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Cardiovascular adaptations and exercise

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Abstract

Chronic endurance training increases an athlete's aerobic capacity and results in improved cardiocirculatory work economy, maximum performance, and enhanced oxygen uptake. The extent of the adaptation depends on individual factors such as frequency, intensity, and duration of training, muscle fibre type, and genetics. The functional range of heart rate, cardiac contractility, diastolic function, and blood pressure increases, while cardiocirculatory work is more economical, and maximum oxygen uptake (VO₂max) is improved.

Symmetric enlargement of the heart begins when endurance training exceeds individual functional limits, resulting in both left and right ventricular and atrial hypertrophy in response to the increased workload placed on the heart. Routinely examined by echocardiography, the myocardial hypertrophy is related to the increase in the interior end-diastolic volume. In contrast to pathological forms of cardiac wall hypertrophy, the mass/volume ratio, and therefore the maximum systolic wall stress remains constant. In addition, functional and structural changes occur in the vascular system.

Cardiac adaptations to exercise, including function and size of the heart, regress in healthy people who become inactive and have no structural heart disease. **Keywords:** physical activity, cardiovascular adaptation, athlete's heart, performance

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Introduction

For thousands of years, hunting and gathering was the most important subsistence strategy for the human race. Therefore the human cardiovascular system was designed to cope with much higher workloads than are imposed by the prevailing sedentary lifestyle of modern society.

From a physiological perspective, a healthy sedentary person may be regarded as "maladapted" with respect to the human heritage. The initiation and continuation of regular prolonged physical exercise, particularly endurance exercise, is associated with favourable adaptations within the human cardiovascular system and should promote optimum health. The cardiovascular adaptations include both functional and structural alterations in the heart, arteries, veins, and muscles^{6,14,19,12}. The final result of these adaptations is mainly an increase in maximal oxygen uptake and improved physical performance. There is evidence that functional adaptations occur prior to structural adaptations. Chronic dynamic training, in contrast to static or motor training, has the

greatest effect on the functional regulation or structural adaptations within the cardiovascular system⁶.

Mechanisms and regulation of functional adaptations

Regular dynamic (aerobic) physical exercise leads to typical adaptations in skeletal muscles including an increase in the number and size of mitochondria, the activity of enzymes in the oxidative pathway, in the muscle capillary density, and in specific muscle fibre types. An increase in parasympathetic activity can be observed during resting conditions in trained individuals depending on their genetic predisposition, the basal tonus, and their amount of physical training. Similarly, in the course of a graded exercise test, the increase in work-related sympathetic activity occurs later in endurance trained athletes.

The enhanced parasympathetic tone decreases cardiac excitation, atrioventricular conduction, and increases tolerance to orthostatic stress²¹. This regulatory adaptation is associated with a higher diastolic filling rate, a higher maximal blood flow velocity of early



diastolic passive left ventricular filling, and a higher early diastolic filling fraction at rest and during exercise^{8, 18, 21}. The associated improved performance is mainly shown during exercise and in the upright position: On a sub-maximal level, the heart rate of endurance trained subjects is lower than in untrained controls despite a comparable cardiac output. It has been shown that there is no significant difference in maximal heart rate between untrained and endurance trained subjects¹⁶; therefore, cardiac output and oxygen uptake is clearly higher in trained subjects during maximal performance due to increased ventricular volume. This is further pronounced by the higher arterio-venous oxygen difference in the skeletal muscles of trained individuals.

The adaptations within the autonomous nervous system can mainly be observed following dynamic exercise. Athletes who are statically trained have no clear evidence of increased parasympathetic tone. It has been found that static training reduces the sympathetic tone at a given exercise intensity, but this has not been associated with improved cardiovascular hemodynamics⁶.

Mechanisms and regulation of structural adaptations

Structural and electrophysiological myocardial adaptations occur when dynamic training exceeds 3-4 hours/week. These adaptations, also called the "athlete's heart," produce enlargement of the heart cavities, hypertrophy of the myocardium, and an increase in parasympathetic tone^{14, 18}. There is still an ongoing debate regarding the physiological nature of the athlete's heart^{10, 15, 17}. Recently, it has been hypothesised that the myocardial functions and dimensions in some elite endurance athletes are on the cusp of cardiomyopathy⁹. Nevertheless, there is no data indicating an increased cardiac mortality in these athletes and no evidence to suggest that former endurance athletes have an increased risk for heart failure⁴. In addition, the risk for ventricular ectopy was not related to the amount of physiological left ventricular hypertrophy.

The greater left ventricular wall thickness and mass may be caused by the increased volume load on the left ventricle following extensive endurance training, and associated with the higher plasma volume in endurance athletes. In addition to the volume and intensity of training, the extent of the myocardial adaptation and hypertrophy is influenced by genetic predisposition³. Therefore the identical training programme may lead to divergent adaptations for individual athletes.

