrected number. Any questions about the subtraction tasks were answered prior to the start of Minute 1. After Minute 1, each subsequent problem was adjusted for difficulty so that effort was relatively constant across participants (i.e., approximately 10 serial subtractions per minute; see Cacioppo et al., 1995). In order to increase the relevance of the task, participants were encouraged to work as quickly and as accurately as possible because their responses would be recorded and compared with the performance of others in the study. Impedance cardiography measures were recorded continuously throughout the task, whereas blood pressure was assessed during Minutes 1, 3, and 5 of the task. Performance during the task was measured as the percent of incorrect serial subtractions. At the end of the math task, participants again completed the state anxiety measure. Upon completion of both psychological stressors, participants were compensated, debriefed, and thanked for their participation.

Measures

Cardiovascular measures.—A Minnesota Impedance Cardiograph Model 3048B (IFM Inc., Greenwich, CT) was used to measure the electrocardiogram (ECG), basal thoracic impedance (Z₀), and the first derivative of the impedance signal (dZ/dt). Four mylar bands were placed in the tetrapolar configuration according to published guidelines (Sherwood et al., 1990). A 4 mA AC current at 100 kHz was passed through the two outer bands, and Z₀ and dZ/dt were recorded from the two inner bands. The ECG, Z₀, and dZ/dt signals were digitized at 500 Hz. The impedance data were ensemble averaged within 1-minute epochs. Ensemble averaging uses the R-point of the ECG as a reference for the successive averaging of the ECG and subsequent dZ/dt signals. This procedure reinforces both signals while randomly occurring movement artifacts and lower frequency respiratory influences are effectively filtered out (Kelsey & Guethlein, 1990). Each ensemble-averaged waveform was verified or edited prior to analyses by a trained scorer.

Stroke volume was estimated using the Kubicek equation (see Sherwood et al., 1990) and the subsequent cardiac output in liters/minute was calculated by multiplying heart rate × (stroke volume/1000). Total peripheral resistance was measured in resistance units (dynes-second · cm⁻⁵) based on mean arterial pressure and cardiac output (i.e., total peripheral resistance = mean arterial pressure/cardiac output × 80).

The systolic time intervals consisted of pre-ejection period, left-ventricular ejection time, and electromechanical systole. Pre-ejection period was calculated as the time interval in ms between the Q-point of the ECG and the B-point of the dZ/dt signal, whereas left-ventricular ejection time was calculated as the time interval in ms between the B-point and X-point of the dZ/dt signal. Electromechanical systole was assessed as the time interval in ms between the Q-point and X-point (i.e., pre-ejection period + left-ventricular ejection time). These minute by minute impedance-derived measures were averaged across minutes within each epoch (e.g., baseline, task) to increase the reliability of these assessments (Kamarck et al., 1992).

Respiratory sinus arrhythmia was calculated based on the
digitized interbeat intervals that were checked and edited for artifacts using the detection algorithm of Berntson, Quigley, Jang, and Boysen (1990). A heart period time series was created from the interbeat interval series using a “weighted” beat algorithm (Berntson, Cacioppo, & Quigley, 1995). Sharp transitions in the heart period time series (e.g., due to arrhythmia) were detected, using the aforementioned algorithm (Berntson et al., 1990), and removed by smoothing. A linear (first order) polynomial was fit to, and subtracted from, the heart period time series (Litvack, Oberlander, Carney, & Saul, 1995). Subtraction of this first order polynomial (linear detrending) acts effectively as a high pass filter, removing very large ultra-low frequency trends (including the DC component) from the input signal. After linear detrending, the heart period time series was band-pass filtered from .12 to .40 Hz using an interpolated finite impulse response filter (Neuvo, Cheng-Yu, & Mitra, 1984). The power spectrum of the heart period time series was calculated using a Fast Fourier Transform and scaled to ms²/Hz. Respiratory sinus arrhythmia was calculated as the natural log of the area under the heart period power spectrum within the corner frequencies of the band pass filter (see Litvack et al., 1995). Respiratory sinus arrhythmia was calculated on a minute by minute basis and aggregated across minutes within each epoch to increase reliability.

