Augmentation Index and Systolic Load Are Lower in Competitive Endurance Athletes

David G. Edwards and Jesse T. Lang

Background: Arterial stiffness increases with age, and chronic endurance exercise has been shown to attenuate increases in arterial stiffness in older individuals. The aim of this study was to assess the effect of varying fitness levels on wave reflection in young healthy adults.

Methods: A total of 32 subjects (16 competitive endurance athletes and 16 recreationally active subjects) underwent brachial artery blood pressure (BP) measurement (by sphygmomanometry) and central aortic pressure (by non-invasive radial artery applanation tonometry and use of a generalized transfer function) measurements at rest. Central aortic augmentation index (AI), an index of wave reflection, and tension–time index (TTI), an indicator of systolic load, were calculated from the aortic pressure waveform. Physical fitness was determined through a maximal oxygen consumption (VO$_{2\text{max}}$) test performed on a treadmill.

Results: The two groups did not differ in age, height, weight, resting BP, or blood lipids. The VO$_{2\text{max}}$ was significantly higher in the competitive group compared with the recreational group (65 ± 1.9 v 49 ± 1.8 mL/kg/min, $P < .05$). The AI was lower in the competitive group compared with the recreational group ($-2.1\% \pm 2.1\%$ v $4.5\% \pm 2.9\%, P < .05$), as was TTI (1679 ± 61 v 1868 ± 58, $P < .05$).

Conclusions: Wave reflection is lower in competitive endurance athletes who have higher fitness levels and who exercise at a higher intensity, for a longer duration, and more frequently as compared with recreationally active individuals. These differences may be due to functional changes that occur as a result of training. Am J Hypertens 2005;18:679–683 © 2005 American Journal of Hypertension, Ltd.

Key Words: Exercise, wave reflection, arterial stiffness.
of chronic intense exercise training on wave reflection in young healthy adults by assessing the aortic augmentation index using pulse wave analysis. We hypothesized that wave reflection would be lower in competitive endurance athletes whose training is higher in intensity, longer in duration, and more frequent compared with recreationally active subjects.

**Methods**  
**Subjects**

A total of 32 healthy men and women (24 men and 8 women, age 18 to 45 years) were recruited for participation in the study. The subjects were divided into two groups (12 men and 4 women per group) \( n = 16 \), competitive endurance–trained athletes and recreationally active individuals. The competitive endurance–trained athletes had participated in an event within the past 3 months and ran a minimum of 30 miles per week. The recreationally active individuals participated in light to moderate activities 2 to 4 times per week.

All subjects were nonobese and devoid of any overt chronic diseases as assessed by medical history questionnaire and blood lipid analysis. None of the subjects smoked or were taking any medications. All subjects refrained from alcohol consumption or exercising 24 h before testing and caffeine 12 h before testing.

The protocol was reviewed and approved by the University of New Hampshire Institutional Review Board, and all subjects gave informed consent to participate in the study.

**Measurements**

**Pulse Wave Analysis**

After a seated resting period of 10 min, the subject’s brachial BP was taken in duplicate with an automated BP cuff by oscillometric sphygmomanometry. An arterial waveform was recorded by placing a high-fidelity strain-cuff by oscillometric sphygmomanometry. An arterial brachial BP was taken in duplicate with an automated BP cuff. The central aortic pressure waveform was obtained from the measured radial artery pressure waveform using a generalized transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). The central aortic pressure wave was synthesized from the measured radial artery pressure waveform using a generalized transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). The central aortic augmentation index (AI), an index of wave reflection, was obtained from the synthesized aortic pressure waveform. Additional calculations derived from the synthesized aortic pressure wave were the tension–time index (TTI), diastolic pressure time index (DPTI), and subendocardial viability ratio (SEVR). The TTI, or area under the systolic portion of the curve, has been shown to be related to systolic load or the work of the heart and oxygen consumption. The DPTI, or area under the diastolic portion of the curve, is associated with coronary perfusion. The SEVR is the ratio of DPTI to TTI and an index of subendocardial perfusion. Pulse pressure (PP) amplification was calculated as the ratio of brachial PP to central aortic PP.

