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Is The Exercise-Induced Increase in Central Arterial Stiffness a Risk Factor for Health?

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Abstract

Central arterial stiffness (CAS) is an independent predictor of cardiovascular mortality. If in one side, exercise training can reduce CAS and protect health; on the other side, CAS might be increased in individuals with very strenuous training routines. This chronic increase in CAS is observed acutely, right after a strenuous exercise session. Both chronic and acute increases in CAS are associated with cardiovascular risk factors such as left ventricle hypertrophy, reduction in baroreceptor sensitivity, increased aneurism formation and stroke incidence; besides, they contribute to hypertension, myocardial infarction, congestive heart failure, dementia and atherosclerosis. However, there are many reasons to believe that trained individuals are protected by other training adaptations and that increased CAS would not be too dangerous in these cases. Nevertheless, even when CAS is increased through exercise training, other harmful adaptations (e g, left ventricle hypertrophy) are aroused together. After debating these questions along the text, it was concluded that individuals should make a complete clinical check-up when decided to face strenuous training routines. A new debate is suggested: personalized exercise programs might be prescribed according to individual cardiovascular risks including CAS and the guidelines for exercise prescriptions proposed by health organizations need to attend these special cases.

Keywords: Exercise, Vascular Stiffness, Cardiovascular Physiology

1. Introduction

Since the present discussion is about exercise effects on arteries, it should be reminded that the structure and function of the central and peripheral arteries differ in different physiological and pathological adaptations in life. The different adaptations are also evident through exercise stimulus (1); therefore, the current study specifically focuses on central arteries: aorta and carotid.

Central arterial stiffness (CAS) is an independent predictor of cardiovascular mortality (2), and it is associated with many other risk factors (3, 4). The benefic reduction in CAS through endurance exercise interventions is highlighted in a variety of human population (5). A number of studies show that CAS is reduced in physically-active individuals compared to their sedentary counterparts and this reduction is more pronounced in endurance-trained individuals (6-12). Nevertheless, CAS is increased in marathon and ultramarathon runners compared to active individuals (13, 14) suggesting that the CAS adaptation can be represented by a U-curve, according to endurance exercise level (Figure 1).

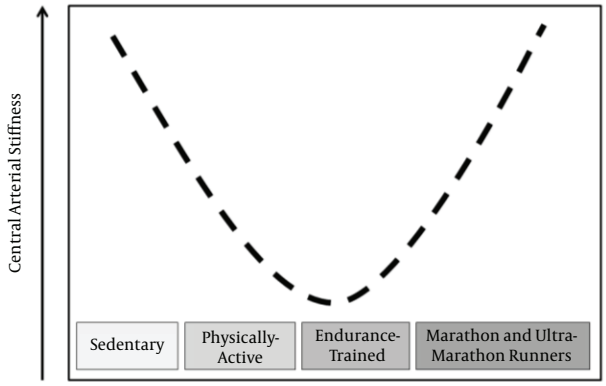

Figure 1. Proposed Scheme for Adaptations of Central Arterial Stiffness According to Endurance Training Level

Corroborating the chronic adaptations, many studies have showed an endurance exercise session reduce (15-17) or do not change of CAS acutely (18, 19). Furthermore, similar to the chronic effects, three studies showed increase in CAS after endurance exercise session when it was more strenuous (20-22). It is noteworthy that the malefic effects of endurance training over CAS are neglected, and the increased CAS in very highly endurance-trained individuals as well as its acute increments should be investigated. The

central arteries of these individuals are stiffer, even though they might have other optimal physiological adaptations. However, if it is a maladaptation or a protection to endure such strenuous training sessions will be discussed in the next paragraphs.

2. Arguments

2.1. Risks of Increased Central Arterial Stiffness

2.1.1. Acute Risks

Albert et al. (23), in a 12-year retrospective study, found that out of 122 sudden deaths (among 21,481 males) 18.8% happened during or 30 minutes after vigorous efforts. Acutely, exercise really increases the risk of cardiovascular events and the risk of sudden cardiac death might be increased in athletes from various modalities, due to higher frequency of exposure to strenuous exercise sessions (24, 25). Several physiological changes trigger sudden deaths during vigorous efforts, whether it would be related to known diseases or not. Among them, the following might be mentioned: increase in sympathetic outflow for heart and blood vessels, increasing electric instability and myocardial contraction strength; rupture of atherosclerotic plaques and appearance of embolisms through increase in blood pressure (BP), circulating catecholamines and vasomotor oscillations and rupture of aneurysms due to abrupt changes in BP (26).

Obviously, avoiding the CAS increase during acute efforts is not enough to protect from sudden death risk; therefore, it might reduce part of it. The stiffer the central arteries, smaller is the wall deformation and worse is the baroreceptor sensitivity. It enables higher BP variability, which in turn increases sympathetic outflow and circulating catecholamine levels, leading to a cardiovascular risk environment (23, 24).

There are two other factors that evince acute increase in CAS leading to immediate vulnerable state to myocardium. First, elevation in the left ventricle afterload requires higher myocardial efforts. Second, during vigorous efforts the increase in heart rate reduces diastolic duration, which concomitantly increases afterload, and impairs coronary irrigation.

Besides the immediate vulnerable state, the acute effects of exercise might also represent an opportunity to chronic adaptations. In the same way post exercise hypotension might predict chronic BP reduction (27), there are good reasons to believe that the sum of acute effects on CAS could lead to chronic adaptations in arterial wall.

