Palestrica of the third millennium – Civilization and Sport
Vol. 18, no. 4, October-December 2017, 210–214

REVIEWS

Arterial stiffness in athletes

Rigiditatea arterială la sportivii de performanţă

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Abstract

Competitive athletes are exposed to intensive and prolonged training which may determine structural and functional adaptations in the arterial system. Arterial stiffness is a characteristic of the aging process and an important predictor of cardiovascular morbidity and mortality. Hemodynamic stress induced by intensive training may influence arterial elastic properties. This article synthesizes the results of studies evaluating arterial stiffness parameters at rest and after acute bouts of exercise in athletes performing various types of intensive training programs.

In predominantly endurance trained subjects, some of the studies indicated reduced arterial stiffness and central pressures, while others showed no differences between the athletes and controls. Acute bouts of exercise increase pulse wave velocity and decrease central pressures. However, in ultra-marathon runners, decreased systemic compliance after the race, compared to basal values, was reported.

The differences between studies may be determined, at least partly, by the type, intensity and duration of physical training. The various methods used to measure arterial parameters in these studies make difficult the comparison between their results.

The mechanisms of vascular distensibility adaptation during intensive training are not yet elucidated, but hemodynamic factors, oxidative stress and systemic inflammation are discussed.

Keywords: arterial stiffness, central arterial pressures, competitive athletes.

Rezumat

Sportivii de performanţă sunt expuşi unui efort intens şi prelungit care poate duce la modificări adaptative structurale şi funcţionale ale sistemului arterial. Rigiditatea arterială este o caracteristică a procesului de îmbătrânire şi un predictor independent al morbidităţii şi mortalităţii cardiovasculare. Stresul hemodinamic indus de antrenamentul intens poate influenţa proprietăţile elastice ale arterelor.

Acest articol sintetizează rezultatele studiilor care au evaluat parametrii de rigiditate arterială atât în condiţii bazale, cât şi în urma efortului acut, la subiecţi aflaţi în programe diferite de antrenament.

La sportivii care desfăşoară activităţi în care predomină efortul aerob, unele studii au constatat o reducere a rigidităţii arteriale şi a presiunilor arteriale centrale, în timp ce altele nu au identificat diferenţe semnificative între sportivi şi lotul de control. Efortul acut a dus la creşterea vitezei undei de puls şi la reducerea presiunilor centrale la sportivii de performanţă. Cu toate acestea, la ultra-maratonişti, s-a constatat o scădere a complianţei arteriale sistemice la sfârşitul cursei, comparativ cu valorile iniţiale.

Mecanismele adaptării distensibilităţii arteriale la sportivii de performanţă nu sunt încă elucidate, intrând în discuţie rolul factorilor hemodinamici, al stresului oxidativ şi al inflamaţiei sistemice.

Cuvinte cheie: rigiditatea arterială, presiuni arteriale centrale, sportivi de performanţă.

Received: 2017, July 14; *Accepted for publication:* 2017, August 20
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https://doi.org/10.26659/pm3.2017.18.4.210

Introduction

It is now accepted that the cardiovascular system undergoes structural and functional changes during athletic adaptation. Athletic heart is characterized by an increase in ventricular mass and chamber dimensions, after prolonged and intense training (Green et al., 2012).

Over the last twenty years, researchers have given increasing importance to the study of arterial structure and function in athletes. It has been shown that both resistance and conduit arteries are enlarged in athletes, possibly due to increased shear stress from chronic hemodynamic overload (Green et al., 2012). A decrease in wall thickness of conduit arteries has also been reported as a remodeling process that accompanies prolonged training (Green et al., 2012; Tijssen et al., 2012). These morphological adaptations may impact upon arterial function (Green et al., 2012).

Arterial stiffness develops with aging as a result of a reduction in arterial distensibility. The increase in aortic pulse wave velocity (PWV), the gold standard measure of arterial stiffness, is an independent predictor of cardiovascular morbidity and all-cause mortality (Laurent & Boutouyrie, 2007).

