

Cardiac Fatigue in Athletes: A Reality We Must Think About

Fatiga cardíaca en los deportistas: Una realidad en la que hay que pensar

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Prolonged and intense physical exercise can produce acute cardiovascular changes that may impact on the athletes' health and performance. Echocardiography has allowed the discovery and understanding of the morphological and functional response experienced by the trained heart, contributing to assess the possible cardiac injury triggered by high intensity exercise.

The athlete heart is characterized by global chamber remodeling, with a preserved biventricular systolic function, or in the lower limit of normality, and normal, or even improved, relaxation. There is some debate about whether the athlete systolic function at rest is a reliable parameter of left ventricular (LV) contractile capacity. Some authors support that a low left ventricular ejection fraction (LVEF) in athletes is not equivalent to systolic dysfunction, as the myocardium preserves the ability to increase its contractility with the increased preload elicited by exercise in response to the Frank-Starling law.

New myocardial strain imaging techniques with speckle tracking have allowed to study cardiac function, providing additional information beyond ejection fraction. Caselli et al. (1) defined the characteristics of LV myocardial mechanics with 2D speckle tracking in 200 Olympic Italian athletes. They showed normal global longitudinal strain (GLS) values, though slightly below that of controls ($-18.1\% \pm 2.2\%$ vs. $-19.4\% \pm 2.3\%$, $P < 0.001$). Systolic strain rate (SRS) and early diastolic strain rate (SRE) were not different between athletes and controls and late diastolic strain rate (SRA) was lower in athletes. However, this reduction was not clinically relevant and should not be misinterpreted as reduced function.

Athletes participating in resistance disciplines presented higher LV chamber diameter, wall thickness and mass index, as well as higher left atrial size compared with other disciplines, which represents adaptation to high hemodynamic load. Conversely, the differences in strain and strain rate related with the type of sport practiced were only slight, suggesting that, although LV morphological remodeling is associated

with the accumulated dose of physical training type and intensity, myocardial performance is independent of the degree and morphological characteristics of LV physiological remodeling.

Gender differences were only minor and characterized by insignificant reductions of GLS, SRS and SRE in male athletes.

On the other hand, it is reasonable to think that changes in strain and strain rate will occur after acute changes in preload. However, myocardial strain studies in athletes have shown that left ventricular function remains normal after intense training. Zilinski et al. (2) analyzed cardiac modifications in 45 marathon runners, before and after an 18-week controlled training program prior to the 2013 Boston Marathon, and found no significant differences.

Conflicting results have been reported for right ventricular (RV) systolic and diastolic evaluations; most studies refer comparable systolic function parameters between athletes and controls. (3-5) Right ventricular myocardial deformation results in athletes are also contradictory. While some studies report a reduction of RV systolic function at rest in athletes, (5-7) other studies do not find significant changes (8) or, even, a slightly increased function. (9, 10)

Some authors have related the degree of RV dilation with decreased RV longitudinal strain, (6) while others have associated it with the increase in tissue Doppler displacement velocities (10) or have found no relationship between dilation and myocardial deformation. (9) An association between RV myocardial deformation and increased training has been also found throughout the sporting season, (11) during isometric exercise (12) or during incremental exertion, (7) though it could decrease after high-intensity exercise. (13) Despite the decrease in resting systolic function described by some authors, normal or higher values are attained during maximum exercise, (7) which would simply indicate the lower work necessary to maintain systolic volume when RV volume is increased, and not a pathological abnormality.

REV ARGENT CARDIOL 2019;87:403-405. <http://dx.doi.org/10.7775/rac.v87.i6.10645>

SEE RELATED ARTICLE: Rev Argent Cardiol 2019;87:436-441. <http://dx.doi.org/10.7775/rac.v87.i6.16435>

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Data of RV diastolic function are also contradictory; while some authors describe an improvement in diastolic function (3, 10) others do not find significant changes. (9) The works performed by D'Andrea et al. (3, 4) have shown that resistance athletes present greater E wave velocity and, consequently, a greater E/A relationship at the level of the tricuspid valve compared with strength athletes and control subjects.

The most recent atrial studies have measured not only diameter and volume but also atrial contraction by speckle tracking, providing physiological information to atrial contraction assessment. Peak atrial longitudinal strain (PALS) and peak atrial contractile strain (PACS) are reduced in athletes. The explanation of decreased atrial strain during both ventricular and atrial systole is based on the improved ventricular relaxation capacity enabling it to be more in charge of its own filling as a consequence of training. This could be related to increased left ventricular muscle elasticity and flexibility and greater end-diastolic distensibility in athletes.

There is concern about the hemodynamic stress repercussion produced by prolonged aerobic exercise on the cardiac homeostasis of athletes. The impact of prolonged strenuous exercise on cardiac function seems to have a transient negative effect both on the right and left ventricles. Specifically, the analysis of strain may identify subclinical myocardial impairment and be useful in the clinical scenario to identify situations of cardiac fatigue. High intensity resistance exercise increases transiently ventricular wall stress, causing functional cardiac and acute biochemical abnormalities, with uncertain long-term consequences. Cardiac dysfunction and biomarker release induced by exercise are not exclusive of very high resistance sporting events and are produced during high intensity and shorter duration exercise, a common and usual component of daily training regimens performed by resistance athletes. (15)

Although functional and structural cardiac adaptations are well characterized in professional athletes, there is only limited information for recreational runners suffering from this supraphysiological stress.

In the study published in this issue of the Argentine Journal of Cardiology, from Instituto de Cardiología y Deportes de Mendoza, Argentina (Cardiology and Sports Institute of Mendoza) pre- and post-exertion echocardiographic changes were observed in 23 ultra-trail (mountain races above 42 kilometers) athletes, as a function of sex, age and training load, using myocardial strain techniques by speckle tracking. A significant decrease of LV ejection fraction and LV diameter was found after the exertion and an increase of basal right ventricular diameter. Left ventricular deformation parameters analyzed by means of GLS, were significantly reduced, same as RV free wall deformation parameters. The weekly training load in all the group did not correlate with these changes; how-

ever, the analysis by gender shows that these results are not conclusive because they are due to contradictory results in women, whose number was very low. It seems that ultra-marathons generate an acute RV deformation impairment, same as an increase in size, which affect in a lower degree the LV size and function.

It should be pointed out that these findings should be placed in the context of races performed at high altitude, so that, without knowing the contribution of hypoxia, they should be interpreted with caution when extrapolating them to long-distance races. It is a pity that no information on the reversion data and biochemical markers was provided to support some of the hypotheses to justify their findings.

In conclusion, works such as the one performed by Dr. Picco and Wolff supply new information on the possible implications of competitive sports in ultra-resistance disciplines. However, there are still great voids to fill in order to understand the physiological mechanisms that might be involved in the adaptations to competitive sport. Further studies will be necessary comparing echocardiographic findings with possible metabolic and enzymatic changes.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/Supplementary material).

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