It is interesting to notice that the rationale for this hypothesis mainly comes from the studies finding an increase in CAS following resistance training. Many researchers assume that acute changes in BP during exercise are the mechanical stimulus responsible for arterial wall remodeling they found following a period of training. To date, only Ozaki et al. (28) supported this relationship between acute stimulus and chronic adaptation, showing the higher BP peak during resistance exercise in the group increasing CAS following the role period of training. In this way, the acute intermittent elevations in BP that occur during resistance exercise, might overload the viscoelastic tissue of the arterial walls, and consequently alter the arterial wall structure and/or load-bearing properties (29).

Traditionally, during endurance exercise, BP does not reach such high levels as in resistance exercise. Otherwise, the two studies increasing CAS acutely after endurance exercise noticed that this increase was due to maximum BP levels during exercise sessions. Hu et al. (20) explained that it was due to very high intensities hit by one of the trial groups. Michaelides et al. (21), in spite of defending other mechanisms to explain CAS increase, also showed an association between higher BP values with higher duration of exercise session (post 24 hours) in sedentary elderly with coronary arterial disease. Other physiological mechanisms could prompt acute increase in CAS at strenuous exercise sessions such as: increase in inflammation and oxidative stress that in turn reduces the nitric oxide bioavailability and vasodilation (21, 30); and/or increases factors that modulate extracellular matrix turnover (31). Thus, independent of what is the stimulus for increased CAS after exercise session; this acute increase in CAS may mediate chronic adaptations. Even so, it is a huge matter of debate that needs to be proved in future researches.

2.1.2. Chronic Risks

Besides the acute stimulus to arouse chronic adaptations through exercise training, CAS would also be increased by other chronic physiological adaptation mediations. The hypervolemia and bradycardia induced by strenuous exercise training might overload arterial wall, leading to fatigue and fragmentation of elastin fibers (32), and consequently CAS increase. Exercise trainings that overpass the limits of individuals might incite several stimuli such as released chemotactic factors, onset of inflammatory process, increased sympathetic output and also prompt other types of unbalances, and in turn increase CAS.

Beyond the harmful consequences of acute increase in CAS, chronically, CAS is known as an independent predictor of cardiovascular mortality (2). Increased CAS is associated with the left ventricle hypertrophy, reduction in baroreceptor sensitivity, increased aneurism formation and stroke incidence, besides contribution to hypertension, myocardial infarction, congestive heart failure, dementia and atherosclerosis (33). Among all these cardio-

vascular risk factors, following exercise (resistance exercise), at least left ventricle hypertrophy is associated with CAS increase (34-37).

It should also be considered that the BP elevation is the higher risk factor for premature cardiovascular, cerebrovascular, renovascular, and other vascular diseases worldwide (38). It is possible, these cardiovascular risks related to high BP depend on the arterial stiffness and hypertension is only one of the consequences (39).

The accumulating evidence regarding the relationship between increased CAS and mortality risk is based on epidemiologic studies (i.e. individuals with higher CAS are prone to increased risk of mortality compared to the individuals with lower values) and not many studies are looking for consequences of CAS changes following exercise programs, mainly in apparently healthy people such as the exercise trained ones. It is interesting to notice that regarding CAS increase by resistance exercise in two studies (40, 41) when participants had baseline CAS values greater than normal reference (4), CAS increased following training, while it did not change in other studies (42-45) in which the participants' pre-training CAS values were within the normal range. In this way, neither resistance training nor strenuous endurance training should be prescribed for all. Instead of that, the accurate intensity volume and training variables should be planned individually as a personalized exercise program according to cardiovascular risks.

2.2. Benefits of Exercise Training

Even though the risks of high CAS were completely highlighted, the consequences of increased CAS through exercise training need to be discussed. Perhaps, such increased vascular risks are offset by other beneficial cardiovascular adaptations, at least following endurance training, since this type of exercise has increased life expectancy also in endurance athletes (46, 47). Anyway, the associations between the dose of exercise (i.e. intensity and volume during training and competition) and benefit of the adaptations acquired by exercise trained individuals, need to be better investigated. Furthermore, the increase in CAS through exercise training might be a protection to the high elevations in BP that arteries have to bear during this kind of strenuous exercise.

Numerous position stands recommend exercise training for diverse preventive and therapeutic healthy benefits, for different populations and different pathological conditions. They also show that acute risks from exercise sessions do not overcome exercise benefits, since people exercising more days per week reduce the risk of myocardial infarction (24, 48). Likely, it is due to prevention of lethal arrhythmia and ischemia by improved autonomic control and consequently reduction of electrical instability (48, 49). Nevertheless, it cannot be rejected that the aforementioned protective effect hypothesis could not be applicable to exercise trained individuals, mainly marathon and ultramarathon runners.

3. Conclusion

Increase in CAS, generated by illness or exercise training, could be a stimulus to increase left ventricular load and myocardial oxygen demands, and in turn compromising coronary irrigation, besides the fatigue on its own elastic components favor arterial dissection and rupture. Thus, CAS increases following exercise training despite being a possible protective mechanism during exercise, it can be pathophysiological during rest. In addition, as the risk to increase CAS seems to be higher in people with higher baseline CAS values, individuals should make a complete clinical check-up when they decided to face the strenuous training routines. A new matter of debate is suggested: personalized exercise programs might be prescribed according to individual cardiovascular risks including CAS, and the guidelines for exercise prescriptions proposed by healthy organizations need to attend these special cases.

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Footnotes

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