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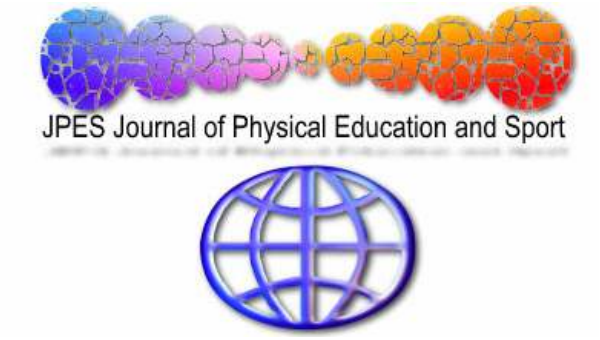

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ORIGINAL RESEARCH

EFFORT ADAPTATION OR SUDDEN CARDIAC DEATH?

Musat Carmina Liana[1], Alexandru Pacuraru[2], Coman Malina[1]
[1]„Dunarea de Jos" University of Galati, Faculty of Medicine and Pharmacy
[2]„Dunarea de Jos" University of Galati, Faculty of Physical Education

Abstract

During training processes, the human body gradually adapts itself, yet it is hard to believe that it has been conceived in such way that it could endure the conditions of winning a modern Olympic or world medal. With respect to the physical effort, there is the following paradox: if the physical effort is acknowledged as a protector of the heart on the long term, then what causes these sports-related conditions that may result in sudden death? Thus arises the necessity of tracking and evaluating the cardiovascular risk targeting the professional sportsmen, their EKG fluctuations, the cardiovascular causes of sudden death, the part played by the physician and the sportsman in preventing the sudden death, as well as numerous clinical cases of sports cardiology.

Key words: athletes, hypertrophic cardiomyopathy, sudden death

Introduction

The prevalence of hypertrophic cardiomyopathy with regard to the general population is of 1 out of 500 subjects, its origin being considered to have genetic causes and a dominant autosomal transmittance.
With respect to young sportsmen, 35-45% of the sudden deaths are caused by hypertrophic cardiomyopathies, the athletes facing a 2.8 times greater risk than the persons not engaged in a professional sport for the occurrence of sudden death. Although certain states have forbidden the taking part of sportsmen suffering from hypertrophic cardiomyopathy in any competition, setting a diagnosis is not an easy task, sometimes it being announced post-mortem, at the autopsy, by means of a macro and microscopic examination. The forensic pathologists may set the cardiac hypertrophy diagnosis, but cannot express a positive opinion regarding the mechanism having led to the patient's death.
Given the fact that the cardiac morphofunctional modifications, displayed by the professional sportsmen as an adaptation to the physical effort, are sometimes similar to the ones attributed to the hypertrophic cardiomyopathy, being able to make differences between the functional and/or morphological modifications of an athlete's heart as being of physiological or pathological nature and drawing a line with this regard, requires additional investigation methods. Nonetheless, there is a certain doubt as to whether these particular investigations are cost-effective and whether the pre-competition testing methods involving the professional sportsmen risking the occurrence of cardiovascular events (a family background of heart failure, sudden deaths, cardiomyopathies) may be based on the anamnsesis, taking into account that the electrocardiogram is not part of the screening.

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Cardiovascular adaptations involving sportsmen

Through the blood, the circulatory system is the one assuring the oxygen and nutrient supply of the cells and is responsible at the same time for transporting the substances resulting from metabolic degradation as to eliminate them from the organism. The heart is a muscular-cavitied four-chambered (2 atriums and 2 ventricles) organ, located in the thoracic cavity, in the mediastinum. It has a conical shape, its top (apex) being pointed towards the diaphragm. It is mainly formed of a rhythmically-contracting muscular structure that pushes the blood through the entire body. The contractions begin in the embryo, three weeks after its formation and continue throughout the entire life of the individual. The muscle only rests for a fraction of second between the beats. In a life span of 76 years, the heart will beat for almost 2.8 billion times and will pump 169 million liters of blood. The size of the heart varies with age, being proportional to the size of the thoracic cavity. Concerning the healthy individuals leading a sedentary life, the weight of the heart depends on their age, gender and body weight. The average weight of a male heart stands for 0.45% of the body weight, while the average weight of a female heart stands for 0.40% of the body weight. With regard to an average-weighted adult, a male heart can reach at most 400 g, while a female heart reaches 350 g. [3]

The process of reaching top sports performances, usually pushing the physiological limits of the human organism, not adequately correlating the training technique with the age of the sportsman, has lately brought along unwanted medical consequences. Regular exercise, leading to undoubted benefits for the health of an individual, is not to be mistaken with taking up a sport professionally.

