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PHENOMENON OF ATHLETE'S HEART, POSITIVE PHYSIOLOGICAL ADAPTATION TO EXERCISE: WHEN AND HOW?

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(Original scientific paper)

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Abstract

Physical activity places a substantial demand on the whole body, especially on the cardiovascular system. The main goal of circulatory system is to provide enough energy, through transport of nutritional substrate and oxygen, to metabolically hyperactive tissues (skeletal muscles). Cardiac adaptation to exercise encompasses first morphological and therefore functional and electrophysiological changes that are referred to as a phenomenon of the athletic heart. PURPOSE: The aim of this research is to reveal contemporary knowledge of physiological mechanisms which contribute to develop the athlete's heart, criteria for diagnosing the athletic heart, defining of volume (duration, frequency and intensity) and modality of physical activities which cause this cardiovascular adaptation.. METHOD: Specific key words: "athlete's heart", "training" "duration", "intensity", "mechanism", were used to search relevant electronic databases, such as PubMed, Web of Science and Scopus. RESULTS: Individual studies showed that cardiac adaptations may differ according to the type of sports. Numerous publications have confirmed an eccentric left ventricular hypertrophy in endurance athletes and concentric left ventricular hypertrophy in resistance trained athletes. The volume of exercise, intensity and duration of exercise, which are needed to cause the development of athletic heart are still questionable. CONCLUSION: The findings suggest that whatever the mechanism, it is evident that hypertrophy is a genuine response to training which is followed by withdrawal after a period of de-training. Sports scientists and sport's experts who designed the training regimes should be well informed about consequences of physical efforts on cardiac health. General recommendations for physical activity for better cardiac health is up to seven hours vigorous aerobic activity per week.

Key words:

Introduction

Moderate endurance exercise has long been considered as a crucial tool to maintain and/or improve the cardiovascular health in general population. Growing body of scientific and empirical evidence for beneficial effects of regular exercising inspire great number of people all over the world to be active. Significant group of active people performs an intense exercise that could be challenging for the cardiovascular system and to lead to multiple heart adaptive changes that are commonly called "athlete's heart" (Gabrielli et al, 2018). Chronic, excessive sustained endurance exercise may cause adverse structural remodelling of the heart and large arteries (Patil et al.,2012). The athlete's heart is characterized by enlargement of cardiac chambers and eccentric hypertrophy with preserved myocardial function as normal physiological adaptations due to prolonged and intense endurance physical stress. Unfortunately, long-term excessive endurance exercise may induce pathological structural remodeling of the heart, structural changes in the right heart and inter-ventricular septum which could be substrate for dangerous functional events such as arrhythmias (Shavit et al, 2016). High-intensity exercise training may cause heart adaptations which compensate for dynamic circulatory changes which could be followed by increasing cardiac muscle mass. Physiological cardiac hypertrophy is associated with normal or enhanced cardiac function, but recent studies have documented decrements, especially in right ventricular (RV) function (Carbone & D'Andrea, 2017). Besides functional disruptions, the release of cardiac damage marker are noted in athlete's blood. Athlete's heart is a physiological condition and does not require a specific treatment. Many Olympic

champions have cardiac dimensions within the normal range, whereas a college athlete may exhibit pronounced hypertrophy (Oakley, 2001). The fact that nature and magnitude of structural changes is sport specific, raises the question how much and how long of different types of exercise is needed to develop the athletes heart.

The aim of this paper is to summarize the common knowledge of modality and quantity of physical activity which cause the development of athletes' heart. This data could be useful for sport scientist, coaches and athletes in terms of avoiding unwanted implications on cardiac health.

Functional adaptations – physiological mechanisms for cardiac remodelling

Due to numerous beneficial effects on overall health, physical activity could be compared to pharmacological agent, and the safe upper-dose limit should be determined to avoid the adverse effects (O'Keefe et al, 2012). The benefits of exercise training are noticeable even at the volume of 15 minutes moderate activity daily, up to 60 minutes moderate exercise every day, which is accountable for upper limit for optimal health (Haskell et al. 2007). Highly trained endurance athletes perform 200 -300 metabolic equivalent hours per week which is 5 to 10-fold greater than the recommended doses of physical activity for better cardiovascular health (Sharma & Zaidi, 2012).

