A Walking Program’s Attenuation of Cardiovascular Reactivity in Older Adults With Silent Myocardial Ischemia

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Silent myocardial ischemia (SI) has been linked to increased risk of future coronary events. Enhanced systolic and diastolic blood pressure (SBP and DBP, respectively) and heart-rate (HR) reactions to stress (cardiovascular reactivity [CVR]) have been associated with greater severity of SI and are related prospectively to coronary-artery-disease endpoints. The authors examined the potential attenuating effects of 6 months of walking (aerobic exercise) versus control on CVR to three laboratory stressors in 25 older adults with exercise-induced SI. Maximal aerobic capacity was significantly improved by 12% for the exercise group and decreased by 8% for controls ($p < .001$). Groups had similar biomedical profiles pre- and postintervention. Walkers had significantly reduced DBP reactivity (pre, 12 ± 2; post, 4 ± 2 mm Hg) compared with controls (pre, 10 ± 2; post, 11 ± 2 mm Hg; $p = .05$), but no differences between groups were found for SBP or HR reactivity. These findings are the first to suggest that increased physical activity (via walking) can attenuate BP reactivity to emotional stressors in apparently healthy older adults with SI.

Key Words: aerobic exercise, emotional stress, blood pressure, heart rate

Transient myocardial ischemia is a symptom of coronary artery disease (CAD). Myocardial ischemia not accompanied by angina is labeled “silent” myocardial ischemia (SI; Cohn, 1985). Previous findings demonstrate that otherwise healthy middle-aged and older individuals without a history of symptomatic CAD but who manifest asymptomatic exercise-induced ischemia are at a two- to four-fold increased risk for subsequent cardiac events (Fleg et al., 1990; Katznel, Sorkin, & Goldberg, 1999).

In patients with symptomatic CAD, mental or emotional stress has been found to provoke episodes of symptomatic ischemia and SI (Rozanski et al., 1988). Mental-stress-induced SI typically occurs at lower rate-pressure product than exercise-induced ischemia does (Blumenthal et al., 1995), and several studies have demonstrated that such SI is associated with future risk for cardiac events (Sheps et al., 1989).
Coronary vasoconstriction coincident with an increase in oxygen demand during stress has been implicated as a potential pathophysiologic mechanism mediating the relation of mental stress to myocardial ischemia (Blumenthal et al., 1990; Treiber et al., 2003). For instance, studies have shown coronary vasoconstriction (Kop, Krantz, et al., 2001; Lacy et al., 1995) and acetylcholine-related vagal withdrawal during mental stress (Kop, Verdino, et al., 2001). In addition, severity of myocardial ischemia has been positively associated with blood-pressure (BP) and heart-rate (HR) responses to mental stress (Blumenthal et al., 1995; Krantz et al., 1991). Enhanced BP and HR responses (cardiovascular reactivity [CVR]) to mental or emotional stress are prospectively related to hypertension, atherosclerosis, and CAD endpoints (Jennings et al., 2004; Kamarck et al., 1997; Rozanski, Blumenthal, & Kaplan, 1999).

Benefits of exercise on CVR have been shown in some studies (Blumenthal et al., 1990; Crews & Landers, 1987; Georgiades et al., 2000; Sherwood, Light, & Blumenthal, 1989; Stein & Bouchcher, 1992) but not in others (Albright, King, Taylor, & Haskell, 1992; De Geus, van Doornen, & Orlbeke, 1993). This research demonstrates that aerobic exercise has variable effects on CVR (depending on the length, intensity, and type of exercise; sample characteristics; and type of mental stress) and suggests that aerobic training attenuates CVR to anger-related stressors in particular. The American College of Sports Medicine (ACSM, 1993) and others (Hagberg, Park, & Brown, 2000; King, Haskell, Young, Oka, & Stefanick, 1995; King, Rejeski, & Buchner, 1998; Pescatello et al., 2004; Pollock et al., 1991; Young & King, 1995) prescribe training sedentary older adults in a low- to moderate-intensity (rather than a vigorous) exercise program such as walking for 20–60 min 3–5 days/week. Such a walking program is related to lower risk of exercise-induced cardiac events and injuries and to health benefits (ACSM; Hagberg et al.; King et al., 1995, 1998; Pollock et al.; Young & King). Walking is more easily adopted, increased, and maintained than other types of exercise (King, 2001; Wilcox & Storandt, 1996), and it is unknown whether more vigorous exercise programs have additive health benefits (ACSM; Young, 2000).