A Dinamap Model 8100 monitor (Critikon Corp., Tampa, FL) was used to measure SBP and DBP. The Dinamap used the oscillometric method to estimate blood pressure (see Epstein, Huffnagle, & Bartkowski, 1991; Gorbach, Quill, & Lavine, 1991 for validation studies). Blood pressure assessments were obtained via a properly sized occluding cuff positioned on the upper left arm of the participant according to the manufacturer’s specifications. Mean SBP and DBP for each epoch were averaged across minutes to increase the reliability of these assessments.

State anxiety.—A short form of the Spielberger State-Trait Anxiety Scale was administered to participants at the end of the resting baseline and following completion of each of the psychological stressors (Marteau & Bekker, 1992). Participants were asked to rate their current feelings on a scale of 1 (not at all) to 4 (very much). Consistent with prior work, the internal consistency of the scale in our study was high (Chronbach’s alpha of .78 to .80).

**RESULTS**

**Preliminary Analyses**

Repeated measures analyses of variance (Epoch: baseline, speech task, math task) were conducted on state anxiety scores and the cardiovascular measures to examine the efficacy of the stress protocol. Missing data on some measures occurred for several participants due to experimenter or computer error (i.e., 1 to 4 participants depending on the measure). Participants with missing data were dropped from the analyses and the degrees of freedom were adjusted accordingly. Results revealed that the stressors led to increases in state anxiety, $F(2,260) = 11.89, p < .001$, Huynh-Feldt epsilon = 1.00.

The psychosocial stress protocol was designed so that the speech and math tasks elicited comparable and significant changes in cardiovascular function (Cacioppo et al., 1995). As shown in Table 2, the psychological stressors were associated with significant increases in SBP, $F(2,256) = 351.95, p < .001$, Huynh-Feldt epsilon = .93; DBP, $F(2,256) = 211.52, p < .001$, Huynh-Feldt epsilon = .95; cardiac output, $F(2,256) = 20.65, p < .001$, Huynh-Feldt epsilon = .91; and heart rate, $F(2,258) = 197.33, p < .001$, Huynh-Feldt epsilon = .85. In addition, pre-ejection period, $F(2,256) = 45.62, p < .001$, Huynh-Feldt epsilon = .85, and electromechanical systole, $F(2,256) = 6.62, p < .01$, Huynh-Feldt epsilon = .93, were shortened, and respiratory sinus arrhythmia decreased, $F(2,254) = 9.54, p < .001$, Huynh-Feldt epsilon = .75, in response to the stress protocol.

Ancillary analyses including task order as a between-participants factor revealed that the only significant effects for task order on reactivity were for SBP, $F(2,256) = 9.33, p < .001$, Huynh-Feldt epsilon = .93, pre-ejection period, $F(2,256) = 4.25, p < .03$, Huynh-Feldt epsilon = .83, and left-ventricular ejection time, $F(2,256) = 4.44, p < .02$, Huynh-Feldt epsilon = .90. For all of these task order effects, reactivity was slightly higher to the first, in comparison to the second task. However, none of the tests of significance reported in all subsequent main analyses involving gender and age, and the gender $\times$ age interactions below were changed when controlling statistically for task order.

In prior research, concerns have been raised about the psychometric properties of reactivity assessments (Kamarck et al., 1992). Due to the comparability of cardiovascular responses to the two stressors, we aggregated cardiac responses across tasks

