Effect of Aerobic Exercise Training on Aortic Wave Velocity in Obese Subjects: A Report of Five Cases

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Summary

Background: Aerobic exercise training clearly improves a number of traditional cardiovascular risk factors in populations at elevated risk, such as the obese. The purpose of this pilot investigation was to examine the effect of an aerobic exercise training program on arterial compliance in obese individuals.

Case Report: Five subjects [1 male/4 female, mean age 43.8 (±9.7) years] participated in this study. Each subject underwent a 10-week training program (20-40 minute aerobic training sessions 3-5 times per week). Aortic wave velocity (AWV), a metric of vessel stiffness, was assessed in the thoracic aorta using a magnetic resonance imaging technique. Non-parametric statistics (Wilcoxon Signed Ranks and Friedman Test) were used to assess the impact of aerobic exercise training on maximal oxygen consumption (VO₂Max), oxygen consumption at ventilatory threshold (VO₂ at VT), body mass index (BMI), and AWV. Improvements in VO₂Max [24.3 (±6.0) vs. 26.0 (±5.9) ml·kg⁻¹·min⁻¹], VO₂ at VT [15.0 (±3.2) vs. 17.9 (±2.6) ml·kg⁻¹·min⁻¹] and BMI [41.1 (±5.2) vs. 39.4 (±5.8) kg/m²] were all significant following training (p<0.05). AWV prior to, at the midpoint and following the completion of aerobic exercise training was 9.9 (±1.2), 7.6 (±1.8) and 6.2 (±1.4) m/s, respectively. The difference between pre and post training AWV measurements was significant (p=0.007).

Conclusions: The results of the present pilot study indicate that aerobic exercise training significantly improves arterial compliance in obesity. The positive impact of aerobic exercise training on cardiovascular risk in obese individuals may therefore partially be the result of improved arterial compliance.

Key words: aerobic exercise • arterial stiffness • obesity


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References: 24

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BACKGROUND

Obesity is a significant healthcare burden that increases risk for the development of cardiovascular disease (CVD) and independently contributes to thousands of premature deaths each year [1,2]. Aerobic exercise training has been shown to improve a number of CVD risk factors such as decreased functional capacity, insulin resistance, hypertension and dyslipidemia, which disproportionately affect obese populations [3,4]. As such, aerobic exercise training is recognized as a key component in both the primary and secondary prevention of CVD [5].

In recent years, central arterial stiffness has emerged as a marker that has been shown to significantly predict cardiovascular dysfunction. A number of investigations have linked increased aortic stiffness with a greater likelihood of cardiovascular events and mortality [6-8]. Laurent et al. [7], for example, reported that aortic stiffness significantly predicted all-cause and cardiovascular mortality independent of other risk factors such as age, previous cardiovascular disease and diabetes. Cruickshank et al. [8] similarly found aortic stiffness to significantly predict all-cause and cardiovascular mortality in diabetic and glucose intolerant groups independent of age, sex, and systolic blood pressure.

Reported effects of aerobic exercise training on arterial stiffness are a matter of some controversy. On the one hand, studies have demonstrated that aerobic exercise significantly reduces arterial stiffness in apparently healthy individuals [9] as well as in subjects with CVD risk factors [10]. On the other hand, there have been reports of no arterial effects [11,12,13] and even negative effects (increased arterial stiffness) [14] at higher exercise intensities.

Similar studies in obese populations are rare. In one of the few previously conducted studies, Balkenstein et al. [15] concluded that weight loss alone reduced carotid artery stiffness in obese men, but there was no additional improvement due to exercise. The initial mean body mass index (BMI) of subjects in this investigation [15] was 32.3 kilograms per meters squared (kg/m²), indicating the level of obesity in the study group was not severe. The purpose of the present pilot work was to begin to expand this area of research by testing the hypothesis that 10 weeks of aerobic exercise decreases aortic stiffness in a small, predominantly female, group of individuals classified as being moderate to severely obese by BMI (>35 kg/m²).

CASE REPORT

Five subjects (1 male/4 female, mean age 43.8 ± 9.7 years) with a BMI greater than 35 kg/m² (class II-III obesity) were recruited for this study. The Virginia Commonwealth University Internal Review Board approved the study, and all subjects gave informed consent. None of the subjects had been previously diagnosed with CVD. Baseline characteristics of the five subjects are listed in Table 1.

Table 1. Baseline Subject Characteristics.