During the intensive exercise cardiac output or minute volume of the heart may increase significantly, up to several times (5-6 times) than in sedentary condition. Increasing of cardiac output is linear function of its two components, stroke volume and heart rate. Stroke volume is around 70ml in adults, but in athletes it could be much greater. The size of the stroke volume depends on the size of the cardiac chamber. As a result of prolonged intensive exercise training cardiac chambers, especially left ventricle, become larger and stronger (Carbone et al, 2017). The cardiac output mainly depends on the heart rate, which could vary in athletes from 40 bpm at rest up to >200 bpm during exercise in young maximally trained subjects. The ability of the athlete's heart to adapt to greater demands during the intensive physical activity is consequence of structural changes of the heart (larger chamber volume, greater muscle mass) and adaptation of autonomic nervous system, primarily parasympathetic predominance, which contribute to lower rest heart rate (Baggish & Wood, 2011).

Regarding hemodynamic differences exercise activity could be classified as isotonic or isometric exercises. Isotonic or endurance exercise represents activities such as long distance running, cycling, rowing and swimming. Endurance exercise increases cardiac output and causes a volume challenge for the whole heart, affecting all four chambers of the heart. Other form of exercise, isometric or strength training, has a completely different effect on the cardiovascular system. Isometric exercise causes an increase of peripheral vascular resistance, transient systolic hypertension and normal or slightly elevated cardiac output. Strength training is dominant form of exercise in sports such as weightlifting, track and field throwing events etc. (Kovacs & Baggish, 2016). The endurance-based exercise and strength-based exercise leads to distinctly different changes in left ventricle. Athletes exposed to strength training demonstrate concentric hypertrophy whereas individual exposed to endurance training demonstrate eccentric left ventricle hypertrophy (Baggish & Wood, 2011).

Whatever the mechanism, it is evident that hypertrophy is a genuine response to training. Athletes who train seasonally exhibit seasonal variation in left ventricular dimensions (Okley, 2001). Regression of hypertrophy caused by long years of training, can be observed after the process of the de-training (Fagard, et al., 1983).

Factors influencing cardiac remodelling

Several internal and external factors influence remodelling process of the heart structure and function. Besides genetic predispositions, anthropometric and genetic factors, the exercise stimulus with all its features, especially duration, intensity and modality are associated with cardiac remodelling.

Temporal determinants and cardiac remodelling

Exercise induced cardiac remodelling (EICR) is a term that describe changes in the cardiac structure as a result of repetitive and sustained exposure to an exercise stimulus. The minimal dose of exercise required to stimulate the increasing process of the cardiac walls and cavities is insufficiently explored. The majority of studies regarding the duration and intensity of exercise stimulus and cardiac remodelling are designed as cross sectional and their results do not establish causality relationship (Kovacs & Baggish, 2016). Several longitudinal studies of repetitive and sustained exposure to an exercise stimulus have established a

mechanistic link between exercise exposure and sport/physiology-specific EICR (Baggish et al., 2008). The study of young athletes from the Harvard Athlete Initiative has shown that 90 days of intense team-based training is sufficient to produce left ventricular increasing.

Sports modality and cardiac remodelling

Individual studies showed that cardiac adaptation differs according to the type of sports. The impact of different sports and training is noted on cardiac structure and function, and on electrocardiographic alterations associated with athlete's heart (Fagard, 2003). Cardiovascular responses to exercise differ according to the nature of the activity. The main haemodynamic features are increasing of the heart rate and stroke volume, the two components of cardiac output. Dynamic exercise, for example long-distance running, results in increased heart rate and stroke volume, reduced peripheral vascular resistance and a modest rise in blood pressure, creating a volume load on the left ventricle. Static exercise, such as weight lifting, results in a slight rise in heart rate and a significant rise in blood pressure, causing a pressure load on the left ventricle. Most sporting disciplines involve a combination of static and dynamic exercise in varying proportions (Zaid & Sharma, 2011). Volume load would lead to enlargement of the left ventricular internal diameter and a proportional increase of wall thickness; this type of adaptation is termed eccentric left ventricular hypertrophy. The pressure load would induce thickening of the ventricular wall with unchanged internal dimension, or concentric left ventricular hypertrophy. It was suggested that these cardiac adaptations serve to normalize wall stress (Fagard, 2003). The results of some references regarding the sports modality, duration of training regime and cardiac remodelling outcomes are presented in table 1.