## Table 2. Mean (SD) Baseline and Changes in Cardiovascular Reactivity During Speech and Math Tasks

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline</th>
<th>Speech Task</th>
<th>Math Task</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)**</td>
<td>116.92 (12.32)</td>
<td>15.44 (7.90)</td>
<td>15.42 (8.61)</td>
</tr>
<tr>
<td>DBP (mmHg)**</td>
<td>68.70 (9.19)</td>
<td>8.31 (5.73)</td>
<td>8.84 (5.97)</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>59.72 (25.29)</td>
<td>-2.11 (13.20)</td>
<td>-0.42 (13.60)</td>
</tr>
<tr>
<td>Cardiac output (l/min)**</td>
<td>3.98 (1.64)</td>
<td>0.38 (1.02)</td>
<td>0.55 (1.14)</td>
</tr>
<tr>
<td>Total peripheral resistance (Dynes-s·cm⁻⁵)</td>
<td>2188.71 (1242.47)</td>
<td>-48.07 (847.53)</td>
<td>-70.24 (868.83)</td>
</tr>
<tr>
<td>Heart rate (BPM)**</td>
<td>67.97 (11.46)</td>
<td>9.04 (6.47)</td>
<td>9.37 (7.03)</td>
</tr>
<tr>
<td>Pre-ejection period (ms)**</td>
<td>104.63 (17.10)</td>
<td>-8.09 (11.26)</td>
<td>-7.43 (12.53)</td>
</tr>
<tr>
<td>Left-ventricular ejection time (ms)</td>
<td>293.65 (25.35)</td>
<td>2.82 (19.47)</td>
<td>2.46 (19.64)</td>
</tr>
<tr>
<td>Electromechanical systole (ms)**</td>
<td>398.28 (30.93)</td>
<td>-5.26 (19.90)</td>
<td>-4.97 (19.36)</td>
</tr>
<tr>
<td>Respiratory sinus arrhythmia (natural log)**</td>
<td>5.81 (1.19)</td>
<td>-0.28 (0.81)</td>
<td>-0.26 (1.02)</td>
</tr>
</tbody>
</table>

*Note: For Epoch main effect, *p < .05; **p < .01; ***p < .001.
in order to increase the measurement reliability and generalizability of our reactivity protocol (also see Jennings et al., 1997; Kamarck et al., 1992; Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). Of course, this procedure also assumes adequate intertask correlations. For our cardiovascular measures, the correlation between reactivity during the math and speech tasks was high and ranged from $r = .63$ to $r = .88$, with an average intertask correlation of $r = .74$. Therefore, all subsequent analyses of reactivity data were performed on averaged task changes from baseline using regression procedures that allow for the analysis of continuous predictor variables (i.e., age).

Age and Gender Differences in Resting Cardiovascular Function

We first attempted to replicate prior research on age and gender differences in resting cardiovascular function. Regression analyses were performed to examine the independent prediction of age and gender, and the statistical interaction between age and gender on resting cardiovascular function. As suggested by Aiken and West (1991), the main effects of age and gender were centered and entered into the first step of the regression equation. The Age $\times$ Gender cross-product term was entered in the second step of the regression equation. Body mass index was also entered in the first step of the regression equation to account for its potential influence on resting cardiovascular function.

Consistent with prior research, older adults had higher resting levels of SBP ($B = .42$, $t = 5.40, p < .001$) and DBP ($B = .19$, $t = 2.89, p < .01$) than their younger counterparts. Age was also associated with lower resting levels of stroke volume ($B = -9.5$, $t = 5.98, p < .001$) and the resulting cardiac output ($B = -0.06$, $t = 5.54, p < .001$). Resistance to blood flow was increased as a function of age as indexed by total peripheral resistance ($B = 40.05$, $t = 4.89, p < .001$). These data suggest that the age-related differences in resting blood pressure were primarily due to an increase in total peripheral resistance because decreases in cardiac output might be expected to lower blood pressure. Consistent with this suggestion, controlling statistically for resting total peripheral resistance ($B = 12$, $t = 0.08$), but not resting cardiac output ($B = 15$, $p = .03$), rendered the age-related increase in DBP nonsignificant.

No age-related differences were found in resting heart rate and pre-ejection period. However, age predicted increases in left-ventricular ejection time ($B = .83$, $t = 4.55, p < .001$) and electromechanical systole ($B = .77$, $t = 3.27, p < .01$). Consistent with prior work, resting levels of respiratory sinus arrhythmia were reduced in older individuals ($B = -0.05$, $t = 6.24$, $p < .001$), suggesting lower basal parasympathetic control of the heart.

In terms of gender main effects, men had higher resting DBP ($B = -4.80$, $t = 3.35, p < .01$) and lower resting heart rate ($B = 4.55$, $t = 2.33, p < .001$) than women. The only significant interaction involving age and gender was for resting SBP ($B = .41$, $t = 2.73, p < .01$). We examined this statistical interaction by computing predicted SBP levels based on scores one standard deviation above and below the mean for age in men and women (see Aiken & West, 1991). Inspection of these predicted values revealed that although both men and women showed an age-related increase in resting SBP, women had a greater age-related increase due primarily to the lower SBP in younger women (predicted SBP = 108.63) compared to younger men (predicted SBP = 115.19).