<table>
<thead>
<tr>
<th>Subject Number</th>
<th>Gender</th>
<th>Age</th>
<th>Weight (kg)</th>
<th>BMI (kg/m²)</th>
<th>Prescription Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male</td>
<td>53</td>
<td>119.5</td>
<td>36.90</td>
<td>Quinapril, Gemfibrozil, Atorvastatin, Niacin</td>
</tr>
<tr>
<td>2</td>
<td>Female</td>
<td>50</td>
<td>120.3</td>
<td>42.60</td>
<td>Tolterodine, Albuterol</td>
</tr>
<tr>
<td>3</td>
<td>Female</td>
<td>44</td>
<td>97.0</td>
<td>36.30</td>
<td>Cetirizine</td>
</tr>
<tr>
<td>4</td>
<td>Female</td>
<td>28</td>
<td>134.5</td>
<td>49.10</td>
<td>Progesterone, Fexofenadine</td>
</tr>
<tr>
<td>5</td>
<td>Female</td>
<td>44</td>
<td>128.6</td>
<td>40.60</td>
<td>Estrogen</td>
</tr>
</tbody>
</table>

All subjects underwent cardiopulmonary exercise testing (CPET) prior to and following a 10-week aerobic exercise program using the Ellestad treadmill protocol. Ventilatory expired gas analysis was obtained using a metabolic cart (Vmax 29, SensorMedics, Inc., Yorba Linda, CA). Monitoring consisted of continuous 12-lead electrocardiography, blood pressure (BP) measurements at regular intervals during the exercise test, heart rate recordings every stage via the electrocardiogram, and rating of perceived exertion (Borg 6-20 scale). Subjects were encouraged to exercise to muscular fatigue. Test termination criteria followed American College of Sports Medicine guidelines [16]. Oxygen consumption (VO₂ in L/min, ml/min and mlO₂·kg⁻¹·min⁻¹), carbon dioxide production (VCO₂ in L/min) and minute ventilation (VE in L/min) were collected throughout the CPET. Maximal oxygen consumption (VO₂max) was defined as the final 30-second averaged value during the last stage of the exercise test. Ten second averaged VE/VCO₂ and VE/VO₂ data were input to a spreadsheet and VO₂ at ventilatory threshold (VT) was determined by the ventilatory equivalent method [17].

Aortic wave velocity (AWV) was measured prior to (week zero), at the midpoint (week five) and following (week 11) an aerobic exercise program using a rapid magnetic resonance (MR) technique previously
described [18]. The strategy of the measurement is to simultaneously record the initial systolic flow waveforms at two sites within the descending thoracic aorta, separated by a known distance (84 mm). Since the wave propagation rate is finite, a distinct temporal delay can be discerned between the two arterial flow waveforms. The separation distance divided by this observed delay time yields the AWV. After each AWV measurement, MR data analysis was performed offline on a personal computer [19]. Reported AWVs represent the mean of five individual measurements.

Subjects underwent 10 weeks of supervised aerobic exercise training primarily through the use of a motorized treadmill. Exercise intensity was initially set at the heart rate corresponding to VT on the initial treadmill test. Exercise intensity (via treadmill speed and/or grade) was gradually increased to maintain this heart rate throughout the training program. Subjects participated in 20-40 minute continuous aerobic training sessions an average of four times per week.

The Wilcoxon Signed Ranks Test was used to assess differences in BMI, VO_{2max}, VO_{2} at VT and exercise test duration before and after the 10-week training period. The Friedman two-way analysis of variance (ANOVA) by ranks assessed differences amongst AWV and BP measurements before, during and after the 10-week training period. A multiple comparison test was used to assess pairwise differences in the Friedman two-way ANOVA. All statistical tests with a p-value <0.05 were considered significant.

All five subjects completed the aerobic training program. Mean adherence to the exercise training sessions was 90%. Mean BMI, VO_{2max}, VO_{2} at VT and exercise test duration values pre- and post-training are listed in Table 2. The improvement in all four variables assessed was statistically significant following exercise training (p<0.05).

Mean AWV and resting BP results prior to, at the midpoint of and following exercise training are presented in Table 3. The multiple comparison test revealed that pre- and post-training AWV values were significantly different (p<0.01). Pre- and post-training resting systolic BP values were also significantly different (p<0.05), but resting diastolic BP values were not. Individual AWV data are illustrated in Figure 1.