The intense physical effort carried out on a long period of time may induce functional, adaptive and morphological cardiac modifications. These being considered, the most frequently studied condition is the left ventricular hypertrophy. Whenever being associated to a prolonged physical effort, its condition is usually moderate and can be clearly attributed to physical training. However, there are cases when this hypertrophy is severe enough to require a differential diagnosis, correlated to other medical conditions, especially the cardiomyopathies mainly causing sudden deaths in young athletes. [1,2]

The cardiac hypertrophy, a synonym of the miocardic hypertrophy is caused by a hypertrophy of the working cardiomyocytes. It may either appear as a physiological adaptation mechanism (growing up, taking up sports professionally) or from a pathological cause (arterial hypertension, genetic anomalies). A sportsman develops cardiac hypertrophy as a result of various factors, among which the most important are considered to be the hemodynamic, neuro-hormonal and genetic ones, as they act in a synergetic manner. The data gathered from the experiments carried out on animals shows that the physiologic hypertrophy is moderate and well-balanced, lacking any form of fibrosis and with a capillary density adapted to the myocytic hypertrophy [4.6], being, from the anatomo-pathological point of view, radically different from the secondary hypertrophy of a pathological process, when the left ventricular hypertrophy associates a significant fibrosis and a vascular inadequacy.

The sportsmen's hypertrophy is, by definition, non-pathological and has no complications, although some authors take into consideration a ventricular or supraventricular arrhythmia risk. [7]

There are two types of cardiac hypertrophy: the concentric one, induced by the isometric, static effort, leading to the thickening of the blood vessel's wall without enlarging the VS cavity and the exocentric one, appearing as a result of the dynamic and isotonic effort, the cavity being enlarged proportionally to the thickening of the VS walls. Other considered triggering mechanisms could be certain hormonal factors and bradycardia. Although the majority of the studies reaches similar conclusions, there are also some controversies regarding the connection between the effort type the individual makes and the modifications suffered by the heart.

For the professional athletes, the sports are actual jobs that unfortunately may leave unwanted traces. The superior parietal hypertrophy (above 13 mm) has been frequently discovered, especially in the male patients (16mm), the ones practicing endurance sports: cyclists, swimmers, kayakers. The research carried out in Italy has proven that strength/force trainings of the anaerobe type, mostly accounting for an isomeric effort (halters, tossing weights, and swimming – on short distances) produce VS wall hypertrophy, resulting in the decrease of the VS cavity's internal diameter (concentric HVS). Consequently, the highly-demanding sports (halters) lead to parietal thickening rather than VS dilatation. All the other sports lead to VS dilatation and exocentric hypertrophy. The sports associated with maximal dilatation and thus maximal hypertrophy, are ranked as follows: first comes cycling, then kayak-canoeing, natation, athletic sports (long/short distance) and football. Tennis and hockey induce average modifications, while volleyball and equitation induce minor modifications. Due to the fact that sportsmen training on the long term display a VD dilatation and also a variety of depolarizations/ repolarizations and electrocardiogram anomalies, a differential diagnosis with VD dysplasia is more and more required. Nonetheless, an ultrasonography might make it rather difficult. However, the MRI

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allows a better diagnosis of this condition. Both the global/segmentary dysfunction and the substantial VD dilation sustain the VD dysplasia diagnosis. Should a VS dilatation/thickening be associated, then the heart is sure to be "athletic". In Italy, an increased arrhythmogenic dysplasia VD dysplasia incidence was discovered in athletes having suffered a sudden death [5].
Another research dealt with a comparison of the VD diastolic function (pulsating eco Doppler) between the long distance swimmers [12] and short distance swimmers [14], all displaying HVS signs. The initial and final diastolic speed quota has been found to be greater in the short distance swimmers; the VD myocardial function at the beginning of the diastole is positively influenced by the increase in the pre-charge and represents an independent effort determinant. There are various mechanisms through which the hypertrophy becomes a pathological phenomenon. Among them, an important part is played by the relative coronary failure and the irrigation and metabolism disorders of the excessively hypertrophied myocardium. There is a certain quota of the capillaries irrigating the myocardium fibers and the fibers themselves. At birth, 6 myocardium fibers are attributed to each capillary vessel.[8] As time passes, this quota changes so that once the heart has reached adulthood, only one myocardium fiber is attributed to each capillary, the quota having changed to 1:1. Consequently, the heart's effort adaptation capacity in children is much more reduced. It has been shown that the myocardium's hypertrophy does not lead to the increase of the cardiac fiber number, but an increase in their diameter. A study carried out on three groups of male adolescents, aged 16 to 18 years old has proven the following: the endurance sports athletes presented a 10% increase in the VS mass; the sprint competition athletes were found not to display this increase of the VS mass, while the individuals engaged in strength sports displayed a VS mass increase of 4%. Moreover, another study carried out on 1451 athletes has confirmed that if the athlete combines aerobic and anaerobic trainings, his heart's morphology displays intermediate features of the concentric hypertrophy and the exocentric one. In 267 of the athletes having exclusively been engaged in endurance trainings, the telediastolic VS volume has increased proportionally to the length of the training.[9]