Great arteries have two major hemodynamic functions: the conduit function, which delivers blood to peripheral organs, and the dampening function, which transforms the intermittent flow produced by left ventricular ejection into a continuous steady flow, which ensures peripheral tissue perfusion. Each ventricular ejection produces a forward pressure pulse wave that travels downward and reflects at the site of arterial bifurcations, producing backward waves that return toward the heart as reflected waves. The pressure wave is the result of the summation of the forward and the reflected wave. In elastic arteries, the reflected wave comes back to the heart during diastole, augmenting diastolic pressure. In stiff arteries, PWV increases and the backward wave arrives faster during ventricular ejection, augmenting central systolic pressure. Aortic stiffness, stroke volume and ejection velocity are the most important determinants of systolic and diastolic aortic pressures. The effects of the reflected wave on pressure wave amplitude are expressed as augmentation pressure or augmentation index (Aix). In peripheral arteries which are closed to the reflected sites, systolic pressure increases, leading to pulse pressure amplification (Avolio et al., 2009). In the presence of tachycardia, the duration of left ventricular ejection is reduced and the reflected wave reaches the ventricle during diastole. In case of bradycardia, the reflected wave comes back towards the ventricle during systole, increasing aortic systolic pressure and pulse pressure (London & Pannier, 2010). Pressure wave morphology and Aix index may also be influenced by the properties of small arteries, such as peripheral resistance and remodeling. It has been suggested that central blood pressures may be more important for patient prognosis than brachial pressures. In fact, central aortic pressure opposes left ventricular ejection and its increase may directly impact left ventricular function (Roman et al., 2007).

A great body of evidence indicates a favorable role of exercise, particularly aerobic exercise, on arterial stiffness. A recent meta-analysis concluded that aerobic exercise induces a significant improvement in aortic PWV and Aix. The favorable effect was more important in trials with intense and long duration interventions (Ashor et al., 2014).

While the favorable role of habitual exercise on vascular function has been documented, the effect of high intensity training as seen in competitive athletes is less well determined. The aim of this review is to present and analyze the results of studies evaluating the effects of sustained endurance and resistance exercise training on various measures of arterial stiffness.

Effects of various types of physical training on arterial stiffness

A number of studies evaluated arterial stiffness and wave reflection parameters, using various methods, in endurance- and/or resistance-trained subjects, and compared them with measures obtained in recreationally active or sedentary controls.

In one study, which included young healthy endurance-trained subjects and recreationally active controls, the authors determined central pressures (systolic, diastolic and pulse pressure) and pulse pressure amplification using the analysis of the central pulse pressure wave derived from the radial wave. The results indicated that endurance-trained athletes had lower wave reflection and systolic load compared to controls of similar age, with similar anthropometric parameters and peripheral blood pressures. The study also found a decreased tension time-index, which is related to systolic load and heart oxygen consumption, and an increased index of subendocardial viability, which reflects a better subendocardial perfusion, in athletes compared to controls. Athletes had significantly reduced heart rate, but the adjustment of Aix for heart rate did not alter the initial results (Edwards & Lang, 2005).

Another study, which included 212 young endurance athletes, found a reduced aortic PWV in athletes compared to controls. The evolution of aortic stiffness with age showed that PWV in athletes remained inferior to that of controls before the age of 30 years, but after the third decade of life, the progression of arterial stiffness was similar in both groups. The attenuation of vascular stiffness with age, due to aerobic training, seems to disappear after a certain age, probably because of sustained mechanical stress of arterial wall elastic fibers (Maldonado et al., 2006).

An unfavorable vascular hemodynamic profile was found in a study that evaluated endurance athletes aged 24 to 55 years in comparison with sedentary controls matched for age, height, brachial systolic and mean blood pressure. The authors reported significantly reduced heart rate and an increase in carotid systolic pressure and pulse pressure, as well as a reduction in pulse pressure amplification in athletes. The increase in systolic load observed in this study was explained by the reduced heart rate and by a possible increase in stroke volume and ventricular ejection. The authors could not explain the augmentation in central systolic load in the context of the well-known favorable cardiovascular effect of aerobic exercise (Laurent et al., 2011).

Some other studies reported no differences in aortic

PWV between endurance athletes and healthy non-athlete controls (Dulai et al., 2011; Rátgéber et al., 2017).

The effects of intense resistance training on arterial compliance were also evaluated, but the results were contradictory (Miyachi, 2013).

