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The Effect of an Acute Bout of Aerobic Exercise on Arterial Stiffness and Wave Reflection in Patients with Coronary Artery Disease

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research ARTICLE

The Effect of an Acute Bout of Aerobic Exercise on Arterial Stiffness and Wave Reflection in Patients with Coronary Artery Disease

—Alyssa L. Borell and Cristin A. Davis

Aerobic exercise increases the compliance of our arteries, providing a way to improve and maintain heart health. Compliant arteries are flexible and dilated, allowing for more blood, oxygen, and other nutrients to flow easily to the vital organs and skeletal muscle. But in patients with Coronary Heart Disease (CAD), arterial stiffness is elevated.

CAD is associated with the hardening of the vessels that supply blood to the heart. If elevated and left untreated, systolic blood pressure, or the pressure exerted when the heart contracts, can increase the risk of heart failure, heart attack, stroke, and kidney failure. Our research examined how a single bout of aerobic exercise (i.e. acute exercise) can alter arterial stiffness and blood pressure to reduce the risk of cardiovascular events, such as heart attack, in patients with CAD. We hypothesized that arterial stiffness and systolic blood pressure would be reduced following a single bout of sub-maximal, aerobic exercise.

The Arterial Stiffness/Systolic Blood Pressure Relationship

When the heart contracts, a pressure wave is sent through the blood vessels. In patients with arterial stiffness, this pressure wave travels faster. An increase in arterial stiffness decreases the arterial system's ability to absorb pulsations from the heart and causes an early return of reflective waves from the periphery, both of which lead to an increase in systolic blood pressure. The increase in central aortic systolic pressure leads to an increase in the heart's oxygen demand, which can cause myocardial ischemia, or lack of oxygen to the heart, and infarction, or heart attack. Since systolic blood pressure is a better predictor of death due to cardiovascular events than diastolic blood pressure (the pressure within the vasculature when the heart is at rest), arterial stiffness is an important consideration in the treatment of CAD (1).

Chronic, or regular, exercise training benefits CAD patients by decreasing blood lipids and blood pressure. Research shows that as little as twelve weeks of exercise training can decrease arterial stiffness in CAD patients (2); however, the effect of an acute bout of exercise has yet to be investigated. Animal studies have shown that one day of exercise training provides "cardioprotection" against subsequent periods of ischemia (3).

Acute endurance exercise is typically accompanied by post-exercise hypotension (PEH), or lowered blood pressure following exercise, in both normotensive (i.e. those with normal blood pressure) and hypertensive individuals (4). PEH is traditionally thought to occur because of decreased resistance in the vasculature of the

periphery (4), but alterations in arterial stiffness and wave reflection may be partly responsible for the cardioprotection associated with short-term (1-day) training.

Research in Motion

Two male subjects from the University of New Hampshire Cardiac Rehabilitation Program were recruited for this study. Both subjects, ages 59 and 70, had documented CAD as well as prior cardiac events and/or heart surgery. They remained optimally medicated throughout the study. Each subject signed a written consent for his participation.

All testing was performed in the Robert Kertzer Exercise Physiology Laboratory. Participants received a detailed description of the expectations and procedures. Both subjects fasted for approximately twelve hours prior to this first meeting, and blood glucose and blood lipid levels were measured. After we performed the lipid profile, we took a resting measurement of arterial stiffness.

We estimated the amplitude of central systolic arterial wave reflection by the augmentation index (AI). Essentially, AI is an indicator of systemic arterial stiffness, simple to measure and highly reproducible. In adults above approximately twenty years of age and of average height, a distinct inflection point (Pi) occurs when the heart contracts either before or after peak systolic pressure (Ps) (7). AI is calculated as $(Ps - P_i) / (Ps - P_d)$ where Ps = systolic pressure, Pd = diastolic pressure, and Pi = pressure at the inflection point.