In the present study, to our knowledge we are the first to test the hypothesis that a 6-month walking program would attenuate CVR to anger-provoking stressors in sedentary older adults who manifest exercise-induced SI and have no history of overt CAD.

**Methods**

**Participants**

Twenty-five participants (diagnosed with SI as part of ongoing studies at the University of Maryland, Baltimore) agreed to participate in the present exercise-intervention study. The study was approved by the university’s institutional review board, and all patients provided written consent. Older (age 56–83 years), nonsmoking men (n = 17) and women (n = 8) with at least a high school education (68% having bachelor’s or professional degrees) and without a history of symptomatic CAD were recruited from the Baltimore–Washington metropolitan area and the Charlestown Retirement Community for participation in exercise-intervention studies. These individuals were selected because they were at increased risk for future coronary events but were free of the potential confounders (i.e., physiological
effects of drug therapy for cardiac symptoms, symptomatic myocardial ischemia, prior myocardial infarction) found in prior studies investigating the relation between mental-stress-induced CVR and SI (Krantz et al., 1991).

At the initial screening visit, a medical history was obtained, a physical examination was performed, and fasting blood chemistries were drawn. A resting ECG was also collected. Exclusionary criteria included a history of overt CAD, resting-ECG evidence of CAD as defined by significant Q waves, major ST-segment abnormalities (Deanfield et al., 1983), left-bundle-branch block, complex arrhythmias, poorly controlled hypertension (blood pressure >180/105 mm Hg), stroke, peripheral arterial disease, poorly controlled hyperlipidemia (low-density lipid concentrations >190 mg/dl or plasma triglycerides >400 mg/dl), diabetes mellitus, dementia (Mini-Mental State Examination score <24), history of psychiatric disorders, current smoking, history of heavy alcohol consumption (>14 drinks per week), or other comorbid diseases that would interfere with the ability to participate in the study. Participants were also excluded if they had severe hypertension requiring multiple antihypertensive medications (3+) because of potential safety concerns of temporarily discontinuing their medications during the measurements of CVR.

Procedures

**Exercise-Treadmill Screening.** A screening exercise-treadmill test to voluntary exhaustion was performed according to the Bruce protocol (Bruce & Horstein, 1969). These tests were performed with the hypertensive participants on their routine hypertension medications. A 12-lead ECG recorded every minute during exercise and every other minute for 7 min into recovery. Respiration rate, minute ventilation, O2 consumption, and CO2 production were measured every 20 s during the treadmill test by indirect calorimetry using a metabolic measurement cart (model 2900, Sensormedics, Inc., Anaheim, CA) as previously described (Katzel et al., 1994). The O2 consumption was determined by averaging the final two 20-s oxygen-uptake values. The exercise ECG was interpreted according to the Minnesota code criteria (Prineas, Crow, & Blackburn, 1982). A positive ECG response for ischemia was defined as horizontal or down-sloping ST-segment depression for ≥1 mm for at least 0.08 s after the J point. Lesser degrees of ST-segment depression were interpreted as negative.

On a subsequent visit, an exercise-treadmill test with measurement of maximal aerobic capacity (VO2max) was performed using a Bruce (Bruce & Horstein, 1969) or modified Balke protocol as previously described (Katzel, Sorkin, & Fleg, 2001) and in accordance with American College of Cardiology and American Heart Association guidelines for exercise testing (Gibbons et al., 1997). The VO2max tests fulfilled at least two of the following three criteria: (a) HR at maximal exercise was >95% of the age-adjusted maximal heart rate (220 – age), (b) respiratory-exchange ratio was >1.10, and (c) a plateau in oxygen uptake was achieved on the basis of a change in VO2 of <0.2 L/min during the final two oxygen collections.