The structural adaptations occur in all four heart chambers (Figure 1). In contrast to pathological myocardial alterations, the normal exercise-related hypertrophy of the heart walls is paralleled by an increase in ventricular diameter and volume. Therefore the ratio between muscle mass and heart volume remains constant and the systolic function is not impaired. The exercise-induced myocardial adaptation is an eccentric hypertrophy with a concomitant symmetrical increase in muscle mass and wall diameter. Any form of concentric hypertrophy is incompatible with the "athlete's heart" designation^{6, 11}. Several lines of cross-sectional evidence suggest that the structural increase in heart volume is limited to 17-20ml/kg bodyweight in male athletes (normal range in untrained: 10-12ml/kg bodyweight) with 10-15 % less in female athletes.



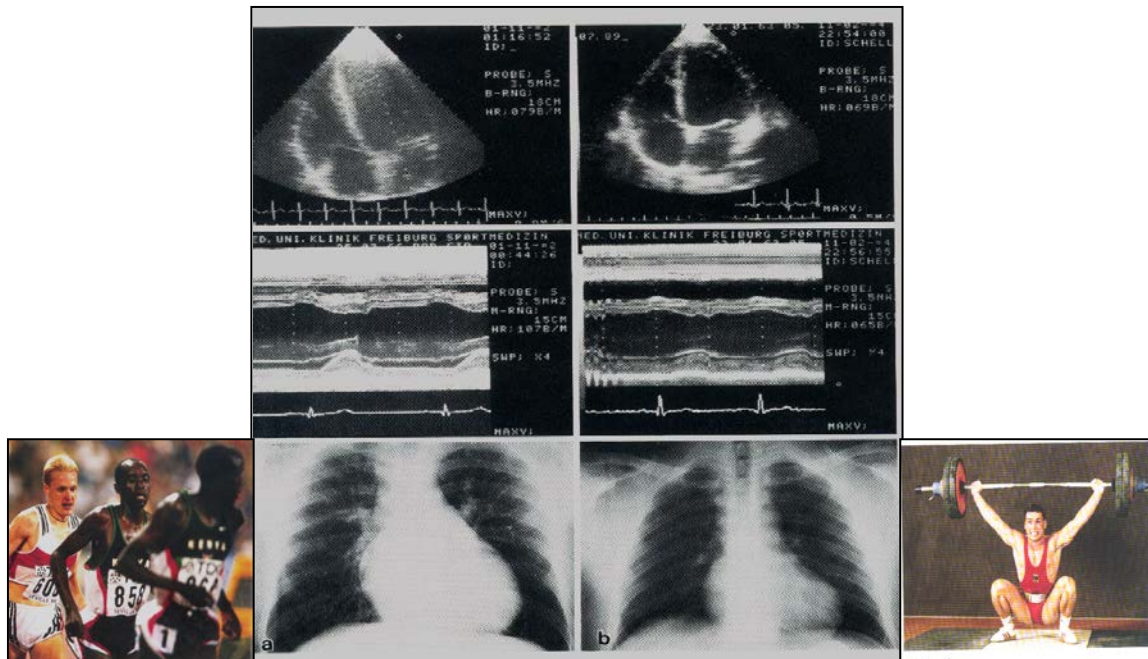


Figure 1: Typical findings in a world-class endurance athlete (left) and power athlete (right)

- (a) Four chamber view, two-dimensional echocardiography
- (b) short-axis magnetic resonance tomography
- (c) anterior-posterior X-ray

Echocardiographic investigations have shown that the increase in both end-diastolic diameter and wall thickness of the left ventricle does not extend beyond 20% of initial values. Therefore by considering bodyweight, absolute measures do not exceed 55-63mm left ventricular end-diastolic dimension (LVEDD), 12-13mm wall thickness of the septum and 11-12 mm of the posterior wall^{10,14}. The upper limit of left ventricular myocardial mass should not exceed 3.5g/kg bodyweight (critical left ventricular weight)^{1,18}. Beyond this threshold, it can be assumed that physiological myocardial hypertrophy has taken place, but not myocellular hyperplasia. Together with the functional adaptations, the structural enlargement of the heart further increases cardiac output and maximal oxygen uptake. From a technical viewpoint, the athlete's heart works with a higher engine displacement and reduced revolution speed.

The structural myocardial adaptations are only

found following chronic dynamic training. Static training, even in world class champions of various disciplines, has not induced comparable myocardial alterations⁶. Structural, mainly concentric myocardial alterations or left ventricular relaxation properties, found in strength-trained athletes are indicative of hypertension or may be related to the intake of anabolic steroids^{11,12}.

Myocardial adaptations are not limited to a specific age group, and have been documented both in children (age 9-10- years) and in senior athletes (age 60-years and older). The myocardial hypertrophy and enlargement of the cavities in these age groups may be less pronounced than in athletes aged 20-35-years, but this is most likely related to a lower training volume and intensity than to a reduced ability for cardiac adaptation.

