Effect of Exercise on Arterial Compliance

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In an era of molecular biology, “gene therapy,” and increasingly sophisticated technological approaches to treat cardiovascular disease, it is easy to overlook the physical forces generated by and acting on the human cardiovascular system. In this issue of Circulation, Tanaka and colleagues report that aerobic exercise training can blunt the age-associated stiffening of large blood vessels in humans. In this context, why is this important, what are some of Tanaka and colleagues’ key observations, and what mechanisms might explain them?

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It is well known that arterial compliance declines with age even in healthy individuals with no overt cardiovascular disease. This means that the large conducting vessels (ie, the aorta and its major branches) all lose their ability to distend in response to an increase in pressure. As a consequence of this stiffening, when blood is ejected from the heart during systole, there is a smaller change in arterial diameter with aging, and this reduction in arterial compliance appears to be an independent risk factor for the development of cardiovascular disease. This reduction in compliance also contributes to isolated systolic hypertension in the elderly. Additionally, as the vessels stiffen, the physical forces that oppose aortic valve opening increase and can contribute to ventricular hypertrophy, aortic root dilation, valvular dysfunction, and heart failure.

To evaluate the impact of physical activity on arterial compliance, Tanaka and colleagues used 2 approaches. In the first phase of the study, they performed a cross-sectional study of a large number of men who were either inactive, recreationally active (participating in moderate exercise 3 or more times per week), or endurance-trained individuals who exercised vigorously and participated in competitive endurance events. Their sample included young (18 to 37 years), middle-aged (38 to 57 years), and older (58 to 77 years) subjects. In this cross-sectional portion of the study, they demonstrated that arterial compliance (measured at the carotid) fell from roughly 2 mm$^2$/mm Hg to 1.2 to 1.3 mm$^2$/mm Hg in middle-aged and older sedentary humans. In the highly trained middle-aged and older subjects, exercise appeared to reduce the decline in compliance with aging by about 50%, and positive trends were seen in the recreationally active groups. These findings provided substantial cross-sectional evidence that recreational activity, and especially vigorous physical activity, can limit reductions in arterial compliance with aging.

In the second phase of the study, 20 healthy middle-aged and older sedentary subjects were studied before and after 3 months of aerobic exercise training. By the end of the protocol, the subjects were walking briskly or jogging 40 to 45 minutes per day, 4 to 6 days per week, at an intensity equal to 70% to 75% of their maximal heart rate. This exercise intervention caused a substantial increase in arterial compliance, thereby partially reversing the age-related changes.

What mechanisms might explain the reduced arterial compliance with aging, and how are they modified by exercise? A variety of changes in arterial structure with aging probably contribute to reduced arterial compliance and increased arterial “stiffness.” These include vascular smooth muscle hypertrophy, replacement of viable cells with connective tissue, and increased cross-linking of connective tissue. Exercise training, or moderate physical activity, might modify these changes in several ways. First, and perhaps most simply, when humans exercise there is an increase in arterial pressure and heart rate. These changes and the physical forces acting on the large conducting vessels might cause the vessels to deform and act in a manner that is similar to “stretching exercises” in skeletal muscle. In other words, occasional periods of increased deformation of the large blood vessels may combat some of the connective tissue cross-linking that occurs as a result of aging. Second, skeletal muscle vasodilates dramatically during exercise, and at least some of this vasodilation in resistance vessels is propagated upstream to large conducting vessels. Third, the increased pulsatile flow in the aorta associated with exercise training might evoke the release of nitric oxide acutely, as well as lead to an upregulation of nitric oxide production and an increase in the production of other vasodilating factors. These factors, if upregulated, might directly relax vascular smooth muscle in conducting arteries, and the nitric oxide itself might have a potent antiinflammatory effect that would inhibit vascular smooth muscle proliferation. These changes would all operate to limit or reverse age-associated reductions in arterial compliance. In this context, it should be noted that improvements in endothelial function with endurance exercise training are most consistently seen in conducting arteries, and training the legs can cause improvements in nitric oxide–mediated vasodilator function in the arms.

Another potentially important impact of stiffer vessels on cardiovascular function in older humans might be at the level of the arterial baroreflexes. In response to aortic arch or carotid artery distension during systole, afferent nerves in...
these regions send signals to the brain stem that act centrally to inhibit sympathetic outflow to the periphery and augment vagal tone to the heart. If the vessels were stiffer, then there might be less afferent firing for a given change in arterial pressure, less inhibition of sympathetic outflow, and less augmentation of vagal tone. In this context, do stiffer vessels contribute to the increases in baseline sympathetic traffic seen with aging, and do they also play a role in the reduced heart rate variability seen with aging?13,14

In summary, Tanaka and colleagues1 have demonstrated that regular physical activity appears to slow the normal loss of elasticity and compliance in the human cardiovascular system. They have also demonstrated that acute exercise interventions similar to those advocated as part of the Healthy People 2000 Program can reverse some of the age-related declines in arterial stiffness. Thus, physical activity and exercise training appear to modify another independent risk factor for cardiovascular disease. This means that physical activity has now been shown to have positive effects on blood lipids, glucose tolerance, coronary collateralization, and a variety of other risk factors, and that arterial stiffness appears to be another “modifiable” risk factor.15,16

In a larger context, aging and perhaps obesity are 2 of the biggest emerging challenges to public health in developed countries, and physical activity (of almost any type) appears to operate at multiple levels to limit their impact on the cardiovascular system.15,16 Although the attention paid to the molecular basis of disease and issues related to how best to apply our vast technological resources to treat disease is clearly warranted, the study by Tanaka et al1 is another example of why we must continue to urge our patients and the public at large to increase their level of physical activity and modify their lifestyles.17

References

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