Arterial Baroreflex Control of Cardiac Vagal Outflow in Older Individuals Can Be Enhanced by Aerobic Exercise Training

Gaelle Deley, Glen Picard, J. Andrew Taylor

Abstract—Maintained cardiac vagal function is critical to cardiovascular health in human aging. Aerobic exercise training has been considered an attractive intervention to increase cardiovagal baroreflex function; however, the data are equivocal. Moreover, if regular exercise does reverse the age-related decline in cardiovagal baroreflex function, it is unknown how this might be achieved. Therefore, we assessed the effects of a 6-month aerobic training program on baroreflex gain and its mechanical and neural components in older individuals (5 women and 7 men, aged 55 to 71 years). We assessed baroreflex function using pharmacological pressure changes (bolus nitroprusside followed by bolus phentolamine) and estimated the integrated gain (∆R-R interval/∆ systolic blood pressure) and mechanical (∆ diameter/∆ pressure) and neural (∆R-R interval/∆ diameter) components via measurements of carotid artery diameter in previously sedentary older individuals before and after 6 months of aerobic training. There was a significant 26% increase in baroreflex gain that was directly related to the amount of exercise performed and that was derived mainly from an increase in the neural component of the arterial baroreflex (P<0.05). We did find changes in the mechanical component, but unlike integrated gain and the neural component, these were not related to the magnitude of the exercise stimulus. These results suggest that exercise training can have a powerful effect on cardiovagal baroreflex function, but a sufficient stimulus is necessary to produce the effect. Moreover, adaptations in the afferent-efferent baroreflex control of cardiac vagal outflow may be crucial for the improvement in arterial baroreflex function in older humans. (Hypertension. 2009;53:826-832.)

Key Words: baroreflex ■ aging ■ exercise ■ nervous system ■ autonomic ■ carotid arteries

Maintained cardiac vagal function is critical to cardiovascular health in human aging. Although this may be doubly true for those suffering from age-related cardiovascular complications (patients with coronary disease or hypertension), in elderly individuals, even after accounting for all other risk factors, reduced vagal control of the heart predicts greater cardiac and cerebrovascular mortality.1 Perhaps the most critical control mechanism for cardiac vagal outflow is the arterial baroreflex, because it is equipped to exquisitely regulate cardiovascular autonomic activity in response to the moment-by-moment demands of arterial pressure regulation.2 Indeed, the progressive reduction in cardiovagal baroreflex sensitivity that characterizes human aging directly relates to increased cardiovascular morbidity and mortality.3,4 This reduction in reflex control of vagal outflow could derive, in full5 or at least in part,6,7 from decreased distensibility of barosensory vessels. This would effectively blunt the ability of stretch-sensitive, baroreflexive afferent nerves to respond to pressure changes. The potential effect on baroreflex control may also contribute to the relationship of increased arterial stiffness with increased cardiovascular risk.8,9

Given the cardioprotective effects of vagal outflow and the importance of the arterial baroreflex in determining its level, various strategies have been pursued to increase cardiovagal baroreflex function. Among these, aerobic exercise training has been thought to be an attractive intervention, because cross-sectional studies suggest that age-related declines in baroreflex function may be prevented by habitual physical activity.10–12 Indeed, research suggests that older individuals with a lifetime history of regular aerobic exercise have cardiovagal baroreflex function greater than their sedentary peers5,13 and even comparable to sedentary young individuals.7 However, it remains unclear whether this maintained function results primarily from lesser vascular stiffening of barosensory vessels.5,13 Our previous work suggests that greater neural control of cardiac vagal outflow may be the prepotent effector of maintained function in older athletes.7 However, these differences may simply reflect cohort effects; results from longitudinal, intervention studies on the effects of aerobic exercise training do not unequivocally support the benefits of exercise. Some data do suggest that baroreflex...
indices based on spectral analysis might be improved by exercise training. However, a baroreflex gain derived by simple observation of a spontaneously occurring heart period and arterial pressure oscillations can be suspect, and increases may be an epiphenomenon of greater heart rate variability concomitant with a training-related bradycardia. Direct probes of cardiovascular baroreflex control suggest that sensitivity may be increased in some percentage of older volunteers, may not change, or may even decrease after an exercise training program. The wide variety of methods used to assess the cardiovascular baroreflex (Valsalva's maneuver, neck suction and neck pressure, and phenylephrine bolus) and differences in the age of the subjects (from 47 to 63 years old) and in the training duration (from 3 to 6 months) may explain some of this inconsistency.

