

THE LEFT VENTRICLE IN THE ELITE ATHLETE

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INTRODUCTION

The medical community has been debating for more than a century about the adaptation of the cardiovascular system of trained athletes. In 1899 Henschen was the first to describe some changes during a basic physical examination in cross-country skiers. Namely, using only careful chest percussion, he recognized the enlargement of the heart, that has not been noticed before¹. He concluded that endurance training is associated with both cardiac dilatation and hypertrophy in athletes, and he introduced the term “athlete’s heart”, which is still used. However, proving his hypothesis was challenging due to the lack of adequate non-invasive imaging tools.

The introduction of radiography was a first important step in the evaluation of athlete’s heart. In the middle of 19th century, investigators used chest radiography to show that heart size was increased in athletes, and predominantly in those involved in endurance sports with large

aerobic capacity. However, the investigators were not certain about the role of cardiac dilatation and some of them thought that the heart dilatation in the trained athlete could weaken their heart due to the “strain” created by continuous and strenuous training, inducing deterioration of cardiac function and even heart failure². Since then, there are many controversies about the prognosis of an athlete’s heart. On one hand, investigators claimed that athlete’s heart represents the benign physiologic adaptation and the result of the training which will normalize as soon as training is reduced or stopped. On the other hand, some researchers suggested that this might be pathological that could cause heart failure in the future.

The introduction of echocardiography almost four decades ago enabled the detection of cardiac changes that occur in the heart during and after exercise, as well as those alterations that represent the consequences of athletic conditioning.

Advanced echocardiographic techniques such as 2D and 3D speckle tracking, as well as the development of 3D echocardiography, provided insight into cardiac remodeling in athletes.

Factors of cardiac remodeling in athletes

There are several important elements that determine cardiac adaptation to endurance training in elite athletes: type of sport, gender, ethnic differences and body size³.

Type of sport

Haemodynamic changes are different in various conditioning programmes. Endurance sports such as cycling, long-distance running, swimming, skiing, and rowing are predominantly related with volume overload and reduced peripheral vascular resistance, which altogether leads to increased cardiac output that represents a continuous challenge to all cardiac chambers. Strength (power) disciplines



such as weightlifting and wrestling are principally associated with normal or slightly elevated cardiac output and increased peripheral vascular resistance, which results in increased blood pressure and left ventricular (LV) afterload pressure.

Thus, elite athletes involved in endurance sports are characterized by increased LV size, thickened LV wall and elevated LV mass⁴, while those involved in strength disciplines have normal LV cavity size and increased LV wall thickness⁵. The athletes involved in sports with mixed training programmes (aerobic and anaerobic) such as soccer, basketball, hockey and rugby usually have increased LV cavity size and normal LV wall thickness⁵. On the other side, skill and technical disciplines such as golfing or yachting have only a minimal effect, if any, on cardiac remodeling.

Gender

LV remodeling is present in both genders, among elite athletes, but is more pronounced among men. The extent of LV remodeling is usually less pronounced in women athletes, which might nevertheless be an artefact due to the lower training

volumes. Previous investigations showed that LV wall thickness increases caused by training usually does not exceed 12 mm, which was considered as the upper normal limit, and usually does not fall into a “gray zone” of borderline LV hypertrophy⁶. However, the latest guidelines significantly changed upper normal limits for both genders (10 mm for men and 9 mm for women)⁷. Thus, it is questionable how many women athletes would currently be classified in the “gray zone” of LV hypertrophy. Investigations showed that women athletes have smaller absolute LV cavity dimensions (average, -10%) and wall thicknesses (average, -20%) in comparison with male athletes of the same age, ethnic origin, and sporting disciplines⁶. These differences are probably the consequence of several factors such as smaller body size and lower absolute cardiac output and systolic blood pressure achieved during exercise by women compared to men.

Ethnic differences

Investigations revealed that black male athletes have a higher prevalence of LV hypertrophy than white male athletes.

