S-Ⅱ-3 The adaptation of cardiovascular system to endurance exercise training: Implications for orthostatic tolerance.

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Endurance training results in central and peripheral adaptations to improve exercise performance. For example, endurance exercise training, which delivers an acute volume load to the heart during exercise, alters the compliance characteristics of the left ventricle. This adaptation of left ventricle of heart should be beneficial for performing exercise, because of allowing for increased diastolic reserve and therefore increased stroke volume and cardiac output in response to increased filling pressure. In addition, athletes are commonly assumed to be able to tolerate many kinds of physiological stressors as well as exercise better than non-athletes. However, it has been reported that endurance athletes have a high incidence of orthostatic intolerance. The physiological adaptation to endurance exercise training may be a disadvantage for gravitational stress or orthostasis. Successful integration of multiple elements of cardiovascular control is indispensable for tolerance to orthostatic stress. Indeed, it has been reported that orthostatic intolerance primarily attributed to abnormalities in both cardiac and peripheral vascular baroreflex regulation. For instance, Levine et al. (Circulation 1991) demonstrated that athletes had steeper slopes of their stroke volume (SV)/pulmonary capillary wedge(PCW) pressure curves with greater ventricular diastolic chamber compliance and distensibility than non-athletes. Thus, athletes have larger absolute and relative changes in end-diastolic volume over an equivalent range of filling pressures compared with non-athletes. In addition, the slope of the steep, linear portion of this curve correlated significantly with the duration of LBNP tolerance (an index of orthostatic tolerance). However, the mechanism of endurance-exercise training induced-orthostatic intolerance has not been fully understood from these previous findings.

S-Ⅱ-4 **Effect of aerobic exercise training on arterial stiffness and endothelium-derived vasoactive factors**

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Arterial stiffness increases with advancing age. Increased arterial stiffness has been established powerful and independent risk factor for cardiovascular disease. Therefore, the prevention and treatment of arterial stiffness are of paramount importance. We have investigated the effects of aerobic exercise training on arterial stiffness. Aerobic exercise training reduces arterial stiffness. However, the mechanism underlying aerobic exercise training-induced decrease in arterial stiffness has not been fully elucidated. The adaptation of arteries to aerobic exercise training may, at least partly, be regulated by the endothelium. We have focused on endothelium-derived vasoactive factors such as endothelin-1 (ET-1). ET-1 exerts potent vasoconstrictor and proliferative effects in vascular smooth muscle cells. Using the pharmacological approach, we determined whether ET-1 was involved in the mechanism underlying the influence of aerobic exercise training on arterial stiffness. We found that aerobic exercise training decreased arterial stiffness. The improvement in arterial stiffness was associated with decreased plasma ET-1 concentration. Additionally, decreases in arterial stiffness observed with the ET-receptor blockade before the exercise training were abolished after the exercise training. These results suggest that ET-1 plays a role in the mechanism underlying the exercise training-induced decrease in arterial stiffness.

Key Words: arterial stiffness, aerobic exercise training, endothelin-1