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Considerations on Cardiovascular and Musculotendinous Adaptation to Effort With Regards to Somatotropic Plasma Concentration

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Abstract

For cardiovascular and musculoskeletal adaptations at the effort, it is necessary to establish borders of passage from normal to pathology. The main hormone that modifies the morpho-functional changes of the myocardium and vascular vessels in order to increase the cardiac output, as well as the proportional increase of the striated muscles and the tendons of insertion is the somatotrope. When growth hormone secretion is excessive, cardiac hypertrophy, arrhythmias, and possibly aneurysms and vascular accidents may occur. If endocrine adaptation to effort occurs with a deficient plasma level of the somatotropic, the risk of tendon rupture and possibly of tendon pain increases. In order to prevent these conditions, it would be useful to monitor the somatotropic plasma concentration in athletes, with the possibility of intervention to increase or decrease it.

Keywords: Striated Muscles, Musculotendinous Adaptations, Myocardium

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Introduction

For sports medicine, it is essential to differentiate normal cardiovascular adaptation to effort from pathological conditions (Kovacs & Baggish, 2016). The limit between physiological and pathological can be easily overcome. From the point of view of ergophysiology, it is necessary to study the hormonal factors that influence cardiovascular adaptation to effort. This occurs in parallel with that of muscle tissue, to which must ensure a proper cardiac output.It is

known that somatotropin, also called growth hormone, is secreted by the pituitary gland (Healy & Russell-Jones, 1997). Since somatotrope influences both the development of the striatal muscle and the myocardium (Cittadini et al., 1999) we can consider that this hormone leads to simultaneous adaptation to the effort of both the cardiovascular and muscular system. Research shows that for different sports branches the ratio between muscle strength and tendon insertion thickness varies (Kafkas et al., 2018), which is important for the prevention of tendon ruptures. Also important is the prevention of tendon pain, which is increased in some sports (Kafkas et al., 2019). The present paper aims, on the basis of the literature,to analyze cardiovascular and musculotendinous adaptations to the effort, as well as the hormonal influences that characterize them, in order to increase the knowledge about prophylaxis of cardiovascular diseases and tendon rupture at athletes.

Cardiovascular and musculotendinous adaptation to effort - the somatotropic role

Firstly, growth hormone seems to condition vascular adaptation to effort. Thus, adaptive physiological changes encountered at highly trained endurance athletes (cross-country skiers and kayakers) consist of (Lundgren et al., 2015):

- in cross-country skiers the maximum volume of oxygen is higher

- in cross-country skiers and kayakers adaptation to chronic endurance consists of a larger diameter of the arms arteries

- these morpho-functional adaptations do not associate with the blood volume or hemoglobin mass

An earlier study conducted on experimental animals (mouse) suggests that a high plasma somatotropic concentration may cause increased arterial diameter, probably due to the mechanical factor of increased heart rate (Dilley & Schwartz, 1989).

It follows that, in the case of athletes who are the subjects of the study (Lundgren et al., 2015), increase in diameter of the arteries of the arms may be caused both by local hemodynamic factors and growth hormone, increased as a result of the adaptation of the endocrine system to effort (Soriguer Escofet et al., 1992).

It's hard to say to what extent somatotropic excess produced by intense training favors aneurysms or vascular accidents in athletes, but it is known that children treated with growth hormone for the small stature experienced an increased incidence of stroke in adulthood (Poidvin et al., 2014).

Long-term somatotropic abuse in athletes can lead to acromegaly-like syndrome, with an increased risk of hypertension, cardiomyopathy, malignancy, myopathy, peripheral

neuropathy, glucose intolerance (Healy & Russell-Jones, 1997). This results in the need for periodic dosing of this hormone, preseason, not only when changes occur.

Regarding the physiological aspects of cardiovascular adaptation to effort, electrocardiogram detectable, the following are known (Drezner et al., 2013):

- electrocardiogram in athletes presents some particular aspects, their knowledge being necessary for the differentiation of the physiological aspects from the pathological ones

- in this way, sudden cardiac death can be prevented

- among these, we can mention early repolarisation and T wave inversion in anterior leads in African-American athletes, which is probably a specific ethnic adaptation to regular exercise

Endurance-trained athletes have left ventricular mass, left ventricular cavity and stroke volume higher than the control group, with changes more evident in male subjects (Yilmaz et al., 2013).