Among athletes of African or Afro-Caribbean origin, 18% showed an LV wall thickness ≥ 13 mm, especially in sports like sprinting, boxing and basketball (mean 11 mm, range 8 to 16 mm)⁸. A total of 3% of healthy black athletes had a LV wall thickness of 16 mm. Nevertheless, LV diastolic function in black athletes was similar with white athletes and black sedentary controls although LV mass was significantly higher in black athletes⁸. Rawlins et al reported that systematic physical exercise in black female athletes was associated with greater LV hypertrophy than in white female athletes of similar age and size participating in identical sporting disciplines⁹. On the other hand, Zaidi et al did not find any difference between black and white elite athletes in LV diameters or LV mass¹⁰. White athletes had even higher LV mass, but it was not statistically significant. The authors claimed that the impact of ethnicity on heart chambers is minimal, but they also emphasized the need for race-specific reference values for all cardiac chambers, especially the right ventricle.

These potential differences in training-related cardiac remodelling could be partly explained by polymorphisms of

angiotensinogen and/or the angiotensin-converting enzyme between different ethnical groups¹¹.

Physiological changes during exercise

Cardiac remodelling in response to training has been systematically defined with respect to the type of conditioning: endurance or strength training. However, there are some common changes typical for both kinds of training.

A significant increase in skeletal muscle oxygen demand measured by elevated oxygen uptake and increased transport of the oxygen-enriched blood to the working skeletal muscles is characteristic of intensive endurance training. The acute cardiopulmonary adaptation to endurance exercise in physiological conditions incorporates increases in pulmonary ventilation, maximum oxygen consumption, heart rate, stroke volume and cardiac output accompanied by a moderate increase in peripheral vasodilatation and systolic blood pressure¹². These acute cardiac changes could be maintained for several hours on the aerobic exercise level by the endurance-trained heart.

These adaptations are responsible for the development of athlete's heart in high-performance endurance athletes. The main features of this condition are increased LV and right ventricular volumes, increased LV wall thickness

TABLE 1

<i>Endothelial function</i> ↑
<i>Oxidative stress</i> ↑
<i>Myocardial capillary density</i> ↑
<i>Coronary flow reserve</i> ↑
<i>Tolerance of myocardial ischaemia</i> ↑
<i>Arterial stiffness</i> ↓
<i>Arterial blood pressure</i> ↓
<i>Ventricular fibrillation thresholds</i> ↑

↑ *improvement* ↓ *reduction*

Table 1: The positive influence of endurance training on cardiovascular system.

“**Approximately 50% of trained athletes demonstrate some evidence of cardiac remodeling¹⁵, which consists of alterations in ventricular chamber dimensions.**”

and myocardial mass and functionally by a five- to six fold increased cardiac output and reduced resting heart rate¹²⁻¹⁴. Additionally, long-term cardiovascular adaptation to dynamic training produces increased maximal oxygen uptake due to increased cardiac output and arterio-venous oxygen difference. The main changes of the cardiovascular system triggered by endurance training are presented in Table 1.

Left ventricular hypertrophy

Many investigations have focused on the adaptations of the LV on endurance training, especially in male Caucasian athletes. The investigators agree that the responses of elite athletes to long-term conditioning are not uniform. Approximately 50% of trained athletes demonstrate some evidence of cardiac remodelling¹⁵, which consists of alterations in ventricular chamber dimensions, including increased left and right ventricular and left atrial cavity size and volume, accompanying with normal systolic and diastolic function.

Even though cardiac dimensions in trained athletes are in many cases in the range of age and sex-matched sedentary individuals, studies revealed that athletes have significant increased LV cavity size (approximately 10%), LV wall thickness (around 15%) and LV mass (about 45%)^{3,6,16}. Nevertheless, significant LV enlargement (≥ 60 mm) was noticed in only 14% of highly trained male athletes and 1% of highly trained female athletes⁶. Half of the

variability in LV cavity size was ascribed to body surface area and only 14% and 7% were ascribed to sport discipline and gender, respectively⁶. Roughly 25% of LV cavity size increment might be influenced by genetic factors.

