Rev Port Cardiol. 2018;37(8):703–705

Revista Portuguesa de
Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

EDITORIAL COMMENT

Sports and cardiac arrhythmias☆

Prática desportiva e arritmias cardíacas

Daniel Bonhorst

Instituto Português do Ritmo Cardíaco, Porto Salvo, Portugal

Available online 27 July 2018

The relationship between sports and the occurrence of cardiac arrhythmias has been the subject of considerable interest in recent years. This relationship is affected by various factors including the intensity of training, gender, age, race and the type of sports.

According to Mitchell's classification,[1] different sports can be characterized by their dynamic (resistance or isotonic) and static (strength or isometric) components. Dynamic exercise involves changes in muscle length and joint movement with rhythmic contractions that develop a relatively small intramuscular force, while static exercise involves development of a relatively large intramuscular force with little or no change in muscle length or joint movement. These two types of exercise should be thought of as the two opposite poles of a continuum, with most physical activities involving both static and dynamic components in varying proportions. Thus, each sport can be classified according to the level of intensity of its dynamic and static components.[1]

Increases in preload and afterload during chronic intensive exercise are associated with symmetrical enlargement of the cardiac chambers. Compared to non-athletes of similar age and size, athletes show a 10-20% increase in left ventricular wall thickness and a 10-15% increase in left and right ventricular cavity size.[2]

Generally, the largest cavities are found in athletes who participate in endurance (dynamic) sports, such as rowing or cycling.[3] Predominantly isometric training, such as weightlifting or wrestling, is often associated with left ventricular wall thickness that is near normal but disproportionate in relation to cavity size.[4]

Investigation of the occurrence of arrhythmias in athletes has mainly focused on endurance sports such as marathons, triathlons and cycling,[5,6] in which exercise is extreme and mainly dynamic.

In their article published in this issue of the *Journal*, Mert et al.[7] focus on athletes who participate in a popular sport (bodybuilding) that can be used as a model for studying the relationship between the type of physical exertion and the occurrence of cardiac arrhythmias. Bodybuilding is an intense but mainly static sport that induces cardiac pressure overload and hence considerable stress on the cardiac muscle. By inducing significant muscle hypertrophy and cardiac wall fibrosis, bodybuilding can alter the electrophysiological properties of the heart, potentially increasing susceptibility to malignant arrhythmias.

The study population and a control group were analyzed using standard methods of cardiovascular and systemic assessment, based on demographic, echocardiographic and electrocardiographic parameters, the latter including 24-hour Holter monitoring and measures of dispersion of ventricular repolarization.

☆ Please cite this article as: Bonhorst D. Prática desportiva e arritmias cardíacas. Rev Port Cardiol. 2018;37:703–705.

E-mail address: danielbonhorst@iprc.pt

The study offers an interesting approach to the debate concerning the relationship between sports and the heart, particularly the distinction between athlete's heart and a pathologic heart, the different types of cardiac adaptation and their potential consequences, the effects of intense static exercise on cardiac arrhythmias, and a discussion of possible arrhythmogenic mechanisms and how to assess their risk.

The echocardiographic findings were compatible with cardiac adaptation to a mainly static sport that induces pressure overload, leading to an increase in the relative wall thickness of the left chambers, with little effect on left chamber size.[8] The study also confirmed the association between static exercise and increased right cavity size[4] and pulmonary artery pressure.

Holter monitoring confirmed that sports participation and static exercise in particular can induce cardiac arrhythmias, with a significantly higher number of atrial and ventricular extrasystoles being recorded in bodybuilders than in controls.

However, in this respect, we believe that the study data can hardly be considered to identify a population at high arrhythmic risk. First, few ventricular extrasystoles (VEs) were found (mean of 5.5 VEs/hour in the study group); second, there were practically no complex arrhythmias (only two cases of non-sustained ventricular tachycardia); and finally, Holter monitoring has limited value for identifying arrhythmic risk, especially when there is no structural heart disease, as shown by the spontaneous variability of the arrhythmias documented.

Atrial arrhythmias are of less importance in determining arrhythmic risk, especially for the normal hearts of subjects in this study as shown by the echocardiographic and electrocardiographic findings, and in fact no cases of atrial fibrillation were found.

With regard to the mechanisms underlying the ventricular arrhythmias detected in the study population, the authors focused on the involvement in arrhythmogenesis of heterogeneity in ventricular repolarization.

It has long been suggested that ventricular repolarization is involved in the induction of arrhythmias and that analysis of repolarization can be used for stratification of arrhythmic risk in a range of conditions. Various structural and electrical alterations can affect this phase of the cardiac cycle, by altering refractory periods and conduction velocities in adjacent myocardial areas. This has aroused interest in analyzing spatial heterogeneity and temporal fluctuations in ventricular repolarization as indices of arrhythmogenicity.[9] The simplest method to assess repolarization is to analyze the QT interval or, better, corrected QT (QTc), either of which is a well-known indicator of arrhythmic risk if prolonged.

A more thorough assessment of arrhythmic risk will include analysis of QT dispersion, defined as the difference between the longest and shortest QT intervals on the 12-lead electrocardiogram (ECG). This is a better indicator of non-uniform prolongation of ventricular repolarization and heterogeneity of refractory periods.

Novel markers have been proposed that appear to be superior for assessing the degree of dispersion of left ventricular intramural repolarization and that in some pathological conditions correlate better with the risk of ventricular arrhythmia. They include Tp-e, the interval between the peak and the end of the T wave, and more recent markers such as the Tp-e/QT and Tp-e/QTc ratios.[10] Panikkath et al.[11] proposed that Tp-e prolongation corresponds to transmural dispersion of repolarization in the ventricular myocardium, a period during which epicardial cells have repolarized and are fully excitable, but M cells in the subendocardium are still in the process of repolarization and are vulnerable to the occurrence of early afterdepolarizations. If conditions are favorable, these early afterdepolarizations can trigger polymorphic ventricular tachycardia or ventricular fibrillation.

In the present study, Mert et al. found a positive correlation between the presence of ventricular arrhythmias and changes in these new indices that were not detectable by simple measurement of QT or QTc interval. This supports the notion that these novel markers may be useful to stratify arrhythmic risk in sports of this type.

Given the limitations of the study, its conclusions – that measurement of right ventricular dimensions by echocardiography and analysis of dispersion of repolarization on the ECG using the new indices may be useful in assessing arrhythmic risk in athletes performing strength (static) exercise – are only hypothetical, and remain to be confirmed in future studies with larger samples and with a sufficiently long follow-up of the athletes included. It would also be interesting to compare the value of these indices in bodybuilders with those of athletes who perform mainly dynamic (endurance) exercise, since the effects on the heart differ according to the type of exercise and the mechanisms of arrhythmogenesis may also be different.

Conflicts of interest

The author has no conflicts of interest to declare.

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