In one study that compared endurance-trained athletes, resistance-trained athletes and sedentary controls, aortic PWV decreased and systemic compliance increased in endurance-trained subjects compared to controls, while in resistance-trained athletes, aortic PWV increased and systemic compliance decreased compared to healthy controls. Vascular changes were accompanied in resistance-trained subjects by an increase in endothelin-1 concentrations. Arterial stiffness was associated with plasma endothelin-1 levels, independently of blood pressure values. Nitric oxide did not change significantly (Otsuki et al., 2007). Entothelin-1 is a potent vasoconstrictor which contributes to basal vascular tone and stimulates proliferation of smooth vascular cells (Miyauchi & Masaki, 1999). Increased vascular tone and smooth vascular cell proliferation may increase arterial stiffness. Thus, endothelin-1 may be involved in arterial stiffness alterations during exercise.

Carotid-femoral PWV and carotid-brachial PWV were measured in basketball players (who mainly performed aerobic exercise) and weight lifters (who were resistance-trained). In both groups, the authors reported reduced arterial stiffness compared to controls (Saka et al., 2016). Similar results were found in another study which included resistance-trained participants, endurance runners and healthy controls. Aortic PWV was lower in both endurance and resistance-trained subjects. Long resistance training was accompanied by an increase in pulse pressure amplification and a decrease in central augmentation pressure (Morra et al., 2014).

In ultra-endurance athletes (16.3±3.7h/week) who may have a greater cardiovascular risk due to prolonged and intense cardiovascular strain, the measurement of central pressures and subendocardial viability index revealed no differences in Aix, brachial and central pressures between athletes and controls. However, athletes had an increased subendocardial perfusion capacity, while the ejection duration was prolonged (Knez et al., 2008). This finding which was also reported by Edwards and Lang in competitive endurance-trained athletes, as mentioned above, suggests a favorable adaptation to increased hemodynamic stress.

In marathon runners, however, an increased baseline aortic stiffness was reported compared to controls (Kröger et al., 2010; Vlachopoulos et al., 2010). A U-shaped relationship between the intensity of aerobic training and arterial stiffness during vascular adaptation was proposed. Arterial stiffness decreased progressively in physically active and endurance-trained subjects, and increased constantly in marathon and ultra-marathon runners (Sardeli & Chacon-Makahil, 2016).

In high-intensity strength- and endurance-trained young elite rowers, examined by high-resolution magnetic resonance imaging, endothelial function and aortic distensibility were normal. Arterial remodeling leads to reduced aortic diameters and increased areas of peripheral arteries. The authors concluded that the favorable effects of training on vascular function are only temporary (Petersen et al., 2006).

The effects of acute bouts of exercise on vascular stiffness and wave reflection have been evaluated in several studies. Intense acute exercise may be used to test cardiovascular adaptation to the acute increase in hemodynamic strain.

In one study, 16 healthy male endurance-trained subjects (football and water polo players) younger than 25 years underwent a submaximal test, based on cycling during 30 min. Vascular parameters (aortic PWV, central and peripheral blood pressures, Aix adjusted for 75 b/min, pulse pressure amplification, ejection duration, and the subendocardial viability ratio) were measured before and 15 min after the test. The results indicated an increase in pulse pressure amplification and a decrease in the ejection duration. The subendocardial viability ratio was not reduced, suggesting good myocardial vascularization (Staniszewska et al., 2016). While ventricular contraction increases during acute exercise, good endocardial perfusion may represent an adaptation process which ensures an optimal oxygen supply during exercise.

Carotid stiffness was measured in young competitive basketball players and in healthy controls, at rest and after a bout of acute cycling exercise. Basketball players are exposed to both resistance and endurance training. In resting conditions, athletes had decreased stiffness and augmented diameters of the carotid artery. After four bouts of cycling, carotid stiffness in sedentary controls increased, while the vascular parameters of athletes remained unchanged. Moreover, carotid systolic and mean blood pressures increased significantly less in basketball players than in controls. These results suggest an increased elasticity of the carotid artery reflecting an adaptation to intense exercise. In contrast, oscillatory shear stress was increased in athletes under both rest and post-exercise conditions (Liu et al., 2015). The vascular consequences of oscillatory stress are not entirely elucidated. However, some data indicate its possible proatherogenic role (Newcomer et al., 2011).