In summary, results of these initial analyses replicated prior research on age and gender differences in resting cardiovascular function. Older individuals at rest had higher blood pressure, lower stroke volume and the resulting cardiac output, greater total peripheral resistance, increases in the systolic time intervals, and lower respiratory sinus arrhythmia than their younger counterparts. These age-related differences were largely comparable in men and women. We next examined our primary research questions regarding potential age and gender differences in cardiovascular reactivity during acute stress.

Age and Gender Differences in Cardiovascular Reactivity to Psychosocial Stress

Regression analyses were again performed to examine the independent prediction of age and gender, and the statistical interaction between age and gender on cardiovascular reactivity to psychosocial stress. In all analyses, baseline measures of cardiovascular function and body mass index were included in the first step of the regression equation (along with the age and gender main effects) to account for their potential influences on cardiovascular reactivity.

Replicating prior work, older individuals had greater SBP reactivity ($B = .22$, $t = 3.55, p < .001$) but not DBP reactivity to psychosocial stress. In addition, age was associated with greater stress-induced increases in stroke volume ($B = .32$, $t = 3.18, p < .01$) and the resulting cardiac output ($B = .03$, $t = 3.04, p < .01$). Older individuals also showed an increase in total peripheral resistance reactivity to stress that was marginally significant ($B = 10.48$, $t = 1.89, p = .06$). Therefore, the age-related increase in SBP reactivity appeared to be due to stress-induced changes in both cardiac output and total peripheral resistance. Consistent with this position, only when controlling statistically for both cardiac output and total peripheral resistance reactivity was the age-related increase in SBP reactivity rendered nonsignificant ($B = .06$, $p = .36$).

Stress-induced cardiovascular sympathetic control, as indexed by a shortening of pre-ejection period, was increased in older compared with younger individuals ($B = - .29$, $t = 3.51$, $p < .001$). Older adults also had a greater lengthening of left-ventricular ejection time ($B = .52$, $t = 3.59, p < .001$) but no difference in electromechanical systole compared with younger adults. Thus, the above age-related increase in cardiac output reactivity during stress appeared to be due to the combined influence of greater stress-induced sympathetic cardiac activation and a lengthening of left-ventricular ejection time in older individuals. However, controlling statistically for pre-ejection period ($B = .01$, $p = .11$) but not left-ventricular ejection time ($B = .02$, $p = .02$) changes during stress rendered the age-related increase in cardiac output reactivity nonsignificant.

In comparison to prior research, we found an age-related increase in parasympathetic withdrawal ($B = -.02$, $t = 3.38, p < .01$) during stress as indexed by respiratory sinus arrhythmia. In addition, no age-related differences were found in heart rate reactivity. None of the Age $\times$ Gender interactions were significant $(p > .10)$ on any measure of cardiovascular reactivity during stress. These data suggest that the pattern of age-related differences in reactivity to stress were comparable for men and women.
There were several possible alternative explanations for these age-related differences in cardiovascular reactivity to stress that warranted further exploration. One possibility was that these results were due to greater task-specific affective responses in older adults, perhaps due to age-related differences in task performance. Preliminary regression analyses examining potential age and gender effects on state anxiety changes during stress and indices of task performance only revealed a gender main effect on state anxiety \((B = .28, t = 3.34, p < .01)\). In these analyses, women reported greater changes in state anxiety compared to men. To examine the direct implications of these analyses, we repeated the regression procedures reported above while controlling statistically for changes in state anxiety, the percentage of incorrect math problems, and the number of speech prompts. Importantly, none of our conclusions were altered in these reanalyses (i.e., no effects that were significant were rendered nonsignificant and no nonsignificant results became significant). It was also possible that existing age-related differences in demographic factors might be responsible for the results obtained in this study. However, controlling statistically for the demographic factors of education level and household income did not alter any of our conclusions reported above. Finally, these results were also unchanged while controlling statistically for self-reported parental history of hypertension.