Individuals with abnormal exercise-treadmill tests, as determined by a board-certified internist and a board-certified cardiologist, were advised to see their private physicians for additional cardiac evaluations, with the recommendation that exercise thallium scans be performed. Five participants subsequently had positive cardiac catheterizations for CAD, and 2 others had reversible ischemia on exercise TL scintigraphy and were not enrolled in the study. The 25 participants included...
in the study, 17 men and 8 women age 69 ± 8 years (M ± SD), were identified with exercise-induced SI (Minnesota code) on at least two separate exercise tests. These 25 participants were randomized to 6 months of aerobic-exercise intervention (n = 14) or 6 months of wait-list control (n = 11) as they entered the study (staggered enrollment). After completion of postcontrol testing, control participants were given the opportunity to engage in the exercise-training program (see Figure 1 for random assignment and participant-retention details). Four participants dropped out of the exercise-training protocol because of time conflicts, and 2 participants experienced adverse health events not associated with study participation but precluding them from further participation. During the 6-month waiting period, 1 participant from the control group experienced an adverse health event not associated with study participation but precluding the individual from further participation in the study, and 2 participants refused to return for postcontrol assessment. After postcontrol testing, 4 wait-list control participants decided to engage in the exercise-intervention condition for 6 months and then underwent assessments again after their aerobic exercise training.

**Cardiovascular-Reactivity Session.** Participants on antihypertensive medications were asked to refrain from using these medications for at least 5 days before testing following a weeklong, medically supervised tapering. This tapering period was prescribed by a board-certified internist according to the half-lives of the antihypertensive medications used by the participants. With the participant situated in a supine position and after a 6-min rest/adaptation period, a baseline 12-lead ECG

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**Figure 1** — Participant retention across time and random assignment.
was obtained continuously, and four resting measurements of systolic BP (SBP),
diastolic BP (DBP), and HR were collected every minute. All BP and HR data
were collected oscillometrically at 60-s intervals during rest and task periods with
an automated vital-signs monitor (Dinamap Model # 1846SX, Critikon, Tampa,
FL). Participants then performed the following 3-min tasks, each followed by a
10-min recovery period: (a) personally relevant anger-recall task, in which par-
ticipants were asked to recall and speak about an event that occurred within the
preceding year that made them feel angry, irritated, upset, or frustrated (Neumann
& Waldstein, 2001); (b) standardized role-play scenario designed to elicit anger
and hostility by asking the participant to discuss the inadequate care of someone
to whom the participant is close (e.g., spouse, parent, or sibling) by a hostile and
difficult nursing-home administrator (role played by the experimenter; Waldstein,
Burns, Toth, & Poehlman, 1999); and (c) mental arithmetic consisting of standard-
ized subtraction of serial 7s with minor levels of harassment (i.e., participants were
firmly urged to complete the calculations quickly and accurately; Waldstein et al.).
Tasks were presented in fixed order. HR and ECG were monitored continuously. BP
and HR readings were collected during the last 4 min of the baseline and recovery
periods. CVR for each task was assessed by standard procedures (Llabre, Spitzer,
Saab, Ironson, & Schneiderman, 1991) via creating delta scores (task mean-initial
baseline mean) for SBP, DBP, and HR responses. Participants underwent CVR
testing at baseline (Time 1) and postintervention (Time 2).

**Questionnaire.** Spielberger’s State-Trait Personality Inventory (Spielberger,
1979) was administered immediately after each task period at baseline (Time 1)
and postintervention (Time 2). The state-anger and state-anxiety subscales were
used to assess affective responses to the tasks. Each subscale consisted of 10 items
rated on a 4-point Likert scale (1 = not at all to 4 = very much so). The internal
consistency of these subscales administered under conditions of mental stress has
ranged between .84 and .94, and validity has been supported by several studies
(Spielberger).

**Measurement of Lipoprotein Lipids and Glucose Levels.** After a 12- to 14-hr
overnight fast, blood samples were deposited into chilled 1-mg/ml EDTA tubes.
Plasma triglycerides and cholesterol were assessed enzymatically as described
previously (Freidewald, Levy, & Fredrickson, 1972). Because plasma triglyceride
levels did not exceed 400 mg/dl, high-density lipoprotein cholesterol was measured
in the supernatant after precipitation of the apolipoprotein B-containing lipoproteins
with dextran sulfate. The low-density lipids were calculated using the Friedewald
equation (Freidewald et al.). The fasting glucose levels were determined by the
-glucose oxidase method (Beckman glucose analyzer, Fullerton, CA).

