Brief Communication

Effect of Exercise Training on Central Aortic Pressure Wave Reflection in Coronary Artery Disease

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Background: Arterial stiffness is an important factor for cardiovascular performance and a predictor of cardiovascular risk. We investigated the effect of exercise training on arterial stiffness in coronary artery disease (CAD).

Methods: Arterial stiffness was assessed before and after 12 weeks of either exercise training or standard care in CAD patients. Arterial stiffness was evaluated by aortic augmentation index and time delay of the reflected wave (Δt) using the SphygmoCor system.

Results: The augmentation index decreased (30% v 26%, P < .05) and Δt increased (136 msec v 144 msec, P < .05) in patients with CAD after 12 weeks of exercise.

Conclusion: These results suggest that endurance exercise training improves systemic arterial stiffness in individuals with CAD. Am J Hypertens 2004;17:540–543 © 2004 American Journal of Hypertension, Ltd.

Key Words: Exercise, wave reflection, arterial stiffness, coronary artery disease.

The arterial system constitutes the external afterload placed on the ventricle during contraction and ejection. The load has both static and dynamic components, as the ventricle ejects a pulsatile blood flow into a distensible arterial system. The static or resistive component is dependent on blood viscosity and arteriolar caliber, whereas the dynamic or compliant component is dependent on the elastic properties of the larger arteries and pulse wave reflections1. Central arterial stiffness is increased in coronary artery disease and myocardial infarction.2 An increase in arterial stiffness in coronary artery disease (CAD) can lead to myocardial ischemia as a result of increased myocardial oxygen demand and a decrease in coronary perfusion. Aortic stiffening in animal studies using bandaging3 or a stiff plastic tube bypass4,5 results in increased pulse pressure and cardiac work and a reduction in coronary perfusion. During total coronary occlusion an increase in arterial stiffness was found to result in a marked decrease in myocardial function.5 This evidence suggests that aortic stiffness increases myocardial oxygen demand and reduces myocardial perfusion, particularly in the setting of CAD. In support of this, a recent investigation found that time to ischemia during treadmill testing was inversely correlated with arterial stiffness in CAD patients, suggesting that arterial stiffness is a principal determinant of ischemic threshold in CAD.6

Exercise training appears to be associated with reduced arterial stiffness or increased compliance both in animals7 and in healthy humans8. When the effects of advancing age are controlled for, arterial stiffness is less in persons with a higher maximal oxygen consumption (VO2max)9 and 3 months of aerobic walking has been shown to increase measures of central arterial compliance (inverse of stiffness) in middle-aged and older men.10 Exercise training–induced alterations in arterial stiffness would be of great benefit in CAD, potentially reducing myocardial oxygen demand and symptoms of ischemia. The purpose of this study was to investigate the effects of 12 weeks of endurance exercise training, performed in a standard cardiac rehabilitation setting, on the amplitude and timing of wave reflections in CAD.

Methods

Study Population

Patients with CAD were recruited from the Shands Hospital at AGH Cardiac Rehabilitation Program (Gainesville, FL). All patients were diagnosed with CAD as documented by myocardial infarction or documented coronary

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lesion of $\geq 70\%$ stenosis in at least 1 major coronary vessel. All patients were optimally medicated and none had any medication changes throughout the study. The selection of subjects was not based on sex or ethnicity. A total of 20 patients were assigned to either 12 weeks of standard cardiac rehabilitation or a 12-week control period. Our control group consisted of a cohort of patients with CAD who did not have access to cardiac rehabilitation because of insurance limitations or travel distance. The study was approved by the University of Florida Health Science Center Institutional Review Board and all subjects gave written informed consent.

**Exercise Training**

Exercise training took place in the Shands Hospital at AGH Cardiac Rehabilitation Program. Exercise consisted of treadmill walking, stationary cycling, and upper body ergometry 3 times per week for 12 weeks. Exercise intensity began at 40% to 50% of maximal heart rate reserve or as symptoms allowed and gradually progressed to 70% to 85% of maximal heart rate reserve. The duration of each exercise session began at 15 to 20 min or as tolerated and progressed to 40 to 50 min of sustained walking and cycling. A log of heart rate, blood pressure, duration, rating of perceived exertion, treadmill speed and grade, and cycle workload was kept for each exercise training session.