**DISCUSSION**

Age-related differences in resting cardiovascular function are well documented and confer increased vulnerability to physical health problems. However, much less is known about age-related differences in cardiovascular reactivity during stress. Results of this study suggest that older individuals had a greater stress-induced change in SBP reactivity that was due to changes in both cardiac output and total peripheral resistance. In addition, sympathetic activation and parasympathetic withdrawal during stress as indexed by pre-ejection period and respiratory sinus arrhythmia, respectively, were greater in older compared with younger adults. These age-related differences in cardiac sympathetic control appeared to account for the age-related increase in cardiac output reactivity. Furthermore, these age-associated results were comparable for men and women. We would like to emphasize that although we found no evidence for gender differences, this is the only impendence study that we are aware of that has directly contrasted age-related differences in men and women. Given the myriad of factors that may lead to null results, additional studies will be necessary to address this issue definitively.

The results of this study were generally consistent with data from Jennings and colleagues (1997) suggesting age-related differences in cardiac and vascular changes during stress. As noted earlier, the small sample size, single reactivity task, and/or short task period utilized by Boutcher and Stocker (1996) may have decreased their statistical power to detect these age-related differences. Kamarck and colleagues (1992) demonstrated that the use of aggregation across multiple minutes and stressors increased the reliability and generalizability of reactivity assessments. Future research adopting this approach would be helpful to minimize potential differences in measurement reliability that might obscure comparisons across studies.

There were several differences between our findings and those of Jennings and colleagues (1977) that warrant discussion. First, we only found a marginally significant effect for age on increased vascular responses to stress. There has been much work to document the reliability of cardiovascular reactivity assessments (Kamarck et al., 1992; Manuck, 1994). Most of these studies suggest that heart rate, SBP, pre-ejection period, and cardiac output are measured with good reliability following aggregation across tasks. However, total peripheral resistance reactivity is typically characterized by relatively lower reliability, probably because the formula for calculating total peripheral resistance combines the measurement error in estimating both cardiac output and mean arterial pressure. Although our sample size appeared adequate for most of our measures, the greater measurement error in total peripheral resistance scores may have reduced our power to detect this age-related reactivity difference at the conventional level of statistical significance.

In contrast, it is more difficult to explain why Boutcher and Stocker (1996) found that older individuals had a decrease in total peripheral resistance reactivity compared with younger individuals. One important issue may be that these authors did not control statistically for baseline levels of total peripheral resistance. Our analyses revealed that resting total peripheral resistance was significantly and negatively related to stress-induced total peripheral resistance changes. The direction of this regression weight would imply that individuals with greater resting total peripheral resistance would show less of an increase in total peripheral resistance reactivity unless these baseline differences were accounted for in the analyses. Thus, it is possible that the higher resting total peripheral resistance observed in older individuals may have biased the simple change scores used by these authors. Jennings and colleagues (1997) controlled statistically for baseline levels of total peripheral resistance and reported findings similar to our study.

We also found that age was associated with greater parasympathetic withdrawal during stress as indexed by respiratory sinus arrhythmia. Consistent with this finding, Ferrari, Daffonchio, Gerosa, and Mancia (1991) reported that the vagal response to graded electrical stimulation was greater in older than in younger rats. However, this finding has not been reported in the two prior human studies. It is important to note that we utilized a larger age range than Jennings and colleagues (1977). To examine the potential significance of this difference, we conducted ancillary analyses in which we constrained our analyses to individuals between the ages of 46 and 70. Age no longer predicted respiratory sinus arrhythmia changes during stress in these analyses \((r = -.11, p = .39)\) suggesting the potential importance of examining such changes across a wide spectrum of ages (see Lakatta, 1993).

Age-related differences in cardiovascular reactivity during psychosocial stress may be due to structural and/or neural-receptor changes in the cardiovascular system with chronological age (Fleg, 1986; Lakatta, 1993). For instance, the increased vascular resistance with age may augment blood pressure responses to stress (Fleg, 1986). Although our measures do not allow us to precisely examine this issue, at least some of these differences in reactivity appear to be due to neural processes as evidenced by age-related differences in pre-ejection period and respiratory sinus arrhythmia. A shortening of pre-ejection period during stress appears to be a reliable index of cardiac sympathetic control, whereas a decrease in respiratory sinus...
arrhythmia appears to be a sensitive index of cardiac parasympathetic withdrawal (Berntson et al., 1994; Cacioppo et al., 1994).