Thus, it remains unclear whether regular physical activity can reverse the age-related decline in cardiovascular baroreflex function and through what mechanism regular exercise might counterbalance this decline. The present study assessed the effects of a 6-month aerobic training program on baroreflex gain and on its neural and mechanical components. Based on the results obtained by Hunt et al., we hypothesized that regular exercise training would increase baroreflex gain primarily by increasing neural aspects of cardiac vagal baroreflex control.

### Methods

#### Subjects

Twelve volunteers (5 women and 7 men; ages 55 to 71 years; mean age: 59 ± 5 years) were screened and selected according to the following criteria: (1) no clinical signs or symptoms of heart disease, hypertension, diabetes mellitus, neurologic disease, or cancer; (2) normal resting ECG; (3) no recent weight change; (4) no regular use of tobacco; (5) body weight within 15% of ideal as assessed by body mass index; (6) no history of cardiac complications; (7) regular exercise <1 hour per week; and (8) aerobic capacity <80th percentile for age and gender. The study was approved by the institutional review board at Spaulding Rehabilitation Hospital, and all of the subjects gave written, informed consent.

#### Protocol and Measurements

All of the subjects were familiarized with the laboratory setting on a day before the study day. After initial screening, subjects reported to the laboratory for 2 experimental sessions. Subjects were requested not to engage in vigorous exercise for the previous 48 hours and not to consume either alcohol- or caffeine-containing beverages for 24 hours before being studied. The experimental sessions were performed again after 6 months of aerobic exercise training.

During the first study session, body fat was assessed by 7-site skinfold thickness via a Harpenden caliper. Subjects then assumed the supine position for a 10-minute measurement of resting heart rate and blood pressure. Subjects were then instrumented and tested for maximal aerobic capacity. Online computer-assisted open circuit spirometry was used to determine O2 consumption during a modified Balke protocol; subjects walked or jogged on a treadmill while the grade was increased every 2 minutes until volitional exhaustion was achieved. Throughout, a 12-lead ECG was monitored. Attainment of maximal aerobic capacity was determined by meeting the Karvonen Method: maximal heart rate = resting heart rate + \{(desired intensity) × resting heart rate\} for 20 to 30 minutes, and all of the sessions were directly supervised. Volunteers were also encouraged to exercise on their own 1 day per week at this same intensity and duration. Over the 24-week period, exercise frequency gradually increased to 5 days, intensity to 70% to 80%, and duration to 40 minutes. For all of the exercise sessions, subjects wore a heart rate monitor (Polar S610i) programmed at a recording rate of 15-s intervals and completed an exercise log to track intensity, frequency, and duration. Over the course of training, volunteers became sufficiently independent to exercise on their own and, by the final 2 months, returned once every 2 weeks to download training data from heart rate monitors and to turn in exercise logs. The training impulse was used to quantify the exertional volume and intensity for each subject and was calculated using the recorded heart rate data for each individual exercise session.

This parameter has been used as an integrative marker of the exercise load, and it reflects exercise intensity (heart rate), as well as frequency and duration.