**Exercise Intervention (Walking Program).** The goal of the exercise training
was to increase the participants’ VO\(_{2\text{max}}\) by 10% or more, without changing their
weight. All training sessions were supervised by exercise physiologists who were
qualified to perform CPR. Participants exercised by walking on a treadmill in
supervised sessions three times per week for 6 months. The duration and intensity
of sessions were gradually increased until participants were walking for 40 min at
70% of their HR reserve or at their ischemic threshold as determined during their
exercise-treadmill test (Katzel et al., 1994, 1999). Exercisers had to attend >75% of
the exercise sessions in order to complete the study successfully. Participants
who were assigned to the control condition were given the opportunity to engage in the exercise-training program after completing the wait-list control condition for 6 months and undergo postprogram testing. The wait-list controls received no further contact for 6 months before postcontrol testing.

Data Analyses

Data from men and women were pooled for all analyses (see Figure 1 for participant-retention rates), and outlier-detection procedures were performed. Pretreatment (Time 1) sample and biomedical characteristics of the two treatment groups (control and exercise groups) were compared using analysis of variance (ANOVA) for continuous variables and chi-square for categorical measures (i.e., education, gender, and race). Two-way repeated-measures ANOVAs (Treatment Group × Task) were performed to evaluate potential differences between treatment groups on state-anger and state-anxiety reactivity to the tasks and on CVR to the stressors at Time 1. Intention-to-treat analyses were then performed. The effect of the exercise intervention on fitness level (as measured by VO\textsubscript{2max}), body-mass index (BMI; kg/m\textsuperscript{2}), resting BP and HR, total cholesterol, low- and high-density lipoprotein cholesterol, triglycerides, fasting glucose levels, state-anger and state-anxiety reactivity, and aggregate CVR delta scores was examined between exercise and control groups from pre- to posttreatment using a two-way (Treatment Group × Time) repeated-measures ANCOVA, statistically controlling for age differences at baseline. It is important to note that the CVR delta scores were collapsed across the tasks separately for Time 1 and Time 2 for the latter analyses. Creating an aggregate delta score for each time point has been recommended for prospective studies to provide reliable and stable measures of CVR over time (Kamarck & Lovallo, 2003). Similarly, for affect responses to the tasks, we collapsed the measures for state anxiety and state anger across the tasks for each time point (Time 1 and Time 2). Statistical significance was held at a p value of .05.