The effect of acute moderate exercise was evaluated by Dulai et al., who compared endurance-trained with resistance-trained subjects and sedentary controls at rest and after acute exercise. They found no differences in PWV values between the groups at rest, while after exercise, PWV and systolic blood pressures increased in endurance athletes, but not in controls and resistance-trained athletes (Dulai et al., 2011). Very young sportsmen, aged 11-16 years, were compared to sedentary controls at rest and after dynamic and isometric exercise. At rest, the authors found no differences in PWV and Aix between athletes and controls. However, a slight but not statistically significant decrease in Aix was found in sportsmen. After dynamic exercise, a significant increase in aortic PWV was seen in the oldest group (15-16 years). The authors also reported a decrease in Aix in the same age group, which did not reach statistical significance (Rátgéber et al., 2017).

The increase in aortic PWV observed after exercise may be especially explained by the elevation of systolic pressure and heart rate. The decrease in peripheral vascular

resistance may predominantly influence Aix. The values of Aix tend to decrease after aerobic exercise.

The acute effects of ultra-marathon (a mountain trail running ultra-marathon) were also assessed by determining systemic (small and large artery) compliance, before and after the race. The results indicated that aerobic exercise for 20-40 hours induced a post-race reduction in large artery compliance. The increase in large artery stiffness was more important in subjects with more elastic arteries at baseline (Burr et al., 2012).

Increased oxidative stress (Skendery et al., 2008) and systemic inflammation (Kim et al., 2007) reported in ultra-marathon runners may be responsible for the alteration in endothelial function and sympathetic control of vascular tone, both mechanisms being involved in arterial structure and function alteration. It is considered that arterial stiffness may be an important cause of myocardial fibrosis identified in lifelong endurance athletes (Wilson et al., 2011).

Mechanisms involved in arterial stiffness modulation during intensive training

Regular aerobic exercise has been shown to be protective for arteries and to enhance arterial distensibilty (Ashor et al., 2014), while the effect of resistance training on arterial structure and function remains controversial (Miyachi, 2013). However, the effects of resistance training on metabolic factors may induce an indirect vascular benefit ( ).

The mechanism that modulates arterial stiffness in athletes is complex and incompletely elucidated. Changes in hemodynamic parameters of blood flow, blood pressures and pulsatility during exercise may induce local vascular processes that protect against atherosclerosis (Newcomer et al., 2011).

One possible favorable effect may be an increase in the mean shear stress due to the increase in blood flow and augmentation in nitric oxide synthesis associated with the reduction in nitric oxide inactivation. Suppression of local angiotensin-converting enzyme may also contribute to the improvement of endothelial function (Rieder et al., 1997). Plasma endothelin-1, which is decreased in endurance-trained subjects and increased in resistance-trained subjects, may be another contributor to vascular adaptation (Otsuki et al., 2007). Exercise also increases oscillatory shear stress, which may have deleterious vascular effects, inducing a proatherogenic profile. It has been shown that oscillatory shear stress stimulates adhesion molecule expression, decreases nitric oxide synthesis and enhances proliferation of vascular smooth muscle cells. It is considered that different patterns of shear stress appear during exercise, with a possible different impact on endothelial nitric oxide release and vascular adaptation (Newcomer et al., 2011).

The decrease in sympathetic tone which has been documented in athletes may contribute to peripheral vasodilatation and reduce arterial stiffness (Boutouyrie et al., 1994).

Oxidative stress and systemic inflammation are two well-documented mechanisms involved in arterial wall stiffness (Patel et al., 2011; Park & Lakatta, 2012). The anti-inflammatory and anti-oxidative effects of physical exercise, previously documented (reviewed in Ashor et al., 2014), may also contribute to the favorable vascular profile of athletes. However, in ultra-marathon runners, excessive inflammatory and oxidative reactions may be the cause of the increased arterial stiffness observed in these athletes (Skendery et al., 2008; Kim et al., 2007, Burr et al., 2012).

Conclusions

1. The arterial system engenders a complex adaptation in competitive athletes. In predominantly endurance-trained subjects, some studies indicated reduced arterial stiffness and central pressures, while others showed no differences between athletes and controls.

2. Few data indicated a positive role of resistance training. The discrepancies between studies may be determined, at least partly, by the type, intensity and duration of physical training. Moreover, the various methods used to measure arterial parameters might also have contributed to these differences.

3. The mechanisms involved in vascular stiffness response in competitive athletes are complex and incompletely understood. Hemodynamic stimuli, systemic inflammation and oxidative stress seem to be important contributors to arterial adaptation during competitive training.

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