**Pulse Wave Analysis**

After a supine resting period of 15 min, patients underwent duplicate measurements of brachial artery cuff blood pressure by oscillometric sphygmomanometry and calibrated noninvasive radial artery pulse waveform using applanation tonometry and a high-fidelity strain gauge transducer (Millar Instruments, Houston, TX). A central aortic pressure waveform was synthesized from the measured radial artery pressure waveform using a generalized transfer function (SphygmoCor; AtCor Medical, Sydney, Australia). The travel time of the forward pressure wave from the heart to the major reflection site and back (travel time of the reflected wave $[\Delta t]$) and central aortic augmentation index were obtained from the synthesized aortic pressure waveform.

**Statistical Analysis**

Analysis of variance with repeated measures was used to analyze the data in patients with CAD before and after 12 weeks of cardiac rehabilitation or the control period. When a significant group by time interaction was observed, within-group post hoc comparisons between time points and between-group post hoc comparisons at each time point were performed using Tukey’s post hoc analyses. An $\alpha$ level of $P < .05$ was required for statistical significance.

**Results**

**Subject Characteristics**

Exercise ($n = 10$) and control ($n = 10$) groups did not differ with respect to age ($55 \pm 8 \text{ v } 63 \pm 8$ years), height ($175 \pm 8 \text{ v } 176 \pm 8$ cm), weight ($87 \pm 25 \text{ v } 92 \pm 21$ kg), or body mass index ($28 \pm 7 \text{ v } 30 \pm 6$ kg/m$^2$).

**Hemodynamics**

Hemodynamic variables are listed in Table 1. Resting heart rate was significantly greater in the control group versus the exercise group at both time points. Brachial systolic, diastolic, and pulse pressures were not different between the two groups. Additionally, there were not statistical differences in calculated central aortic systolic, diastolic, or pulse pressures between groups. After 12 weeks of exercise training or the 12-week control period there were no significant changes in any hemodynamic variables in either group. However, there was a nonsignificant reduction in central aortic systolic and pulse pressures in the exercise-trained group, which was likely related to changes in wave reflection.

**Pulse Wave Analysis**

Results from pulse wave analysis of synthesized central aortic pressure wave are presented in Fig. 1. Baseline
Discussion

Pulse wave analysis in the present study demonstrated that 12 weeks of endurance training in CAD patients resulted in a reduction in arterial stiffness as evidenced by a reduction in augmentation index (30% at baseline vs 26% at 12 weeks) and an increase in the travel time of the reflected wave (Δτ) (136 vs 144 msec) in the exercise group. Although there has been limited research in the area of exercise and arterial stiffness, there is independent evidence that supports the findings of the present study. In healthy individuals, higher aerobic fitness levels have been shown to be associated with lower central arterial stiffness as measured by pulse wave analysis, pulse wave velocity, and magnetic resonance imaging, suggesting that large artery arterial stiffness may be influenced by exercise capacity. Additionally, systemic arterial compliance (the inverse of stiffness) has positively correlated, whereas the β-index, a measure of arterial stiffness of the aortic arch, has been shown to be inversely correlated with VO2max. Furthermore, time to exhaustion on a treadmill has shown to be positively correlated with arterial compliance and negatively correlated with rate–pressure product.

Animal studies suggest that arterial stiffness increases myocardial oxygen demand and reduces myocardial perfusion, particularly in the setting of coronary occlusion. In support of this, a recent investigation found that in 91 patients with CAD, time to ischemia during treadmill testing was inversely correlated with arterial stiffness independent of gender, age, mean pressure, degree of disease, left ventricular mass, and smoking status, suggesting that arterial stiffness is a principal determinant of ischemic threshold in CAD.

To date there have been only 2 prospective studies examining the effects of endurance exercise training on elastic properties of the arterial system. A 30-min period of cycling at 65% of VO2max three times per week for 4 weeks resulted in a 30% improvement in systemic arterial compliance in healthy individuals. In a cross-sectional study Tanaka et al found that dynamic arterial compliance of the carotid artery was higher in endurance trained middle age and older men compared with sedentary and recreationally active men of the same age. The same study reported that 3 months of aerobic exercise in a group of 20 sedentary middle-aged and older men resulted in an increase in carotid arterial compliance that was similar to that in an endurance-trained group of men.

In conclusion, the present study prospectively investigated the effects of endurance training on arterial properties using pulse wave analysis in patients with CAD. Our results suggest that in patients with CAD, endurance exercise training improves systemic arterial stiffness. The reduction in the dynamic components of afterload after exercise training may result in a decrease in myocardial oxygen demand and ischemic symptoms.

References

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