Concurrent with the myocardial adaptations, the function and structure of the conduit arteries of

feeding skeletal muscles adapt to regular dynamic training. The vessel diameter increases and endothelial function improves (Figure 2)^{13, 19}. Exercise-induced increases in blood flow lead to an increased shear stress, release of nitric oxide (NO) and other vascular endothelium-derived factors, such as endothelin-1. It can be assumed that exercise-related improvements in endothelial function are also partially responsible for the preventive effect of exercise on atherogenesis⁵.

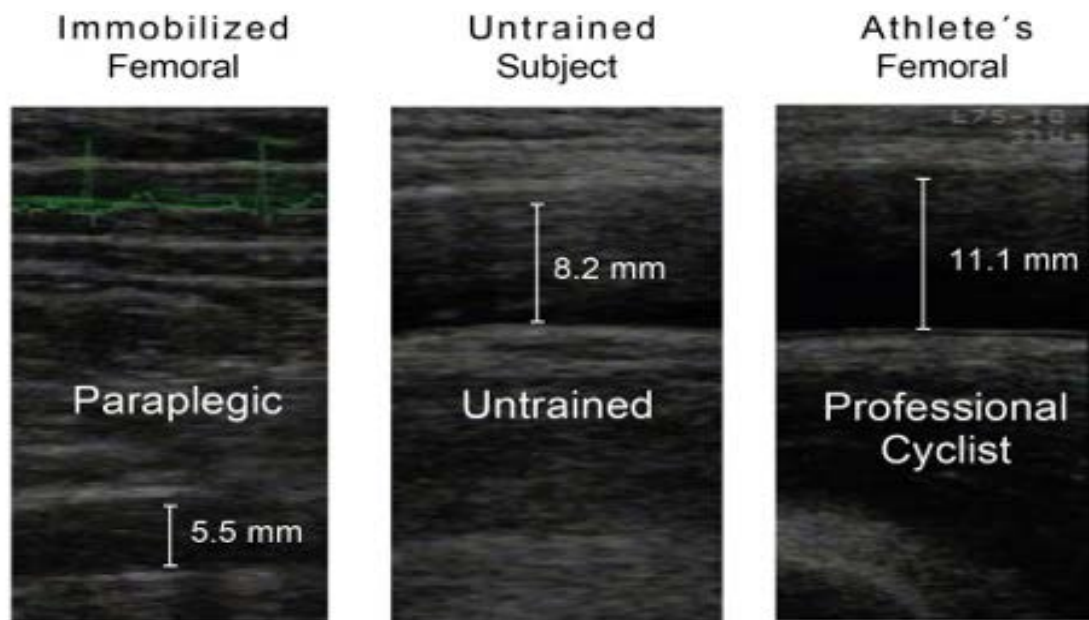


Figure 2: Ultrasonical measured diameter of the femoral artery of a professional cyclist: An untrained subject and a paraplegic¹⁹

Long-term reversibility and possible functional impairment

Following the suspension of high volume endurance training, the heart shows cardiac remodelling with partial or total regression of structural and functional adaptations. Under complete rest conditions, this process can be detectable after a few weeks. The degree and speed of cardiac training changes seems to be related to the duration and extent of the pre-existing myocardial adaptations. Following decades of endurance training, cardiac remodelling is often incomplete. This, however, is not associated with an impaired cardiovascular function^{2, 15}.

A sudden and complete interruption of training

may be associated with disturbances within the autonomic nervous system leading to benign rhythm disturbances. These findings usually disappear after several weeks or resumption of training^{1,2,10}.

In recent years, new biomarkers have improved the diagnosis of myocardial damage. At present, cardiac troponin I (cTnI) or cardiac troponin T (cTnT) are the new “gold standards” in the diagnosis of myocardial damage. They are highly cardio-specific and do not show cross-reactivity with troponin from skeletal muscle fibres. Several studies reported an increase in cardiac troponins following strenuous endurance exercise in symptomless athletes with no clinical history of cardiovascular diseases^{7, 20}. However,

elevated post-exercise troponin concentrations were detectable in only a minority of subjects investigated. Although elevated troponin concentrations after endurance exercise can exceed acute myocardial infarction limits, they are usually of short duration and reach normal values within 24- to 48h. At present, it is generally believed that increased post-exercise concentrations of cardiac troponins are not a mirror of substantial myocardial necrosis but rather reflect a reversible membrane leakage of the free cytosolic pool²⁰. However, more carefully designed investigations, including concomitant complete cardiologic follow-up examinations, in addition to cardiac histological and molecular studies in animal models, are required to further elucidate the association between strenuous exercise, concentrations of cardiac biomarkers, and myocardial damage.

Nevertheless it should be emphasised that from an epidemiological perspective, endurance-trained athletes do not have a reduced life span and show no increased prevalence of heart disease, particularly heart failure⁴. Regular aerobic exercise is likely to be the norm for humans and should be used to promote healthy lifestyles for all.

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