**Discussion**

The results of the present study are consistent with previous investigations demonstrating a significant improvement in aerobic capacity and reduced systolic BP in overweight subjects after exercise training [3,4]. Moreover, our results suggest that an additional

**Table 2. Body Weight and Maximal Exercise Test Results.**

<table>
<thead>
<tr>
<th></th>
<th>Pre-Training</th>
<th>Post-Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>41.1 (±5.2)</td>
<td>39.4 (±5.8)*</td>
</tr>
<tr>
<td>Body Weight (kg)</td>
<td>120.0 (±14.3)</td>
<td>114.7 (±13.8)*</td>
</tr>
<tr>
<td>VO_{2max} (ml·kg(^{-1})·min(^{-1}))</td>
<td>24.3 (±6.0)</td>
<td>26.0 (±5.9)*</td>
</tr>
<tr>
<td>VO_{2} at VT (ml·kg(^{-1})·min(^{-1}))</td>
<td>15.0 (±3.2)</td>
<td>17.9 (±2.6)*</td>
</tr>
<tr>
<td>Exercise test duration (seconds)</td>
<td>401.2 (±54.9)</td>
<td>445.0 (±56.4)*</td>
</tr>
</tbody>
</table>

* Significant difference from pre training, p<0.05

**Table 3. Aortic Wave Velocities and Resting BP Results.**

<table>
<thead>
<tr>
<th></th>
<th>Pre-Training</th>
<th>Mid-Training</th>
<th>Post-Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic Wave Velocity (m/s)</td>
<td>9.9 (±1.2)</td>
<td>7.6 (±1.8)</td>
<td>6.2 (±1.4)**</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>134.6 (±13.9)</td>
<td>125.6 (±16.4)</td>
<td>122.4 (±12.5)*</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>83.0 (±8.0)</td>
<td>75.0 (±11.3)</td>
<td>76.2 (±6.6)</td>
</tr>
</tbody>
</table>

Significant difference from pre training, * p<0.05; ** p<0.01

**Figure 1. Individual Aortic Wave Velocity Values.**
positive adaptation to aerobic training may be a significant reduction in arterial stiffness. All five subjects in this study exhibited elevated aortic stiffness initially; based on our own unpublished data, predicted AWVs for normal weight subjects of these ages range from 4.5 to 5.9 (mean = 5.4) m/s. All five subjects trended toward a reduction in AWV at 5 weeks with a further decrease after 10 weeks of training (Figure 1). While other investigators have reported that aerobic training reduces arterial stiffness [9,10], none have done so in a group considered to be moderate to severely obese (BMI ≥ 35 kg/m²). Balkenstein et al. [15] did report a reduction in carotid stiffness in a group of obese subjects following a combined diet and exercise intervention. However, the subject group in that study was exclusively male and had a considerably lower mean BMI compared to subjects in the present study. Furthermore, these authors attributed the arterial compliance change to weight loss, with no additional influence of exercise. Although subjects in the present study lost weight over the course of the intervention, they nevertheless all remained obese by BMI classification following exercise training, suggesting that normalization of body weight is not required to effect a significant reduction in arterial stiffness. Stewart et al. [13] recently reported that a six-month aerobic training program had no impact on arterial stiffness in a group of older individuals. The mean age and BMI for subjects in this study were greater than 60 years and less than 30 kg/m², both of which were significantly different characteristics compared to the five individuals assessed in the present report. Age and/or weight may therefore influence the effect exercise training has on arterial stiffness. Given the limited amount of research in the area of arterial stiffness using obese subjects and the apparent differences between previous work and our investigation, the results of the present study appear novel.

Although mechanisms linking obesity with elevated arterial stiffness are incompletely known, one identified factor is abdominal visceral fat [20]. In another study [21], aortic distensibility was found to be negatively correlated with visceral fat, but not significantly associated with subcutaneous fat or body mass index. Moreover, aerobic exercise is known to preferentially reduce visceral fat [22]. Additional postulated beneficial influences of exercise include release of growth factors, improved left ventricular diastolic function, vascular remodeling via increased shear stress, increased release of endothelial derived nitric oxide and reduced systemic inflammation [23,24]. All of these mechanistic hypotheses are plausible explanations for exercise-induced reduction in arterial stiffness observed in the present investigation.

**Conclusions**

In conclusion, aerobic exercise training clearly produces multiple positive health benefits in subjects considered to be obese. The results of this pilot investigation indicate that a reduction in aortic stiffness may be an additional positive health adaptation of aerobic training in this high-risk population.

**References:**