Athletes of the same modality, exposed to the same exercise stimulus, duration, intensity, training methods, evidence different cardiac remodelling process (Morganroth et al.,1975). Interesting finding is a relationship between upper limb activity and remodelling process of the heart (Gates et al., 2003). The longitudinal study on healthy adolescents aged 11 to 15 years determined that sports participation from 3.1 ± 1.2 hours per week, plus one match per week, was not associated with increased LV growth (Valente-Dos-Santos et al., 2015). Physiological cardiac adaptation in athletes is influenced by body surface area, gender, age, training intensity and sport type. Sport category has a strong impact on cardiac adaptation. High dynamic – high static sports show the largest changes, whereas low dynamic- high static sports show dimensions similar to non-athletes (Luijkx et al., 2012). Moderate (4-6 METs) and high (6-12Mets) exercise intensity was associated with reduced cardiovascular risk, and risk reductions factors were high, 0.94 and 0.83 compared with low activity intensity (<4 METs) (P =.02 for trend). A half-hour per day or more of brisk walking was associated with an 18% cardiovascular risk reduction (RR=0.82). Walking pace was associated with reduced coronary heart disease risk independent of the number of walking hours (Tanasescu et al., 2002). Six weeks of high-intensity interval training increases left atrial volumes irrespective of training intensity, 85 or 115% maximal aerobic power (Mahjoub et al., 2019).

In the research conducted on military candidates, 76 candidates participated in special operations program which was carried out in two phases. In both phases military candidate train more than 4 hours daily, five times per week. The first phase lasted 10 weeks, and high intensity exercise, different types, tending to achieve 77 to 95% of maximal heart rate. During the second phase, 15 weeks long, intensity of activity was higher, achieving more than 96% of HR max. An increase of left ventricular diameter, left ventricular mass and left atrial mass were observed (Dinis et al., 2018).

Knowing the impact of different modalities, duration and intensity of physical activity, can identify the threshold of potential toxicity of intense exercise training.

Age and gender

Plethora of studies investigated and proved the influence of exercise on the cardiovascular system in all age groups. The cardiac adaptation is evident in adult as well in younger population (Dores et al., 2015). Investigations of dimensions of both atria and left ventricle in large number of young athletes showed significantly greater values in athletes comparing to their sedentary peers (Sharma et al., 1999). In modern world many children and maybe as well many people at older ages are involved in competitive or vigorous recreate physical activity. The impact of these activities on cardiovascular health should be investigated thoroughly to avoid adverse effects.

Male athletes are more affected regarding exercise induced cardiac remodelling. Electrophysiological and anatomical changes are more frequent in male athletes. The reason for higher prevalence of athlete's

heart in males could be result of the male's greater anthropometric parameters, hormonal differences and greater involvement in extreme endurance and competitive sports (Dores et al., 2015).

Many studies show lower prevalence of cardiac enlargement in female athletes compared to male athletes matched with age and training intensity (Pelliccia et al., 1996). Sharma et al. showed similar findings in adolescent athletes (11% lower LVWT and 6% smaller LV size in females (Sharma et al., 2002).

Table 1.: Reference of sports modality, duration of training regime and structural changes of the heart (LV – left ventricular; LA – left atrium)

Reference	Athlete type	Exercise exposure	Primary findings
Ehsani et al. (1978)	swimmers	9 weeks	Increased LV mass
Wieling et al.(1981)	rowers	7 months	Increased LV diameter
Abergel et al. (2004)	cyclists	Professional, 3 years	Increased LV diameter
Du Manoir et al. (2007)	rowers	10 week	Increased LV mass
Baggish et al. (2008)	rowers	90 days	Increased LV mass
Baggish et al. (2008)	football	90 days	Increased LV mass
Weiner et al. (2010)	rowers	90 days	Increased LV torsion and LV early diastolic untwisting rate
Dinis, et al. (2017)	Military candidates	6 months	Increased LV diastolic diameter; increased LV mass; increased LA volume.
Mahjoub, et al. (2018)	HIIT	6 weeks	Increased LA mass

** This table is modified from

Can exercise damage the normal heart

Prevalence of athlete's heart phenomena is between 40% to 50% in male athletes, which showed left and right ventricular cavity dimensions exceeding predicted upper limits. The differentiation between physiology and pathology is subject of complex investigation methods: ECG, CPT, echocardiography, cardiac magnetic resonance imaging (CMRI) and genetic testing. The mechanisms which lead to development of athletic heart are the same mechanism that enable developing of pathologic substrate. The question is when or how positive physiological adaptation of cardiac muscle as result of excessive exercise turns into a pathological condition.