Results

Pretreatment (Time 1) Analyses

At Time 1, the two groups were of comparable gender, race, and education level. The groups did not differ in BMI, VO\textsubscript{2max}, total cholesterol, low- and high-density lipoprotein cholesterol, triglycerides, fasting glucose and insulin levels, and resting BP and HR. The control group was, however, significantly younger than the exercise group, 63 ± 2 years versus 71 ± 2 years, F(1, 23) = 8.2, p < .01. The baseline VO\textsubscript{2max} of 23.5 (ml·kg\textsuperscript{-1}·min\textsuperscript{-1}) is typical of values seen in an older, sedentary population. Baseline biomedical and state-affect characteristics for the exercise and controls are shown in Table 1. Significant increases in SBP, DBP, and HR responses from baseline to task were noted across the three tasks, SBP, F(1, 23) = 110.2, p < .0001; DBP, F(1, 23) = 87.6, p < .0001; HR, F(1, 22) = 57.8, p < .0001 (see Figure 2). No significant differences were noted between the treatment conditions on CVR Time 1. Two-way (Treatment Group × Task) repeated-measures ANOVA analyses were also computed on self-reported state anxiety and anger measures during the tasks. There were no significant differences found between the control and exercise groups on state-affect measures at Time 1 (see Table 1).
Table 1 Pre- (Time 1) and Posttreatment (Time 2) Descriptives ($M \pm SE$)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Precontrol ($n = 11$)</th>
<th>Preexercise ($n = 14$)</th>
<th>Postcontrol ($n = 8$)</th>
<th>Postexercise ($n = 12$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)*</td>
<td>$63 \pm 2$</td>
<td>$71 \pm 2$</td>
<td>$28 \pm 1$</td>
<td>$29 \pm 1$</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>$27 \pm 1$</td>
<td>$27 \pm 1$</td>
<td>$28 \pm 1$</td>
<td>$29 \pm 1$</td>
</tr>
<tr>
<td>VO₂max (ml · kg⁻¹ · min⁻¹)*</td>
<td>$23.3 \pm 1.4$</td>
<td>$23.6 \pm 1.4$</td>
<td>$23.6 \pm 1.4$</td>
<td>$25.3 \pm 0.9$</td>
</tr>
<tr>
<td>Resting SBP (mm Hg)</td>
<td>$140 \pm 6$</td>
<td>$134 \pm 3$</td>
<td>$130 \pm 4$</td>
<td>$128 \pm 4$</td>
</tr>
<tr>
<td>Resting DBP (mm Hg)</td>
<td>$80 \pm 2$</td>
<td>$76 \pm 3$</td>
<td>$72 \pm 1$</td>
<td>$72 \pm 2$</td>
</tr>
<tr>
<td>Resting HR (beats/min)</td>
<td>$71 \pm 3$</td>
<td>$71 \pm 3$</td>
<td>$75 \pm 4$</td>
<td>$71 \pm 3$</td>
</tr>
<tr>
<td>State anxiety (Likert scale: 1 = not at all to 4 = very much so)</td>
<td>$23 \pm 2$</td>
<td>$23 \pm 2$</td>
<td>$23 \pm 2$</td>
<td>$21 \pm 2$</td>
</tr>
<tr>
<td>State anger (Likert scale: 1 = not at all to 4 = very much so)</td>
<td>$18 \pm 2$</td>
<td>$19 \pm 2$</td>
<td>$18 \pm 2$</td>
<td>$18 \pm 2$</td>
</tr>
<tr>
<td>Fasting glucose (mg/dl)</td>
<td>$95.2 \pm 5.1$</td>
<td>$96.1 \pm 2.1$</td>
<td>$102.3 \pm 7.9$</td>
<td>$96.7 \pm 1.8$</td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>$192.1 \pm 11.3$</td>
<td>$189.4 \pm 8.0$</td>
<td>$183.6 \pm 7.9$</td>
<td>$189.9 \pm 8.2$</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>$117.6 \pm 8.4$</td>
<td>$119.8 \pm 7.7$</td>
<td>$104.2 \pm 11.0$</td>
<td>$122.2 \pm 5.8$</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>$50.1 \pm 6.1$</td>
<td>$47.3 \pm 3.8$</td>
<td>$43.4 \pm 6.3$</td>
<td>$43.5 \pm 5.2$</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>$118.3 \pm 21.6$</td>
<td>$115.6 \pm 15.0$</td>
<td>$148.8 \pm 23.6$</td>
<td>$122.4 \pm 17.8$</td>
</tr>
</tbody>
</table>

Note. BMI = body-mass index; VO₂max = maximum volume of oxygen consumption; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; LDL = low-density lipoproteins; HDL = high-density lipoproteins.

*p < .009.
Neumann et al.  

Intention-to-Treat Pre- (Time 1) to Posttreatment (Time 2) Analyses

With respect to the effects of treadmill-exercise training on fitness level, two-way (Treatment Group × Time) repeated-measures ANCOVA analyses performed on VO_{2max} showed significant increases of 12% (Time 1, 23.1 ± 1.0, vs. Time 2, 25.3 ± 1.0 ml · kg⁻¹ · min⁻¹) for the exercise group and decreases of 8% (Time 1, 25.6 ± 1.2, vs. Time 2, 23.6 ± 1.2 ml · kg⁻¹ · min⁻¹) for the control group, \( F(1, 18) = 14.05, p < .001 \) (see Table 1). No significant differences were noted between the exercise and control groups from baseline to posttreatment (Treatment Group × Time) on BMI, resting BP, resting HR, total cholesterol, low- and high-density lipoprotein cholesterol, triglycerides, or fasting glucose levels. Two-way (Treatment Group × Time) repeated-measures ANCOVA analyses performed on SBP, DBP, and HR reactivity (controlling for age) showed a significant interaction effect of Treatment Group × Time on DBP reactivity, \( F(1, 17) = 4.28, p = .05 \) (see Figure 3). Specifically, reductions in DBP reactivity were noted for the exercise group after aerobic conditioning (Time 1, 12 ± 2 mm Hg, vs. Time 2, 4 ± 2 mm Hg) relative to the control group (Time 1, 11 ± 2 mm Hg, vs. Time 2, 10 ± 2 mm Hg). No other significant main or interaction effects were noted with respect to the other CVR measures; the means for both SBP and HR reactivity at Time 2 were lower for both groups. With respect to self-reported state anxiety and anger ratings experienced during the tasks, Treatment Group × Time repeated-measures ANOVA analyses did not yield significant differences between the treatment groups or Treatment Group × Time interaction effects.

