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EDITORIAL COMMENT

Athlete's heart and soldier's heart: Is Morganroth striking back?

O coração dos atletas e dos militares: está Morganroth a contra-atacar?

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The expression 'athlete's heart' describes the physiological adaptations – morphological, functional and electrical – of the heart to physical activity.

The phenotypic expression of athlete's heart is known to be influenced by several factors,[1] including body size (body surface area, fat-free mass, and height), gender, ethnicity and age. Together with these demographic factors, cardiac adaptations to exercise also depend on the type, intensity, and cumulative duration of training protocols, with a dose-effect relationship (more than 10-15 hours per week of exercise usually being associated with cardiac remodeling). The hemodynamic overload caused by training is always characterized by a harmonious and consistent increase in the dimensions of all cardiac chambers, while a disproportionate increase suggests a pathological process.

Almost half a century ago, Morganroth et al.[2] proposed that the morphological adaptations of the heart in athletes result from the type of hemodynamic overload imposed during exercise. According to this hypothesis, dynamic, endurance (isotonic) exercise would, as a consequence of volume overload, lead to eccentric left ventricular (LV) hypertrophy (LVH) (LV mass increase with increased LV cavity size). By contrast, static, strength (isometric) training would, as a consequence of pressure overload, cause concentric LVH (increased wall thickness with no change in cavity size).

Since then, the 'Morganroth hypothesis' has been challenged and tested in several cross-sectional studies. While these studies have in many cases supported the hypothesis for endurance training, they have failed to confirm it in strength sports. This may, at least in part, be explained by the fact that in the real world, many supposedly purely isotonic sports in fact simultaneously involve both types of exercise to a similar extent, leading to mixed remodeling patterns. On the other hand, strength exercise often consists of sequential short bursts of intensive exertion and pressure overload, with relatively short cumulative duration. Other possible explanations include the lack of standardized measurements, the inaccuracy of M-mode and two-dimensional echocardiographic measurements, and the confounding effect of performance-enhancing substances.

Ideally, the Morganroth hypothesis[2] should be retested in longitudinal studies, using well-defined exercise protocols and standardized measurements, with cardiac magnetic resonance imaging (CMRI) and/or three-dimensional (3D) echocardiography.

In this issue of the *Journal*, Dinis et al.,[3] in a prospective case-control study using echocardiography, compare the impact of exercise training in athletes and in soldiers

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undergoing special forces training. They showed that while the soldiers showed a predominantly eccentric remodeling pattern, in athletes the pattern was essentially concentric. The authors postulate that the intensity and duration of training and specific characteristics of the training programs might explain the different remodeling patterns found.

The soldiers received higher-intensity training than the athletes, with greater cumulative duration, and greater preload and volume overload, leading to eccentric remodeling. The athletes, with moderate-intensity exercise and less cumulative duration, developed concentric remodeling as a response to pressure overload.

The authors thus partially confirm the Morganroth hypothesis, explaining that the morphological adaptations of the heart in athletes result from the type of hemodynamic overload imposed during exercise. However, according to them, and contradicting Morganroth, it is not the type of exercise, so statically defined by Morganroth, but the cumulative amount and intensity of exercise in the training program (a pattern of phases over time) that in real life determines the predominant remodeling pattern, as suggested in a previous small longitudinal study with a relatively long follow-up.[4]

As stated above, the time-honored 'Morganroth hypothesis' should be retested in longitudinal studies, using well-defined exercise protocols and standardized measurements, with CMRI and/or 3D echocardiography. However, the study by Dinis et al. does not fulfill all these criteria; although the study was longitudinal, the exercise protocols were not defined by the researchers, so they should have been more precisely controlled for; furthermore, neither CMRI nor 3D echocardiography was used; finally, their follow-up was too short.

Regarding athlete's heart and soldier's heart, it is not the quality (the type of sport) but the quantity (duration and intensity of exercise) that matters. Is Morganroth striking back? To be confirmed...

Conflicts of interest

The author has no conflicts of interest to declare.

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