Multiple studies have demonstrated evidence of raised biomarkers as indicators of post-exercise cardiac damage. Repeated bouts of life long endurance exercise is associated with sufficient myocyte necrosis that could transform health cardiac tissue into myocardial fibrosis which is a dangerous substrate for arrythmogenic events (Sharma et al., 2015).

The current European and American guidelines recommend a minimum of 150 min moderate intensity exercise per week for an adult. Competitive and some recreational athletes perform way above these recommendations and regularly engage in over 20 h of intense exercise (15 MET) per week. The hemodynamic loads during the vigorous physical activity is associated with 10-15% increased internal and external dimensions of both ventricles (Papadakis et al., 2011). Additionally, athletes demonstrate enhanced cardiac filling in diastole, augment stroke volume even at very high heart rates, and exhibit increased oxidative capacity and capillary conductance within the skeletal muscle which results in high-peak oxygen consumption during exercise (Sharam et al., 2015). The excessive endurance exercise was found harmful for middle aged endurance athletes, involved in chronic exercise reported a 5-fold risk of atrial fibrillation (AF) compared with the sedentary population (Abdula & Nielsen, 2009). In a recent large study of 52 000 long-distance cross-country skiers, the subjects which has bigger number of races and better finishing times had bigger risk for dangerous cardiac events (Andersen et al., 2013). In men, the combination of current and prolonged lifetime sport practice, more than 1500 active hours is associated with higher risk of AF (Elosua et al., 2006). Leisure-time exercise at younger age is associated with an increased risk of AF, whereas walking/bicycling at older age is associated with a decreased risk. It has been reported as a significantly higher risk for developing AF in men who practiced more than 5 hour of intensive exercise per week at the age of 30 years than in men who workout averagely 1 hour weekly (Drca et al., 2014). According to La Gerche et al. 20 years of exercise are required, more than 20 hours per week, to cause

pathological remodelling of the heart which could lead to fatal arrhythmias (La Gerche et al., 2012). Current evidence indicates that only a small number of athletes may be at risk of cardiac damage from long-standing intensive exercise and the endorphin-mediated euphoria with such practice in some athletes may be 'cardiotoxic' in only a minority (Sharma., et al, 2015).

The plethora of investigations of exercise dose and cardiac remodelling reported certain structural and functional changes related to different types and volume of exercise. But still there is no simple answer on the question of how much exercise is required to develop athlete's heart or to provoke pathological changes on the heart. There are no uniform and explicit recommendations for this dose – response relationship.

Here we will present the suggestions, according the Patil et al, (Patil et al., 2012) for an exercise routine that will optimize health and fitness without causing the adverse cardiovascular effects:

- Avoid a daily routine of exhaustive strenuous exercise training for periods greater than one hour continuously. An ideal target might be no more than seven hours weekly of cumulative strenuous endurance exercise time.
- When doing exhaustive aerobic exercise time, take intermittent rest periods (even for a few minutes at an easier pace, such as slowing down to walk in the middle of a run).
- Once or twice weekly, perform high-intensity interval exercise training to improve or maintain peak aerobic fitness. This is more effective in improving overall fitness and peak aerobic capacity than is continuous aerobic exercise training.
- Incorporate cross training using stretching, for example, yoga, and strength training into the weekly exercise routine.
- Avoid chronically competing in very long distance races, such as marathons, ultra-marathons, Iron-man distance triathlons, 100-mile bicycle races, etc., especially after age 45 or 50.
- Individuals over 45 or 50 years of age should reduce the intensity and durations of endurance exercise training sessions, and allow more recovery time.

Conclusions

Athlete's cardiovascular health is primary goal of sport's medicine physicians. Sports scientist and sport's experts who designed the training regimes should be well informed about consequences of physical efforts on cardiac health. The relationship between exercise dose (intensity, frequency, duration and exercise modality) and cardiac hypertrophy are still not well determined. General recommendation for physical activity for better cardiac health is up to seven hours vigorous aerobic activity per week.

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