Investigators showed that LV enlargement could be related with an increase in LV wall thickness that exceeds limits that were previously considered as upper normal (13 mm). In white male elite athletes this is rare with a prevalence of 2 to 4%, especially in athletes participating in dynamic sports such as rowing or cycling⁸. Black male elite athletes have significantly higher prevalence of LV hypertrophy. The same investigation showed that up to 18% black athletes had LV wall thickness ≥ 13 mm. Comparing with male athletes, female athletes have a significantly lower maximal LV wall thickness, which is probably related with lower body size. Studies demonstrated that no white female athlete exhibited a LV wall thickness >12 mm, whereas only 2% of black female athletes had a LV wall thickness of 13 mm⁶. However, one should keep in mind that these investigations were performed when cut-off values for LV wall thickness were significantly higher than in the most recent guidelines published 2015⁷. According to the current guidelines upper limit for LV wall thickness in men is 10 mm and in women is 9 mm. The guidelines also state that LV wall thicknesses of up to 13 mm are considered 'mildly abnormal' and these are guidelines that are not specific elite

TABLE 2

<i>Parameters</i>	<i>Athlete's heart</i>	<i>Hypertrophic cardiomyopathy</i>
<i>LV hypertrophy</i>	<i>Symmetric</i>	<i>Asymmetric</i>
<i>LV cavity size</i>	<i>> 55 mm</i>	<i>< 45 mm</i>
<i>LV thickness</i>	<i><13 mm</i>	<i>>15 mm</i>
<i>Reduction in LV wall thickness with deconditioning</i>	<i>+</i>	<i>-</i>
<i>LV strain</i>	<i>Normal</i>	<i>Reduced</i>
<i>LVOT obstruction</i>	<i>-</i>	<i>+/-</i>
<i>Left atrial enlargement</i>	<i>+ (mild)</i>	<i>+ (moderate)</i>
<i>Diastolic dysfunction</i>	<i>-</i>	<i>+</i>
<i>T wave lateral inversion in ECG</i>	<i>-</i>	<i>+</i>
<i>VO₂ max < 50 ml/kg/min (<120% predicted)</i>	<i>-</i>	<i>+</i>
<i>Pro-BNP</i>	<i>Normal</i>	<i>Increased</i>
<i>Low relative myocardial blood volume</i>	<i>-</i>	<i>+</i>
<i>African or Afro-Caribbean origin</i>	<i>+</i>	<i>+</i>
<i>Family history of hypertrophic cardiomyopathy</i>	<i>-</i>	<i>+</i>

Table 2: Clinical criteria used to differentiate athlete's heart from hypertrophic cardiomyopathy.

athletes but are applicable to the general population. Interestingly, the remodelling of the LV is more eccentric in endurance athletes, however athletes often maintain balanced hypertrophy.

Considering the fact that hypertrophic cardiomyopathy with excessive LV hypertrophy is one of the main causes of sudden cardiac death in young athletes, it is essential to make the adequate distinction of this pathological condition from athlete's heart. Inadequate diagnosis could be misleading and result with fatal event. Previously was postulated that the so-called "grey zone" refers to LV wall thickness between 13 and 16 mm. This is the reason why Maron et al proposed clinical criteria for differentiation between hypertrophic cardiomyopathy and athlete's heart²⁷, which have been significantly modified 10 years ago and presented in Table 2⁵.

ECG changes in terms of negative T wave in the lateral leads are uncommon in athletes. An apical or asymmetric LV hypertrophy of the interventricular septum is also not characteristics of athlete's heart, but hypertrophic cardiomyopathy. Deconditioning is very important because it may determine the etiology of LV hypertrophy. Namely, even short periods of deconditioning (6 to 8 weeks), lead to resolution of physiologic LV hypertrophy. LV cavity is usually small in patients with hypertrophic cardiomyopathy, while endurance athletes often have enlarged LV cavity size (>55 mm). Left atrial dilatation could be present in both conditions. However, patients with hypertrophic cardiomyopathy usually also have LV diastolic dysfunction which is not present in athletes. Additionally, patients with hypertrophic cardiomyopathy have a low

myocardial capillary density, which is not characteristic of athlete's heart. Patients with hypertrophic cardiomyopathy usually have a significantly lower maximum oxygen consumption (<50 ml/min/kg or <120% of the predicted maximum) comparing with elite athletes. In the case of family history of hypertrophic cardiomyopathy one should always exclude the possibility of this disease, including genetic testing, before any conclusion in the direction of physiological heart response such as the athlete's heart.

