Can Aerobic Exercise Training Be Hazardous to Human Vessels?

To the Editor:

Exercise training is widely used as an efficient method of rehabilitation from or prevention of cardiovascular diseases. Improvement in endothelium-dependent vasodilation could be one of the underlying mechanisms of this effect, as was recently suggested by Higashi et al.\(^1\) Because the intensity and duration of training are directly related to performance improvement, it could be expected that highly trained athletes would be at a particularly low risk of vascular disease. Therefore, the recent description of endofibrosis (an original arterial disease affecting mainly highly trained cyclists) is puzzling.\(^2\) Endofibrosis occurs principally in the external iliac artery and is characterized by the progressive thickening of the endothelial arterial wall by fibrosis. Mechanical trauma, jet lesions, and local shear stress mechanisms have been suggested as causes, but they do not explain the preferential localization of the lesions. The affected athletes generally have no known cardiovascular risk factors. Further, histological examination rarely reveals lipid inclusions in the lesions, even in cases of extremely delayed diagnosis, suggesting that these lesions are not early atherosclerotic lesions. Finally, the relationship between endofibrosis and either metabolic mutations (eg, homocystinuria), or illegal drug use (eg, erythropoietin or growth hormone) is unclear. Because endofibrosis seems to be related to the level and duration of training (the disease is rare in athletes before they have cycled a total of 120 000 to 150 000 km), it is interesting to note that a recent study showed decreased endothelium-dependent vasodilation in humans after intense training,\(^3\) in contrast to the increase reported by Higashi et al.\(^1\) Thus, although aerobic exercise may augment endothelium-dependent vascular relaxation, “excessive,” or at least intense, exercise training could be hazardous to human vessels. Finally, endofibrosis is a unique clinical model of vascular impairment resulting from intense exercise training.

Pierre Abraham, MD, PhD
Jean-Louis Saumet, MD, PhD
Bénédicte Desvaux, PhD
Béréngère Fromy, PhD
Laboratory of Vascular Investigations
University Hospital Angers, France


Response

Regular physical exercise reduces cardiovascular morbidity and mortality in the general population. Exercise-induced augmentation of endothelium-dependent vasodilation through an increase in nitric oxide may, at least in part, contribute to antiatherogenic and antihypertensive mechanisms of exercise.\(^1\) However, we agree with Dr Abraham and colleagues that we must keep in mind the intensity, duration, frequency, and kind of exercise and that intense exercise training should be avoided because excess exercise can be hazardous to human vessels.

Bergholm et al\(^2\) reported that 12 weeks of intense physical training, consisting of four 1-hour training sessions per week at an intensity of 70% to 80% of maximal oxygen consumption, decreased circulating antioxidants, such as uric acid, sulfhydryl groups, a-tocopherol, beta carotene, and retinol and decreased acetylcholine-induced vasodilation in forearm vessels in normal men. If the same exercise regimen is performed by hypertensive patients, their blood pressure may be elevated in association with these unfavorable effects. In addition, we also think that endofibrosis is a unique clinical model of vascular impairment resulting from excess exercise training, although the pathophysiology of this disease and its possible relationship with endothelial function are unknown.\(^3\)

These findings suggest that long-term high-intensity exercise training may impair endothelial function through a decrease in antioxidants, an increase in free radicals, and a change in vascular structure, resulting in a reduction in nitric oxide release. Thus, we recommend a moderately intense exercise training program consisting of 30 to 60 minutes of walking, swimming, or bicycling, performed ≥3 times per week at an intensity of 50% to 60% of maximal oxygen consumption. Most individuals can safely and regularly perform such a level of physical activity.

A special committee on hypertension has confirmed the beneficial effects of moderate-intensity exercise.\(^4\) It has also been shown that high-intensity exercise generally does not alter blood pressure or cause blood pressure elevation.\(^5\)

Further investigation should be performed to determine the grade dependence of intensity and duration of exercise on endothelial function and blood pressure in individuals, including patients with essential hypertension.

Yukihito Higashi, MD, PhD
Shota Sasaki, MD
Satoshi Kurisu, MD
Atsunori Yoshimizu, MD
Nobuo Sasaki, MD
Hideo Matsuura, MD, PhD
Goro Kajiyama, MD, PhD
First Department of Internal Medicine
Hirosima University School of Medicine
Hirosima, Japan

Tetsuya Oshima, MD, PhD
Department of Clinical Laboratory Medicine
Hirosima University School of Medicine
Hirosima, Japan

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