Can Aerobic Exercise Training Be Hazardous to Human Vessels?

To the Editor:

Exercise training is widely used as an efficient method of rehabilita-
tion 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 Endofibro-
sis 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 atheroscle-
rotic 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 relax-
lation, “excessive,” or at least intense, exercise training could be
hazardous to human vessels. Finally, endofibrosis is possibly a unique
clinical model of vascular impairment resulting from intense exercise
training.

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endothelium-dependent vascular relaxation in normotensive as well as
hypertensive subjects: role of endothelium-derived nitric oxide. Circu-
decreases circulating antioxidants and endothelium-dependent vasodi-

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 al2 reported that 12 weeks of intense physical training,
consisting of four 1-hour running sessions per week at an intensity of
70% to 80% of maximal oxygen consumption, decreased circulating
antioxidants, such as uric acid, sulphydryl groups, a-tocopherol, beta
carotene, and retinol and decreased acetycholine-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 phys-
Iopathy 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 consist-
ing 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.

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