**Figure 2** — Baseline (rest) to task period aggregate cardiovascular-reactivity responses at Time 1. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate. *\( p < .0001 \).
Discussion

The tendency to show increased CVR to mental stress might promote CAD and increase the risk of cardiac events (Jennings et al., 2004; Kamarck et al., 1997; Treiber et al., 2003). In this novel study, to our knowledge we are the first to examine the hypothesis that increased aerobic physical activity (i.e., 6 months of walking three times per week) would attenuate CVR to mental stressors in a laboratory setting in older adults with exercise-induced SI and free of overt CAD symptoms. As hypothesized, significant improvements in physical fitness after the exercise intervention without changes in body mass in individuals with SI were associated with significant reductions in DBP reactivity compared with the controls with SI. There was, however, no significant difference in the SBP and HR reactivity to the stressors after exercise training in comparison with controls. Consistent with prior studies in our laboratory, the walking intervention, absent a change in weight, did not significantly influence fasting lipoprotein lipid, glucose, or resting BP and HR (Katzel et al., 1995). Indeed, the ACSM concluded recently that only modest 3- to 4-mm-Hg reductions in resting BP occur after chronic exercise training (Pescatello et al., 2004). The absence of significant differences between groups on the SBP and HR reactivity and resting BP is likely a result, at least in part, of our attenuated sample size and thus insufficient power to detect significant differences between groups. In addition, the lack of significant differences between the groups on SBP and HR reactivity might be partly caused by habituation to the stressors—these values declined in both groups at Time 2; state anger and anxiety responses to the tasks were similar, however, after the intervention.

Figure 3 — Treatment group by time on cardiovascular-reactivity means. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate. *p = .05.
To our knowledge, only two previous studies examined a similar hypothesis, but in patients with CAD (Blumenthal et al., 1997, 2005). One aim of these studies was to evaluate whether ischemia induced by either exercise or mental stress can be reduced by exercise training. BP and HR changes after the exercise intervention were, however, not reported for possible comparison with the present study’s results. Therefore, our findings can only be compared with those of previous research examining the effects of exercise on CVR to mental stress in samples without apparent exercise-induced SI. Partially consistent with the present study’s findings, men with Type A personality and without a history of myocardial ischemia in an aerobic-exercise-training group experienced a greater reduction in DBP and HR reactivity to mental stress than the men with Type A personality in a strength-training group (Blumenthal et al., 1990). Further analysis indicated that the men with borderline hypertension who underwent aerobic training had greater reductions in CVR than the normotensive men with aerobic training (Sherwood et al., 1989). In another study (Rogers, Probst, Gruber, Berger, & Boone, 1996), borderline hypertensive participants who underwent 8 weeks of low-intensity exercise training (40–50% VO$_{2\text{max}}$) displayed attenuated SBP and DBP responses to mental stress. In contrast to the present study, Georgiades and colleagues (2000) compared the effect of aerobic exercise versus wait-list control on resting/persistent BP levels and CVR in overweight individuals with high-normal or unmedicated Stage 1 or 2 hypertension. After 6 months, participants in the intervention group had lower SBP and HR reactivity during laboratory stressors than the controls, and not DBP reactivity as was found in the present study. In another study, Stein and Boutcher (1992) also reported that in sedentary middle-aged men, an 8-week-long aerobic-exercise-training program reduced resting HR and the HR response to mental stressors but did not lower BP responses. In addition, 6 months of aerobic-exercise training did not lower BP and HR responses to mental arithmetic in Type A individuals or in healthy middle-aged men and women (Albright et al., 1992).