Examination methods

Initially, the cardiac hypertrophy regarding athletes may be discovered at the percussion of the thorax and then confirmed by means of a thoracic radiography [8]. At present, the eco-cardiography is considered to be a valuable examination, due to the fact that is reveals the morphological characteristics of the heart. With regard to the athlete, the resting trans-thoracic eco-cardiogram may prove to be normal or may point out cardiac morphologic alterations of the hypertrophy-dilatation type, which affects all the 4 cavities, the alterations being well-balanced and proportional. [14]. These alterations can be observed in both genders and at all ages between 15 and 19. These adaptations are generally moderate. Regarding the male patients, an increase greater than 13 mm is uncommon and rare [13] and if the values range from 13-15 mm, the case is considered to be unclear, additional examinations being required. An increase surpassing the value of 15 mm may be caused by a pathological cause. During the telediastole, an athlete's left ventricle's diameter rarely surpasses 60 mm, reaching 70 mm only in rare occasions [10]. Greater dilatations have been observed in cyclists or in case of an increased body mass – the case of basketball players [12]. The normal limit is considered to be 31mm/m, in which case the quota is obtained based on the body weight.
Concerning the female subjects, the thickness of the wall is not greater than 12 mm. The left ventricle's diameter during the telediastole rarely surpasses 55 mm and exceptionally reaches 65 mm [9,11].
With regard to children, especially the girls, the dilatation of the ventricular cavity is not accompanied by a significant reactional parietal hypertrophy. At puberty, boys are prone to display a parietal hypertrophy no greater than 12 mm.
The morphological modifications of the heart cannot be strictly linked to the physical training in all the athletes. Therefore, it is agreed that the genotype may predispose to a cardiac hypertrophy unrelated to the physical training.

The differential diagnosis between the physiological hypertrophy and the pathological one, differential diagnosis issues including the parietal hypertrophies and/or the significant cavitary dilatations

First and foremost, all the hypertrophies associating a significant drop in the athlete's performance are considered to be "a priori" pathological. Whenever an asymptomatic professional athlete displays a parietal hypertrophy greater than 13 mm in men and 12 mm in women or young athletes, there are three entities which

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must be excluded: HTA and valvulopathies that are easy to eliminate and the hypertrophic cardiomyopathy, posing a real diagnosis problem and a vital risk if simultaneous with an intense physical exercise. [1]
The hypertrophic cardiomyopathy diagnosis is set by measuring the parietal thickness in the left ventricle, based on a two-dimension evaluation whenever it surpasses 15 mm without the patient having a family background to this regard and 13 mm with the patient having a family background of hypertrophic cardiomyopathy.
In order to give a differential diagnosis, the physician may resort to:

- the resting trans-thoracic Doppler eco-cardiography. An isolated hypertrophy discovered in an athlete's case is considered to be an abnormal situation. The parietal hypertrophy is associated to a cavity dilatation that becomes very important should the training be predominantly dynamic. A telediastolic VS diameter greater than 55 mm can easily identify the heart of an athlete, while a diameter not greater than 45 mm, associated to a parietal hypertrophy is pointing towards the hypertrophic cardiomyopathy.
- The parietal hypertrophy of athletes is global and symmetrical. It can be asymmetrical, yet in such case the quota between the posterior wall/ the thickness of the inter-ventricular septum is inferior to 1.5 [13]

Conclusions

The conclusion that can be drawn is that the heart's best protection is given by a long term average-intensity physical effort; the professional athletes are not included in this category, as they are constantly pushing the limits of their body, this leading to specific rhythm (sinus bradycardia) or structure cardiac alterations (hypertrophic cardiopahty), which is not really the synonym of a sudden death risk, this being only an adaptation reaction of the body to the intense physical effort.

Therefore, there is a strong necessity of tracing and assessing the cardiovascular risk targeting the professional athletes, the modifications of their electrocardiograms, the cardiovascular causes of the sudden death, the part played by both the doctor and the sportsman in preventing the sudden death, as well as numerous cases of clinical sports cardiology cases.

Regarding the additional examinations that could be carried out, the latest investigation method not available yet in Romania is represented by the effort eco-cardiography. Consequently, the necessity of the Romanian specialists to channel their interest only on certain domains of their specialty in order to reach the highest level of effectiveness is once more pointed out.

Therefore, professional athletes must undergo thorough examinations even at the slightest symptom they display.

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