After the initial measurement of arterial stiffness, the two subjects completed a forty-minute bout of sub-maximal aerobic exercise at 50-85% of Heart Rate Reserve (HRR), consisting of either brisk walking or riding a recumbent bicycle. HRR is determined by the following equation: $HRR = \text{Max Heart Rate} - (\text{Resting Heart Rate} \times \% \text{ Intensity}) + \text{Rest. Heart Rate}$. Exercise sessions were supervised during the UNH Cardiac Rehabilitation Program to maintain safety and ensure that each subject's heart rate stayed in the 50-85% HRR range. Following the exercise session, a two-minute cool-down brought the patients to near-resting heart rate. We measured arterial stiffness at five minutes post-exercise, and thereafter at fifteen-minute, thirty-minute, one-hour, one-and-a-half-hour, and two-hour intervals.

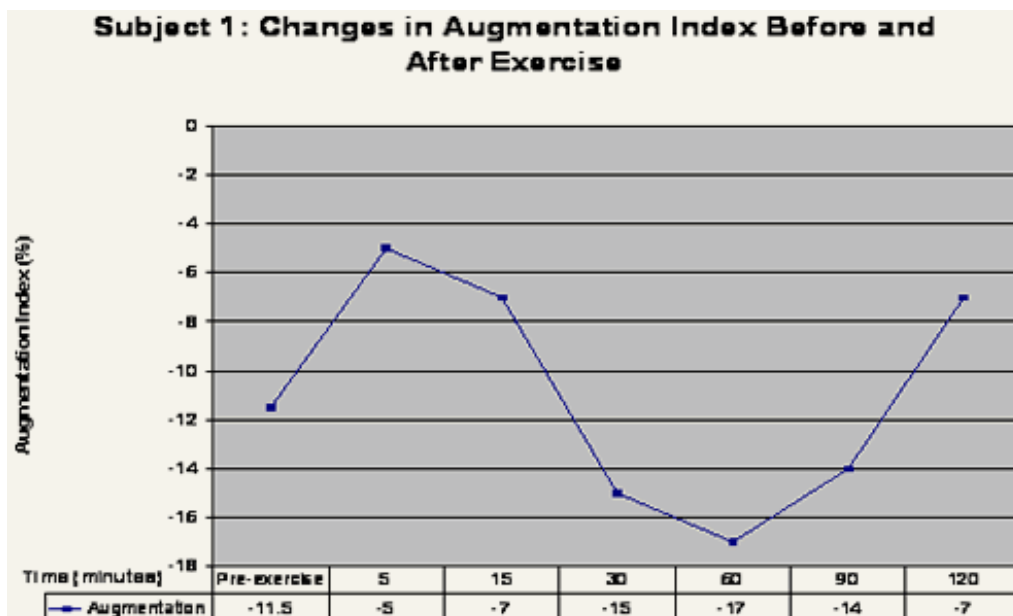


Figure 1. Subject 1 had a lesser degree of CAD, and thus a smaller change in AI because it was negative to begin with.