LV diastolic function

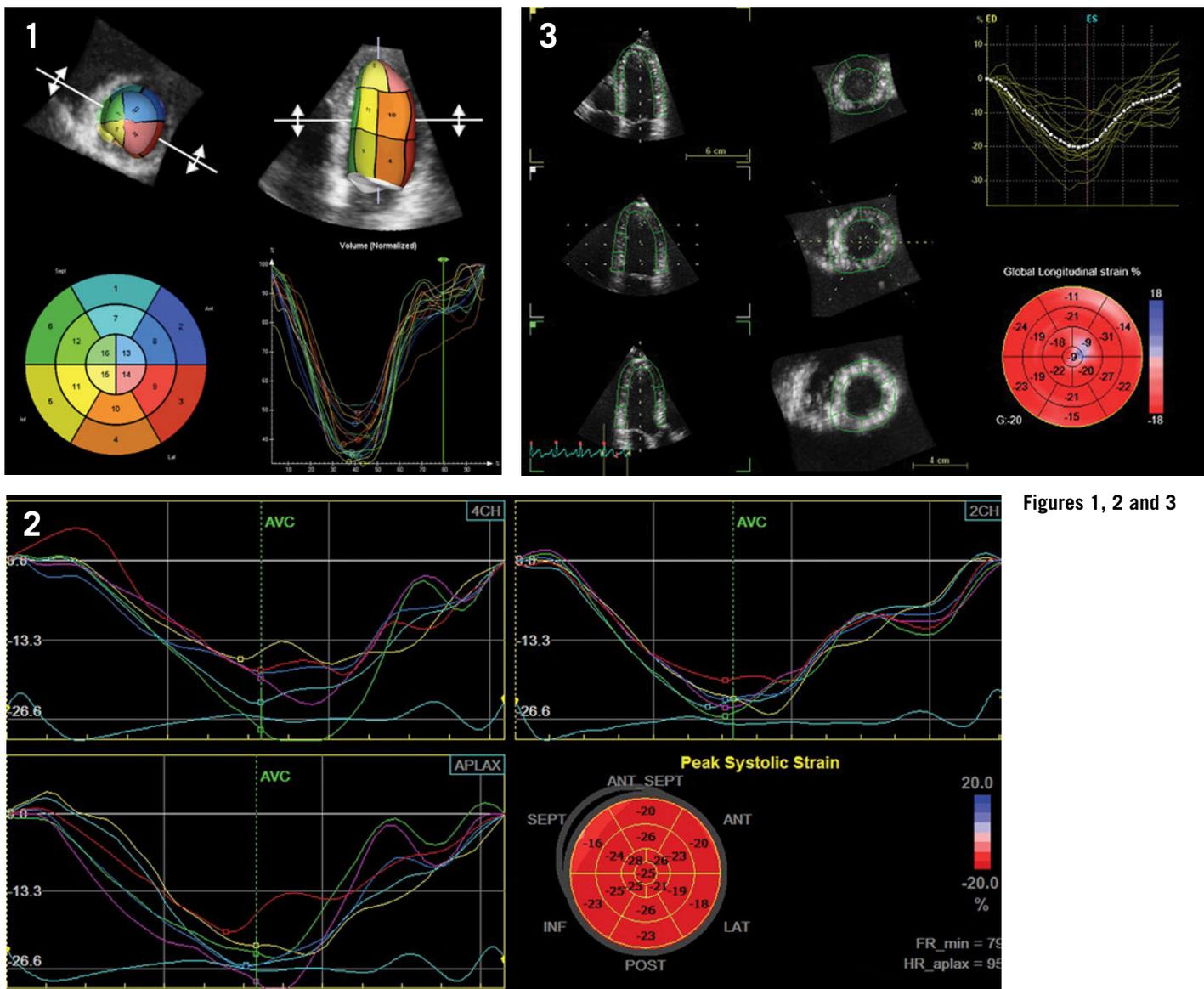
LV diastolic function should be included in comprehensively echocardiographic assessment of the athlete's heart because exercise is associated with changed LV diastolic filling¹⁸. Diastolic dysfunction typically precedes systolic dysfunction. However, it is important to emphasize that athlete's heart typically does not involve LV diastolic or systolic dysfunction.