#### Data Analysis

Time series of systolic blood pressure and R-R interval were derived using signal processing software (Powerlab, ADInstruments). Systolic blood pressure was determined from the maximum of the pressure waveform and R-R intervals from the time difference between successive R waves. Systolic carotid artery diameters were obtained via image analysis software developed in our laboratory. Respiratory sinus arrhythmia was quantified from power spectral analysis of each 300-s R-R interval time series during paced breathing. The time series was interpolated to 4 Hz; based on the Welch algorithm, 7 overlapping periodograms were averaged to produce the spectrum estimate for the entire time series. Power within the respiratory frequency band, defined as 0.2 to 0.3 Hz, was averaged to estimate respiratory sinus arrhythmia. Baroreflex function was estimated from the relation of systolic pressure to RR interval, as described previously. We assessed data from the pressure rise, because it represents baroreflex afferent activation of cardiac vagal outflow. Analysis began at the lowest pressure value after the bolus injection of phenylephrine and ended when pressure peaked. This selection of data points often encompasses threshold and saturation regions of the sigmoid relationship. To make the analysis objective and, therefore, independent of investigator bias, we analyzed the data via a piecewise linear regression that required >5 data points to define the presence of threshold and/or saturation (if any). To gain further insight into baroreflex function, the mechanical and neural components of the baroreflex were calculated. The mechanical transduction of pressure was assessed from the relation of carotid systolic diameter to systolic arterial pressure, and the neural transduction of stretch was assessed from the relation of R-R interval to carotid systolic diameter. The linear portion of these relationships was also extracted with the application of the piecewise test. It must be noted that baroreflex gain and its components were determined independent of each other, which means that threshold and saturation regions were excluded from a given relation without considering where these regions lie in the
other 2 relations. Cook’s distance values were produced for each linear model as a check to remove outliers. Linear relations can have a range of precision as indicated by their SE; therefore, to account for differential precision and provide the most robust estimates, a weighted mean of the 2 trials was calculated for all of the linear relations using the inverse of the SE as the weight.

**Statistics**

A paired t test was performed to examine the effects of training. A simple regression was used to determine the relation between the average weekly exercise (training impulse) and the effects of training on baroreflex function. Differences were considered significant at \( P<0.05 \). Data are presented as mean±SEM.

**Results**

Figure 1 shows the average weekly exercise performed by each subject during the 6 months of training. Based on the training impulse method, 8 subjects averaged the minimum or more of the prescribed exercise, whereas 4 subjects tended to do less exercise than the minimum prescribed. This was not related to subject age or gender.

**Aerobic Capacity, Body Composition, and Resting Hemodynamics**

Across all of the subjects, including those who tended to perform less exercise, maximal aerobic capacity increased on average by 13% after the training program (Table; \( P<0.05 \)), and both weight and body mass index decreased (Table; \( P<0.05 \)).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Before Training (n=12), Mean (SE)</th>
<th>After Training (n=12), Mean (SE)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( VO_2 ) max, mL/min per kg</td>
<td>28.5 (1.6)</td>
<td>31.6 (1.7)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>77.6 (3.5)</td>
<td>74.9 (2.9)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>26.6 (0.6)</td>
<td>25.7 (0.5)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>24.5 (1.2)</td>
<td>22.6 (1.5)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Waist:hip ratio</td>
<td>0.85 (0.03)</td>
<td>0.84 (0.02)</td>
<td>0.2</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>60.4 (1.7)</td>
<td>56.3 (1.5)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>R-R interval, ms</td>
<td>977.4 (29.2)</td>
<td>1102.1 (44.1)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>RSA, ms²</td>
<td>216.7 (56.2)</td>
<td>359.6 (72.0)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Systolic pressure, mm Hg</td>
<td>122.5 (4.3)</td>
<td>115.3 (3.6)</td>
<td>0.05</td>
</tr>
<tr>
<td>Diastolic pressure, mm Hg</td>
<td>69.9 (2.6)</td>
<td>68.4 (2.5)</td>
<td>0.3</td>
</tr>
</tbody>
</table>

RSA indicates respiratory sinus arrhythmia; \( VO_2 \) max, maximal oxygen uptake.