Subject 2: Changes in Augmentation Index Before and After Exercise

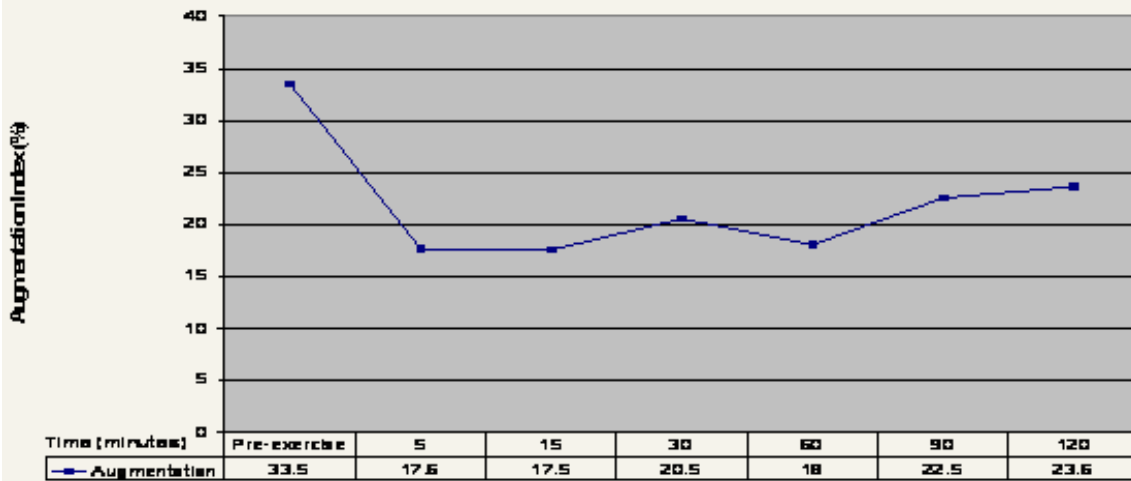


Figure 2. Subject 2 had more severe CAD, measured by the number of heart and ischemic attacks. Therefore, compared to subject 1, there was a greater change in augmentation index (AI) because it was largely positive to begin with.

Pulse wave analysis demonstrated that a single bout of aerobic exercise decreases arterial stiffness, as shown by a decrease in augmentation index (AI). It can be assumed from the collected data that the stiffer the vasculature, the greater the changes in augmentation index and therefore, arterial compliance. Subject 1 had a largely negative AI pre-exercise, which resulted in a smaller overall change in AI. This is likely due to the fact that arterial stiffness was already low to begin with. Subject 2 had a very high AI pre-exercise, due to a stiffer vasculature. This resulted in a larger change in AI following exercise, compared to that of subject 1. Systolic blood pressure mirrored this effect. Blood pressure and AI decreased with a 45-minute bout of aerobic exercise and slowly leveled off over time. Even two hours post, AI was still lower than the initial (pre-exercise) measurement for both subjects.

Our findings suggest that exercise may have an acute effect on improving arterial compliance. This means that exercise may have therapeutic and preventative benefits for individuals with CAD, especially when performed on a daily or almost daily basis, as acute effects are short-lived. While participation in physical activity may not always elicit increases in the traditional markers of human performance such as body composition, it does improve health by reducing heart disease risk factors. Thus, the benefits associated with regular exercise and physical activity contribute to a healthier lifestyle, greatly improving a person’s functional capacity and quality of life.

Additional research with more subjects may show an even greater trend in the reduction of arterial stiffness. The type and severity of disease also may affect the amount of augmentation change, but more research should be performed that considers other factors such as gender, fitness level, and age. Future research may also explore the possibility of positive exercise effects on factors other than just arterial stiffness. In conclusion, exercise is vitally important to the improvement of health as well as the management of CAD.

First and foremost, we would like to thank the Undergraduate Research Opportunities Program for providing us with the generous funding that made this wonderful experience possible. Second, we extend our greatest thanks to Dr. David Edwards who helped us with every aspect of this project. We would also like to

acknowledge our gracious subjects who volunteered many hours of their time and ultimately made this research project possible and quite enjoyable!

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Author Bios

Alyssa L. Borell, from Medfield, Massachusetts, graduated from UNH in 2005 with a degree in kinesiology and a focus on exercise science. As a result of her research, Alyssa has a better understanding of the pathophysiology of heart disease and the importance of regular exercise in the prevention of disease. She and Cristin Davis submitted their article to Inquiry because “Education regarding the importance of exercise for health as well as disease management is very important to us and we want to share our findings with as many people as possible.” Alyssa plans to pursue a master’s degree in applied anatomy and physiology.

Cristin A. Davis, from Foxboro, Massachusetts, graduated from UNH in 2005 with a degree in kinesiology and a focus on exercise science. Through this research project she learned a great deal about exercise and coronary heart disease, as well as about the research process itself. Cristin found the research to be challenging, especially working with subjects; however, she describes it as a very rewarding experience. She explains, “Completing this research has finalized my goals for the future.” In August, 2006 Cristin will begin the doctorate program in physical therapy at Duke University.

Mentor Bio

Dr. David Edwards, former assistant professor in the Kinesiology department at UNH, is an assistant professor in the department of Health, Nutrition, and Exercise Sciences at the University of Delaware. His current research activities focus on cardiovascular physiology as well as oxidative stress and inflammation.