Trained athletes have enhanced early diastolic LV filling, in addition to supernormal medial annular tissue Doppler velocities¹⁹. This kind of LV diastolic changes enables the maintenance of stroke volume during extremes in endurance-trained athletes. LV diastolic function is a crucial factor in differentiating adaptive remodelling in athlete's heart from disease remodelling.

Endurance-trained athletes show greater stroke volumes compared to controls at the same pulmonary capillary wedge pressure, which suggests that endurance training improves LV compliance. Studies revealed that LV filling pressure was lower in athletes than in controls at similar submaximal stroke volumes²⁰. Furthermore, LV filling pressures remained lower in athletes at maximal exercise, when stroke volume and end-diastolic LV volume were significantly higher than in controls. New echocardiographic techniques enable more accurate detection of LV diastolic function. Thus, strain analysis showed that athletes actually have a supernormal LV diastolic function.

LV systolic function and mechanics

Most authors agree that LV systolic function is within the normal range



Figures 1, 2 and 3

among highly trained athletes. However, it is noticed that elite cyclists can have decreased LV ejection fraction²¹. Namely, more than half of 286 examined cyclists of famous Tour de France showed increased LV diastolic dimensions (>60 mm), with a maximum value of 73 mm, associated with mildly reduced LV ejection fraction (<52%) in almost 12% of the athletes²¹.

It should be mentioned that LV ejection fraction at rest might be underestimated in athletes with increased LV volumes. Cyclists with an impaired LV ejection fraction had also large stroke volumes than sedentary subjects. The improvement of LV systolic function during exercise should help to distinguish athlete's heart from that of heart failure patients.

In the last two decades, tissue Doppler imaging was used to distinguish LV systolic dysfunction from LV adaptation

in athlete's heart and this technique confirmed supernormal LV systolic function in these subjects. However, new imaging techniques are far more accurate and could significantly improve our knowledge and assessment of LV function in athletes. Namely, 3D echocardiography provides very accurate LV volumes and ejection fraction, comparable to magnetic resonance. This technique can even provide the information of the contribution of each cardiac segment to overall cardiac systolic function (Figure 1).

In the last few years technique used for borderline cases of LV hypertrophy and dysfunction are 2D and 3D strain analyses (Figure 2 and 3). Using these methods, one can determine myocardial mechanics in all directions: longitudinal, circumferential and radial. Investigation performed in athletes revealed that they have 'supernormal' function. The absence of reductions in global

systolic longitudinal strain supports the use of strain imaging to assess the physiologic increase in LV wall thickness associated with athletic training. Endurance-trained athletes develop biventricular dilation with enhanced diastolic function, whereas strength-trained athletes develop concentric increases in LV wall thickness with diminished diastolic function. Strain echocardiography helps assessing LV systolic and diastolic changes in subjects with structural alterations of the heart and helps in distinguishing pathological conditions such as hypertrophic cardiomyopathy or incipient heart failure from athlete's heart.

CONCLUSION

The knowledge regarding the athlete's heart progressively improved over the last decades, which is the result of enhanced imaging tools, principally echocardiography.

We are now aware of the fact that body size, sporting discipline and ethnicity are independent predictors of LV remodelling in athletes. Although there is no data regarding long-term endurance training and possible LV dysfunction, we should carefully assess LV function and mechanics in athletes and differentiate pathological LV hypertrophy (hypertrophic cardiomyopathy) and dilatation (dilative cardiomyopathy or heart failure) from adaptive LV hypertrophy and increase in LV volume.

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