There are several limitations of the present study that need to be discussed. Due to the homogeneous ethnic composition of our sample and its relatively high education level, future research is needed to establish the generality of these age-related differences in cardiovascular reactivity. It is important to note, however, that we were able to replicate much of the prior research on age-related differences in resting cardiovascular function. This gives us some confidence in the generalizability of our reactivity findings. Nevertheless, it is possible that a broader range of measurement on socioeconomic status–related indices could reveal evidence for gender effects in these age-associated cardiovascular differences.

Another potential limitation of this study is related to our use of self-reported information to screen for a relatively healthy population. We cannot be certain that these individuals are free of underlying disease. However, it is noteworthy that we found results similar to Jennings and colleagues (1997), who rigorously screened their participants and found that age-associated effects on cardiovascular reactivity were minimally influenced by underlying disease. The comparability of our age-related differences in baseline cardiovascular assessments with prior studies that screened for underlying disease is also consistent with the notion that we utilized a relatively healthy population. However, the use of self-report to screen for hypertension may be particularly problematic because hypertension tends to be symptomless. To further investigate this issue we examined our baseline blood pressure assessments to see if anyone in the study would be categorized as hypertensive (i.e., SBP ≥ 140 mm/Hg or DBP ≥ 90 mm/Hg). This is a procedure that would be biased against us because the novelty associated with being in a psychophysiological laboratory could elevate blood pressure acutely. Inspection of the data revealed that only six individuals had SBP over 140 mm/Hg (none had DBP over the hypertensive limit). All of these individuals would be categorized as stage 1 (mild) hypertension if it indeed reflected their “true” resting blood pressure (SBP range = 141 mm/Hg to 151 mm/Hg). As a result, we repeated our analyses deleting these participants. None of our conclusions on age and gender effects for cardiovascular reactivity would be changed as a result of deleting these six participants.

We also utilized a cross-sectional design, therefore whether our data would translate to age-related changes cannot be determined. Well-designed longitudinal studies will be necessary to evaluate this question. We should also note that the direct health consequences of impedance–derived measures of cardiovascular reactivity remain to be determined. However, preliminary data suggest that cardiovascular reactivity during psychological stress, as measured by blood pressure or heart rate, may influence the development of cardiovascular disorders (Manuck, 1994). Furthermore, increased cardiovascular reactivity during stress may have implications for individuals with existing cardiovascular disorders. For example, Krantz and colleagues (1991) found that higher SBP reactivity was associated with greater myocardial ischemia during mental stress in coronary artery disease patients. We also found evidence for an age-related increase in parasympathetic withdrawal during stress. In prior research, relatively low levels of parasympathetic activity appear to be an important mechanism predicting adverse cardiovascular outcomes (Bigger, Kleiger, & Fleiss, 1988; Binkley, Nunziata, Haas, Nelson, & Cody, 1991). Future research should help clarify the direct health relevance of these age-associated differences in cardiovascular reactivity during psychological stress.

In conclusion, the greater reactivity seen in older adults appears to be consistent with the reactivity hypothesis and may help explain part of the increased cardiovascular disease risk with age. It is important to note, however, that the finding of age-related differences in cardiovascular reactivity does not necessarily imply that these changes occur invariably with chronological age. Chronological age is superimposed on genetic (e.g., familial risk), social (e.g., social support), psychological (e.g., perceptions of stress), and behavioral (e.g., diet) factors that may in turn influence physiological function and disease risk (e.g., Baum, Newman, Weinman, West, & McManus, 1997). Data on whether psychosocial processes predict age-associated biological differences are sparse (Baltes & Baltes, 1990; Rowe & Kahn, 1987; Uchino, Kiecolt-Glaser, & Cacioppo, 1992). There are existing developmental models that may provide promising frameworks for examining these issues. For example, Baltes (1997) and Schulz and Hechausen (1996) argue that successful adaptation during the life span may involve the selective use of primary and secondary control strategies that produce a positive profile of gain and losses. Consistent with the importance of control-related processes, preliminary evidence from the MacArthur Successful Aging Studies revealed that intraindividual variability in perceived control predicted greater mortality 5 years later (Eizenman, Nesselroade, Featherman, & Rowe, 1997). The integration of life span developmental perspectives with basic biological theories of aging represents a complex but critically important theoretical challenge for both literatures.

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