\( r^2 = 0.07 \); \( P=0.21 \), age

\( r^2 = 0.41 \); \( P<0.05 \). Although exercise training tended to increase the mechanical component, this increase did not relate to the average weekly exercise performed (\( r^2 = 0.02 \); \( P=0.28 \)). In contrast, similar to integrated gain, the neural component was linearly related to the amount of exercise performed over the 6 months of training (\( r^2 = 0.42 \); \( P<0.05 \)). Regression analysis also showed that the responses to training did not relate to baseline baroreflex sensitivity (\( r^2 = 0.07 \); \( P=0.21 \), age

**Exercise Induced Changes in Arterial Baroreflex Function**

Figure 2. Schematic representation of the average baroreflex-mediated responses to phenylephrine before and after 6 months of aerobic exercise training. Data from each trial from each individual subject were interpolated to provide data across the entire observed pressure ranges without gaps between pressures so that each point represents the mean of all 12 subjects. Threshold and/or saturation were excluded before the averaging to display only the linear range. Note the shift in slope upward (longer average resting R-R intervals) and to the left (reduced average systolic pressure) with an increase in overall gain.

\( P<0.05 \). The weight loss could be attributed to a decrease in the percentage of body fat (\( P<0.05 \)). Resting heart rate and systolic pressure were also decreased after training (\( P<0.05 \)), whereas diastolic pressure remained unchanged. Respiratory sinus arrhythmia was also significantly increased after exercise training (66%; \( P<0.05 \)).

**Baroreflex Function**

Six months of exercise training induced, on average, a 26% increase in baroreflex gain (from 10.6±1.7 to 12.9±2.6 ms/mm Hg; \( P=0.05 \); Figure 2). In addition, exercise training induced a change in the mechanical component that did not quite reach significance (0.012±0.003 versus 0.019±0.005 mm/mm Hg; \( P=0.06 \)) and a significant increase in the neural component that averaged 23% (from 811.8±88.3 to 979.6±137.3 ms/mm; \( P<0.05 \); Figure 3). Because the borderline increase in the mechanical component of the arterial baroreflex might reflect a reduction in pressure, systolic pressure was explored as a predictor of this change. However, analysis of covariance showed that systolic pressure did not explain changes in the mechanical component.

Figure 4 shows the relations between the average weekly exercise performed and the changes in baroreflex gain and its components. The increase in integrated gain was linearly related to the magnitude of the exercise stimulus (\( r^2 = 0.41 \); \( P<0.05 \)). Although exercise training tended to increase the mechanical component, this increase did not relate to the average weekly exercise performed (\( r^2 = 0.02 \); \( P=0.28 \)). In contrast, similar to integrated gain, the neural component was linearly related to the amount of exercise performed over the 6 months of training (\( r^2 = 0.42 \); \( P<0.05 \)). Regression analysis also showed that the responses to training did not relate to baseline baroreflex sensitivity (\( r^2 = 0.07 \); \( P=0.21 \), age

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There are reports that reductions in body fat can result in increased baroreflex gain; therefore, we used body fat as a covariate to determine whether it might be responsible, in part, for the increase in baroreflex function. A multiple linear regression model showed that decreases in body fat did not relate to increases in either integrated baroreflex gain or its neural component. We also explored whether the resting bradycardia observed after exercise training was related to the changes in arterial baroreflex function. However, there were no significant linear relations between the increased resting R-R interval and the improvements in arterial baroreflex function. Although respiratory sinus arrhythmia also increased, this response was not correlated with the amount of exercise performed by the subjects ($r^2=0.01; P=0.96$).

**Discussion**

We found that regular aerobic exercise can increase cardiovagal baroreflex function in previously sedentary, older healthy individuals. This increase was directly related to the magnitude of the exercise stimulus and appeared to derive mainly from an increase in the neural component of the arterial baroreflex. We did find changes in the mechanical component of the reflex, but, unlike integrated gain and the neural component, these changes did not relate to the magnitude of exercise performed. This may indicate that exercise training does alter barosensory vessel distensibility but not in a way that is proportional to either the amount of exercise performed or to the improvements in cardiovagal baroreflex gain. These are the first data that demonstrate the effect of exercise training on the particular neural and mechanical aspects of baroreflex function and the importance of a sufficient exercise stimulus to produce this effect in older humans.