Cardiovascular adaptation to effort is specific to the ethnic group as follows (Papadakis et al., 2012):

- African / Afro-Caribbean descendants athletes have a significantly higher prevalence of repolarization abnormalities and left ventricular hypertrophy compared to Caucasian athletes

- the extrapolation of the ECG and echocardiography criteria used to diagnose potential serious cardiac disorders from the African/African-Caribbean athletes population to caucasian origin athletes results in an unacceptably large number of unnecessary investigations and an increased risk of false disqualifications from competitions sports

Acromegalic cardiomyopathy, a disease caused by an excess of growth hormone, is associated with biventricular hypertrophy, diastolic dysfunction, arrhythmias (Sharma et al., 2017).

These differences can be explained by the fact that in men of the black race the basal somatotrope secretion is higher (Wright et al., 1995). There are also gender differences, generally the somatotropic plasma concentration being lower in males than in females, but at the first IGF1 level (whose synthesis is largely dependent on growth hormone secretion) is superior (Roelfsema & Veldhuis, 2016).

Sport training in adolescence affects the uniformity of muscle and tendon adaptation, with increased mechanical stress of the tendon and potential implications for tendon lesions (Mersmann, 2016).

Tendons and ligaments are structures rich in extracellular matrix (Subramanian & Schilling, 2015), increasing the availability of growth hormone having the effect of stimulating

collagen matrix synthesis in skeletal muscle and tendon without affecting myofibrillar protein synthesis, demonstrating that growth hormone is more important to strengthen the matrix tissue than muscle tissue hypertrophy at the adult (Doessing et al., 2010).

Discussions

Not only somatotrope condition the effort adaptation of cardiovascular and muscular systems, as well as overcoming adaptive mechanisms to pathological phenomena. Endocrine adaptation to effort requires, in addition to growth hormone, the release of anabolic hormones, mainly testosterone and thyroid hormones (Hagiu, 2014).

A regenerative action of thyroid hormones on heart muscle was discussed (Mourouzis et al., 2011), but also their ability to induce cardiac hypertrophy (Ojamaa, 2010). Thyroid hormones are necessary for the growth and regeneration of the striated muscles (Salvatore et al., 2014). However, although the presence of thyroid hormone receptors has been detected in the tendon, thyroid disease is not considered to be related to tendinopathy or tendon tears (Oliva et al., 2013).

A work on experimental animals (rat) has shown that testosterone abuse causes hypertrophy, fibrosis and apoptosis of myocardial cells (Papamitsou et al., 2011).

On the same experimental animals was studied the effect of anabolic steroids on tendons, which however depends on local metabolism and association with physical exercise (Marqueti et al., 2008).

Although angiotensin converting enzyme is thought to be responsible for cardiac hypertrophy and physical fitness improvement, genetic polymorphism does not seem to influence cardiovascular adaptation to sports training (Boraita et al., 2010).

It results that for cardiovascular and musculotendinous morphological changes that occurred as a result of effort adaptation, growth hormone secretion plays a determining role. But also that growth hormone excess or deficiency, especially in the case of athletes, can lead to cardiovascular disease or increased incidence of tendon ruptures. Theoretically, an insufficient plasma level of the somatotropic can not only favor tendon ruptures, but also tendon pains due to physical effort. It is necessary to monitor the growth hormone levels, especially in adolescent and black race athletes, because the former are more exposed to disproportions of muscular-tendon unit development and the latter to cardiovascular disease. Another age group in which periodic dosing of somatotropic is desirable is represented by veteran athletes, knowing that in the elderly the level of growth hormone decreases (Roelfsema & Veldhuis, 2016).

Consideration should be given to the practice of growth hormone doping, which is illegal and has negative consequences for health (Baumann, 2012). On the other hand, in the case of suboptimal plasma concentrations in athletes, can be considered the possibility of increasing the somatotropic level by specific exercises, such as resistance exercises (Collier, Collins, & Kanaley, 2006). But the problem is the interference with the specific training program. Another solution is supplementation with arginine, a known growth hormone secretagogue (Olinto et al., 2012). However, it should be taken into account that the combined effect of arginine administered before strength exercises attenuates the somatotropic secretion response (Collier, Collins, & Kanaley, 2006).

If that requires alleviating the pathological effects of excess growth hormone drug treatments may be used, promising the antisense oligo and antibody-based approaches (Lu et al., 2019).

Conclusions

1. Cardiovascular adaptation to effort takes place in parallel with musculotendinous; for both growth hormone has a driving role.
2. For cardiovascular adaptation to the effort it's dangerous the excess of somatotrope, which can cause cardiac hypertrophy, arrhythmias, and possibly aneurysms and vascular accidents.
3. For musculotendinous adaptation to effort, the deficiency of growth hormone is harmful, because increases the risk of tendon rupture and possibly tendon pain.
4. It is useful to monitor the somatotropic plasma concentration in athletes, with the possibility of pharmacological intervention or by modification of the training program in order to increase or decrease it.

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