**Table 1.** Baseline characteristics of study participants

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Competitive</th>
<th>Recreational</th>
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<tbody>
<tr>
<td>Age (y)</td>
<td>25 ± 1.8</td>
<td>26 ± 1.7</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>174 ± 2.4</td>
<td>173 ± 1.9</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68 ± 2.1</td>
<td>71 ± 2.8</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>177 ± 10.9</td>
<td>191 ± 6.3</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dL)</td>
<td>102 ± 9.8</td>
<td>108 ± 6.4</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dL)</td>
<td>57 ± 3.2</td>
<td>53 ± 3.8</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>96 ± 2.5</td>
<td>100 ± 2.4</td>
</tr>
<tr>
<td>Percent fat (%)</td>
<td>10 ± 1.3</td>
<td>16 ± 1.1*</td>
</tr>
<tr>
<td>VO\textsubscript{2}\text{max} (mL/kg/min)</td>
<td>65 ± 1.9</td>
<td>49 ± 1.8*</td>
</tr>
</tbody>
</table>

Competitive = competitive endurance–trained athletes; Recreational = recreationally active individuals; VO\textsubscript{2}\text{max} = maximal oxygen consumption.  
Values are means ± SE.  
* \( P < .05 \) vs competitive group.

**Maximal Oxygen Consumption**

Maximal oxygen consumption (VO\textsubscript{2}\text{max}) was used to determine each subject’s cardiovascular fitness level. A modified Costill-Fox graded exercise test was performed on a treadmill (Quinton Q65, Seattle, WA) to determine VO\textsubscript{2}\text{max}.\textsuperscript{10} Expired gases were analyzed using a Sensormedics Vmax229 series metabolic cart (Yorba Linda, CA).

**Blood Lipid and Glucose Analysis**

All blood analyses were performed after subjects had completed a 12-h fast and were measured using a Cholestech LDX desktop chemistry analyzer (Cholestech, Hayward, CA).

**Body Composition**

The Siri equation\textsuperscript{11} was used to estimate percent fat after the estimation of body density, using skinfold calipers (Harpenden, Ann Arbor, MI) and the equations described by Jackson.\textsuperscript{12}

**Statistics Analysis**

Results were analyzed using unpaired \( t \) tests to compare between group differences. An \( \alpha \) level of \( P < .05 \) was required for significance. All data are expressed as mean ± SE.

**Results**

**Subject Characteristics**

The subject characteristics for both groups are summarized in Table 1. There were no significant differences between groups in regard to height, weight, or age. Additionally, there were no differences in fasting blood lipids or glucose between groups. As expected, total body fat was significantly lower in the competitive group (\( P < .05 \)).
Table 2. Hemodynamic variables of study participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Competitive</th>
<th>Recreational</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>61 ± 2.5</td>
<td>68 ± 2.9*</td>
</tr>
<tr>
<td>Peripheral systolic pressure (mm Hg)</td>
<td>121 ± 2.3</td>
<td>122 ± 2.8</td>
</tr>
<tr>
<td>Peripheral diastolic pressure (mm Hg)</td>
<td>68 ± 2.1</td>
<td>68 ± 2.3</td>
</tr>
<tr>
<td>Peripheral pulse pressure</td>
<td>55 ± 2.1</td>
<td>54 ± 3.5</td>
</tr>
<tr>
<td>Central systolic pressure (mm Hg)</td>
<td>100 ± 1.8</td>
<td>103 ± 2.3</td>
</tr>
<tr>
<td>Central diastolic pressure (mm Hg)</td>
<td>69 ± 2.1</td>
<td>69 ± 2.3</td>
</tr>
<tr>
<td>Central pulse pressure</td>
<td>31 ± 1.3</td>
<td>34 ± 2.1</td>
</tr>
<tr>
<td>Pulse pressure amplification</td>
<td>1.726 ± 0.02</td>
<td>1.624 ± 0.05*</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1.
Values are means ± SE.
* P < .05 v competitive group.

wise, the competitive group displayed a significantly higher VO2max than the recreational group (P < .05).

Hemodynamics

Peripheral and central BP values for both groups are presented in Table 2. No statistical differences were observed between groups in both peripheral and central systolic or diastolic pressure. Although differences in PP did not reach statistical significance, PP amplification was significantly higher in the competitive group (P < .05).