Only a very few studies have explored the potential of aerobic exercise training to improve cardiovagal baroreflex control, and these have not resulted in consistent effects. Our results suggest that exercise duration, intensity, and frequency are important factors that must be considered. For example, exercise programs <6 months and with lower intensities and frequencies than we used have not found any improvement in cardiovagal baroreflex sensitivity. On the other hand, despite a short duration of exercise training (3 months), Monahan et al reported an average 25% increase in the Valsalva response in previously sedentary older individuals. This might suggest that baroreflex gain can increase early in the duration of exercise training; however Valsalva’s maneuver is a heterogenous stimulus, unloading cardiopulmonary baroreceptors, as well as differentially unloading aortic and carotid baroreceptors. One study reported that 11 months of aerobic training did not alter heart rate responses to phenylephrine-induced increases in arterial pressure. How-

Figure 3. The mechanical (top) and neural (bottom) components of baroreflex-mediated responses to phenylephrine before and after 6 months of aerobic exercise training. The data represent the changes in all of the variables across the linear range for each relation, interpolated to avoid inconsistent gaps so that each point represents the mean of all 12 subjects.

$\Delta$ systolic carotid diameter

$\Delta$ systolic pressure

POST-EXERCISE avg. slope = 0.019 ± 0.005 mm/mm Hg

PRE-EXERCISE avg. slope = 0.012 ± 0.003 mm/mm Hg

Baroreflex Mechanical Component

Figure 4. Relations between the average weekly training (percentage of the minimum prescribed) and the changes in the arterial baroreflex: integrated gain, the mechanical component, and the neural component.

Average weekly exercise, % of minimum prescribed

Integrated gain, msec/mm Hg

Mechanical component, mm/mm Hg

Neural component, msec/mm

POST-EXERCISE avg. slope = 980 ± 137 msec/mm

PRE-EXERCISE avg. slope = 812 ± 88 msec/mm

$\Delta$ R-R interval

$\Delta$ systolic carotid diameter

POST-EXERCISE avg. slope = 0.019 ± 0.005 mm/mm Hg

PRE-EXERCISE avg. slope = 0.012 ± 0.003 mm/mm Hg

Baroreflex Neural Component

$\Delta$ Integrated gain, msec/mm Hg

$r^2 = 0.42$  $P < 0.05$

$r^2 = 0.41$  $P < 0.05$

$r^2 = 0.02$  $P = 0.28$

$r^2 = 0.02$  $P < 0.05$

$r^2 = 0.01$  $P = 0.96$
ever, the use of heart rate instead of R-R interval to assess cardiovagal baroreflex gain may have masked the effects of training. Cardiac vagal outflow is most closely related to R-R interval changes, and when these previous data are examined as a function of R-R interval, exercise appears to augment cardiovagal baroreflex gain.

Increased baroreflex gain with regular aerobic exercise could derive from increased carotid distensibility that increases vessel responsiveness to pressure changes and/or neural adaptations that improve transduction of barosensory stretch into vagal outflow. Thus, we investigated the effects of exercise training on not only integrated baroreflex gain but also on its mechanical and neural components. One cross-sectional study of older master athletes suggested that cardiovagal baroreflex gain is preserved in these individuals solely because of lesser stiffening of barosensory vessels. However, our own previous work in masters athletes suggests that greater neural control of cardiac vagal outflow is at least as important in maintaining baroreflex function, if not more important. Indeed, the current longitudinal study found an increase in integrated baroreflex gain that was most closely related to increases in the neural component of the reflex. Although there was a change in the mechanical component of the reflex, it was highly variable and did not demonstrate an increase that was proportional to the exercise stimulus. It should be noted that our measurement of the relationship between systolic pressure rises and systolic carotid diameter increases is not carotid distensibility, per se. Rather, this measure reflects vascular mechanics during the pressure changes that characterize baroreflex engagement. Although arterial vascular stiffness or distensibility may relate to cardiovagal baroreflex gain, we previously found no obvious relation between basal pulsatile stiffness and mechanical pressure transduction in young untrained, older untrained, and older, physically active individuals.