Paradoxically, De Geus and colleagues (1993) found that high cardiovascular fitness was associated with high CVR and that 8 months of aerobic-exercise training had little effect on CVR and psychological questionnaires on stress and personality. There are several possible reasons for the discrepancies between this previous work and the present findings. First, several previous studies only included Type A individuals or overweight individuals versus a more heterogeneous sample that is more reflective of the general population, as done in the present study. Second, previous studies examined younger to middle-aged adults versus older adults as in the present study. Third, studying individuals with exercise-induced SI with a history of overt, symptomatic CAD as done in previous studies versus investigating apparently healthy individuals with exercise-induced SI might produce different results. Collectively, the present study’s findings taken together with those of this prior literature demonstrate that the effects of aerobic-exercise training on CVR are variable; aerobic exercise might be more likely to produce attenuated CVR to certain types of mental stress (e.g., anger provocation, Stroop, or mental arithmetic) in particular types of individuals (e.g., hypertensives and individuals with Type A behavior patterns).

There are several strengths and limitations with respect to the present study. The main strength of our study was that we chose to study patients with SI who had no prior history of overt CAD as opposed to patients with symptomatic angina.
Studies in our laboratory and elsewhere demonstrate that SI is common in the elderly (Katzel et al., 1994), and as many as 30% of apparently healthy men above the age of 80 have SI (Hedblad et al., 1989). SI increases the risk of subsequent myocardial infarction and sudden cardiac death by two to four times, yet it is rarely diagnosed and treated. Therefore, interventions that decrease cardiac morbidity and mortality in older patients with SI should have significant health benefits. In addition, patients with symptomatic CAD are typically on multiple anti-ischemic medications or have arrhythmias that confound measurements of cardiovascular responses. By contrast, the vast majority of the SI patients in our study were taking vasoactive medications or were on monodrug therapy for mild systolic hypertension. Unlike many patients with overt CAD, our SI participants had not suffered cardiac events and thus should have had intact left-ventricular function and should not have been centrally limited in their ability to exercise.

Despite the strengths of the present study, there are also limitations to our findings. First, although our tasks were designed to reflect the types of angry situations one might encounter in daily life, our results might not generalize to out-of-the-laboratory experiences. It would be beneficial to investigate ambulatory BP and HR changes to real-life stressors in comparison with inducing stress in the laboratory in future studies involving older adults with SI. Second, the sample size is rather small, limiting our ability to assess sex-by-aerobic-training interaction effects on CVR. The absence of accounting for potential moderating effects related to sex might have confounded the results in that either women or men alone could be driving the differences between the groups. It is important to note that on follow-up analysis on the previous possibility, no apparent sex-by-treatment-condition effects on CVR were found. Third, our participants were selected based on the presence or absence of ST-segment depression during maximal exercise. We advised individuals with abnormal exercise-treadmill tests to see their private physicians for additional cardiac evaluations. Nonetheless, additional cardiac evaluations such as exercise thallium scintigraphy were not uniformly used to confirm the presence or absence of ischemia. Hence, some of the patients possibly had “false positive” treadmill tests.

Even so, our findings suggest that walking regularly (approximately 40 min, 3 days/week) for at least 6 months resulted in attenuated DBP responses to the emotional stressors. Contrary to our hypothesis, the exercise intervention did not reduce the SBP- and HR-reactivity responses to the laboratory presentation of stressors. It is important to note, however, that because of our limited power the absence of findings for SBP and HR reactivity might also be a result of Type II error. In addition, it is possible that training programs of longer duration (e.g., 1 year rather than 6 months) would result in more robust changes in health status and CVR to stress.

Although it was not assessed here, we speculate that improved CVR via exercise training might help reduce pathophysiological processes associated with CAD events, possibly by enhancing vagal function and coronary vasomotion (Cohn, Fox, & Daly, 2003; Kop, Krantz, et al., 2001; Kop, Verdino, et al., 2001). Future work should include measures of epicardial vasomotion, flow velocity, and vagal activity to aid in understanding the mechanisms underlying CVR to mental stress in patients with SI after exercise training. Additional studies with larger samples of patients with SI are required, however, to determine whether the reduction in
CVR associated with aerobic exercise in this population indicates decreased risk for ischemic events.

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