The AI was significantly lower in the competitive group than in the recreational group (P < .05) (Fig. 1). Similarly, TTI was also significantly lower in the competitive group (P < .05) (Fig. 2). There were no differences in DPTI (3270 ± 76 v 3254 ± 114), but the difference in TTI resulted in a significantly higher SEVR in the competitive group (199 ± 8.5 v 177 ± 9.1, P < .05).

Discussion

The aim of this study was to assess the effect of varying fitness levels on wave reflection in young healthy adults. The major finding from this study was that wave reflection and systolic load were lower in the competitive endurance–trained group. To our knowledge, this is the first study to demonstrate a difference in the effects of arterial stiffness in young healthy adults with different aerobic capacities.

The AI is known to be affected by a number of variables including age, height, gender, and blood lipids. In the present study the two groups were well matched for age and height and did not differ in the number of women in each group (n = 4). In addition, there were no differences in blood lipids between the competitive and recreational exercise groups. Therefore, it is unlikely that any of these variables accounted for differences in AI between the two groups. Heart rate has also been shown to effect the measurement of AI. A low heart rate allows for more time for the return of the reflected wave thus increasing AI. The competitive group had a lower resting heart rate than the recreational group but still had a lower AI. When we normalized AI at a heart of 75 beats/min, the difference between groups was even greater (−7.1% ± 1.8% v 1.1% ± 2.2%).

Body fat was significantly higher in the recreational group than in the competitive group. However, this variable has not been shown to be an independent predictor of arterial stiffness. Therefore, the difference in AI between groups appears to be due to the disparity in aerobic fitness levels seen between groups. These findings are consistent with earlier studies demonstrating a relationship between arterial stiffness and fitness level.

Previously, no differences have been found in local arterial compliance of the carotid artery between young adults of varying fitness levels. In the present study, the augmentation index was used as a measurement of wave reflection from the periphery. Therefore, it is possible that the different methods of assessing arterial stiffness were responsible for the disparity observed between the results found in the current study and those previously reported.
The difference in AI observed in the current study was accompanied with differences in PP amplification, systolic load, and SEVR. Although the young subjects in this study are not at risk for ischemia, these results may indicate that the competitive endurance group may be better able to maintain a lower central PP and defend SEVR over time. A long-term prospective study will be required to confirm this hypothesis.

Although it is possible that the mechanisms by which exercise affects arterial stiffness could occur in any area of the arterial system, it seems unlikely that dramatic changes in central arterial stiffness would occur so early in young adults. Structural changes in the central arteries, such as a decrease in the amount of elastin and an increase in the amount of collagen take considerable time to occur. Therefore, the exercise-induced changes to the arterial system in young adults probably take place in the muscular arteries or arterioles.

The most likely mechanism by which high-intensity endurance exercise may reduce systemic arterial stiffness is through a decrease in sympathetic tone and an improvement in endothelial function. There is evidence that alterations in sympathetic tone alter the compliance of the arterial system. The subjects in the competitively trained group may have had a lower sympathetic tone as a result of their training. This decreased sympathetic tone could have contributed to a lower arterial stiffness by diminishing the vasoconstriction occurring in the smooth-muscle cells surrounding the arteries.

Furthermore, studies have shown that exercise can improve endothelial function in both young and older individuals. The endothelium releases nitric oxide, which results in smooth-muscle relaxation. Inhibition of nitric oxide synthase results in increased arterial stiffness and an acute increase in augmentation index, demonstrating an important role for nitric oxide in maintaining the buffering capacity of the arterial system. Nitric oxide can also exert a sympathoinhibitory effect, which could further contribute to its ability to maintain compliance of the arterial system. In summary, it is possible that a reduction in sympathetic tone or an improvement in endothelial function, or both, is responsible for exercise-induced alterations of arterial stiffness in younger individuals; however, further research is needed to confirm this.

We have demonstrated that wave reflection is lower in competitive endurance–trained athletes who exercise at a higher intensity, for a longer duration, and more frequently than recreationally active individuals. Future research should focus on the mechanism responsible for these differences and whether reductions in arterial stiffness at a young age reduce the incidence of cardiovascular disease later in life. The AI is associated with a higher incidence of coronary artery disease in middle-aged and older individuals; however, the predictive value of AI at a young age has yet to be investigated. In addition, the effect of each component of exercise prescription (intensity, duration, and frequency) on changes in arterial stiffness should be investigated.

References