In our current study, we found that there were increases in the vessel mechanics with exercise training, but they were highly variable. For example, the mean and median percentage changes for both the integrated gain and neural component were within 2%, but the median change for the mechanical component was 25% lower than the mean change. In addition, there was only 1 case wherein the increase in integrated baroreflex gain could be attributed solely to an increase in the mechanical component, but in every other case, indeed, across the group as a whole, the change in integrated baroreflex gain tended to track the change in the neural component ($r^2=0.33$; $P<0.05$). It may be that the exercise training load to increase the mechanical component of the reflex is lower than that for the neural component and, therefore, occurred earlier in the training program. If so, it would appear that these adaptations do not continue with the increasing load over time or may occur at different rates based on individual vessel properties.

It is of note that every subject but 1 increased aerobic capacity, and this subject demonstrated no training-induced bradycardia or increase in cardiovagal baroreflex gain. However, in general, neither the increase in aerobic power nor the decrease in resting heart rate bore a direct relation to the change in baroreflex function. There are data suggesting that training programs can reduce resting heart rate and increase maximal aerobic capacity without having an effect on cardiovagal baroreflex sensitivity. This may suggest that, like the mechanical component of the baroreflex, the resting bradycardia and the increase in maximum oxygen consumption occur earlier during exercise training than improvements in baroreflex sensitivity.

Our results suggest that, with a sufficient exercise stimulus, the change in integrated baroreflex gain most closely relates to neural improvements in cardiovascular autonomic control. The estimated neural transduction component of the baroreflex encompasses the baroreceptors themselves, their afferent activity, central integration of this activity, generation of efferent parasympathetic outflow, and, finally, the sinoatrial node responsiveness to vagal activity. It is feasible that any one site or a combination of sites might account for the neural adaptations that we found. For example, in rats with heart failure, exercise training significantly enhances baroreceptive afferent sensitivity. However, from this work, it is unclear whether this might arise from the baroreceptor endings themselves or from changes in the distensibility of the carotid sinuses and/or aortic arch. In addition, it has been suggested that exercise training may alter central neural regulation of blood pressure through the plasticity of GABAergic systems in the hypothalamus. This might reverse age-related alterations in central autonomic integration or reductions in tonic vagal outflow. It is also feasible that exercise mitigates the age-related decline in cardiac muscarinic receptor density. However, some of our previous work suggests that age-related declines in cardiac muscarinic receptor function are not impacted by regular aerobic activity. Although the exact site of adaptation is not certain, our observations clearly show that exercise training has marked effects to improve cardiac vagal neural control in older individuals.

**Perspectives**

The resting bradycardia observed in our subjects reflects one of the hallmarks of exercise training and is generally thought to derive from increased cardiac vagal outflow. However, we did not find a relation between increases in R-R interval or respiratory sinus arrhythmia and the greater sensitivity of the arterial baroreflex after exercise training. This might suggest that other mechanisms could be responsible for the bradycardia. Decreased sympathetic outflow and/or decreased intrinsic heart rate are 2 possible mediators of the observed bradycardia. Decreased cardiac responsiveness to catecholamine infusion has been demonstrated in middle-aged active men suggesting that lesser cardiac sympathetic tone may result from exercise training. Pharmacological blockade suggests that the resting bradycardia observed in young endurance athletes can be explained by a lower intrinsic heart rate. However, to our knowledge, this has never been investigated in older active individuals. Moreover, it may simply be that a nonlinear relation exists between the vagal changes that mediate increased baroreflex function and those that mediate increased resting R-R interval and its variability.
The current work is the first to clearly delineate the nature of the baroreflex adaptations in response to exercise training in older individuals. However, we found that only with a sufficient exercise stimulus can cardiac vagal neural control be significantly enhanced. These findings suggest that regular physical activity can be cardioprotective via improvements in cardiac vagal control and may thereby decrease cardiovascular risks associated with aging.

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Disclosures
None.

References


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