My Approach to “Athlete’S Heart”: Evaluation of the Different Types of Adaptation to Exercise

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Abstract
Exercise-induced adaptation may occur in amateur and professional athletes. This condition is commonly named “athlete’s heart”. The alterations observed include dilation of the heart chambers, increased myocardial thickness, improved ventricular filling, increased left ventricular trabeculation, dilation of the inferior vena cava, among others. These changes can also be observed in some heart diseases, such as dilated, hypertrophic and other cardiomyopathies (CMP). Thus, cardiac imaging tests are fundamental in identifying these alterations and in differentiating between “athlete’s heart” and possible heart disease.

Introduction
Exercise-induced structural and/or functional heart alterations have been the object of study for more than 100 years, with their first reports appearing at the end of the nineteenth and the beginning of the twentieth century, referent to a dilated heart and a lower heart rate. The development of complementary exams showed changes in electrocardiograms, chest X-rays, echocardiography, among other exams in athletes.

Exercise-induced cardiac remodelling depends on a wide range of variables, including the type of exercise, the intensity, and the duration of training, generally with a “dose-effect” relation. It is interesting to note that other variables are also involved in the adaptation, such as age, ethnicity, gender, and body size.

The growing number of amateur and professional athletes periodically submitted to a cardiological evaluation before participating in activities or during the years of sports practice has led to a challenge in recognizing exercise-induced adaptation and its difference from potentially fatal heart conditions, such as hypertrophic cardiomyopathy (CMP), dilated CMP, arrhythmogenic right ventricular CMP, and non-compacted myocardium.

Keywords
Athlete’s heart; ecocardiography; cardiomyopathy; adaptive cardiac remodeling

Types of exercise
Sports activities can be divided, in a simplified manner, into four categories: skill, power, endurance, and mixed. These activities mix, in greater or lesser quantity, isotonic (dynamic) and isometric (static) exercises, according to that described in Chart 1:

- Isotonic: Oxygen supply is increased, which can affect the heart's work. Dilation of the heart chambers occurs over time. Heart rate, cardiac output, and arterial pressure are increased, especially with sudden strong muscle contractions.
- Isometric: No change in oxygen supply. The heart chambers tend to dilate. The duration and intensity of exercise determine the dilation level.

Chart 1 – Types of exercise and their alterations in cardiovascular physiology

<table>
<thead>
<tr>
<th>Type of exercise</th>
<th>Characteristic</th>
<th>Cardiac output</th>
<th>Peripheral vascular resistance</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isotonic</td>
<td>Dynamic</td>
<td>Significant increase</td>
<td>Decrease</td>
<td>Volume overload</td>
</tr>
<tr>
<td>Isometric</td>
<td>Static</td>
<td>Slight increase</td>
<td>Increase</td>
<td>Pressure overload</td>
</tr>
</tbody>
</table>

Though explained well in the literature and with a consensus regarding the exercise-induced cardiac remodelling described in this text, it is important to emphasize that not all of the athletes present these adaptations in imaging exams.

For example, many studies conducted to evaluate the different ethnicities and modalities have shown the following...
prevalence of left ventricular myocardial thickness $\geq 12$ mm$^2$ (Chart 5).

One study evaluating elite athletes showed that 48% of them present a left ventricular diastolic diameter (LVDD) of above 55 mm, while only 14% present a LVDD $\geq 60$ mm. The average LVDD among women was $48.4 \pm 4.2$ mm (varying from 38 to 66 mm), whereas among men the average was $55.4 \pm 4.3$ mm (varying from 43 to 70 mm). There was a correlation between the LVDD and the body surface area ($r = 0.76; p < 0.001$). Figure 1 demonstrates an example of dilation of the left atrium and left ventricle in an amateur street race athlete.

It is important to highlight that in one study published by Caselli et al. in 2015 in the Journal of the American Society of Echocardiography, the left ventricular ejection fraction (LVEF) was normal throughout the studied population (1,145 Olympic athletes), with the lower limit for LVEF being 53%.

This same study also showed that the left atrial diameter was larger than that of non-athletes; however, no differences were found when the diameter was indexed for the body surface area. Among athletes, 10% presented an LA diameter of above the reference values. In addition, 5.5% of the athletes presented an LVDD of above 60 mm and only 2.6% showed a myocardial thickness $\geq 13$ mm.

In children and adolescent athletes, one can also observe a dilation in the LA and LV, and a slight increase in the myocardial thickness.

**Differentiation between the exercise-induced adaptation and heart diseases**

Some echocardiographic tools, such as the Tissue Doppler (TD) and cardiac magnetic resonance (CMR) aid in the differentiation between the “athlete’s heart” and heart diseases.

The exercise-induced alterations do not reduce the speed of the wave $e'$ or increase the $E/e'$ ratio.

Another important point, and which can aid in determining the differentiation in relation to heart diseases, is the absence of a decrease in myocardial deformation (Strain values) in the “athlete’s heart”, observing negative values of higher than -18%.

**Chart 2 – Characteristics of the exercises of different sports and intensity of the cardiac remodelling**

<table>
<thead>
<tr>
<th>Skill</th>
<th>Power</th>
<th>Endurance</th>
<th>Mixed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isotonic</td>
<td>+/-</td>
<td>+++/++++</td>
<td>+/+++</td>
</tr>
<tr>
<td>Isometric</td>
<td>+/-</td>
<td>+++/++++</td>
<td>++/++</td>
</tr>
<tr>
<td>Cardiac remodelling</td>
<td>+/-</td>
<td>+++</td>
<td>+/+++</td>
</tr>
</tbody>
</table>

**Chart 3 – Cardiac remodelling according to the characteristic of the exercise**

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Isotonic (dynamic)</th>
<th>Isometric (static)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Dilation</td>
<td>Minimum alteration in volume and without dilation</td>
<td></td>
</tr>
<tr>
<td>LV myocardial thickness</td>
<td>No alteration or minimal alteration</td>
<td>Increase</td>
</tr>
<tr>
<td>LV geometry</td>
<td>Excentric hypertrophy</td>
<td>Concentric hypertrophy</td>
</tr>
<tr>
<td>LA Dilation</td>
<td>Dilation and/or hypertrophy</td>
<td></td>
</tr>
<tr>
<td>RV Dilation</td>
<td>No alteration</td>
<td></td>
</tr>
<tr>
<td>RA Dilation</td>
<td>No alteration</td>
<td></td>
</tr>
</tbody>
</table>

$RV$: right ventricle; $LV$: left ventricle; $LA$: left atrium; $RA$: right atrium.

**Chart 4 – Adaptations observed with sports practice**

<table>
<thead>
<tr>
<th>Structure</th>
<th>Adaptation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved left ventricular filling</td>
<td>Wave $e' &gt; 9$ cm/s</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>$E/e'$ Ratio $&lt; 6$</td>
</tr>
<tr>
<td>Myocardial trabecular</td>
<td>Prominent trabeculations</td>
</tr>
</tbody>
</table>

Increase in trabecular volume

**Chart 5 – Prevalence of LV myocardial thickness $\geq 12$ mm$^2$ according to ethnicity**

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blacks</td>
<td>18%</td>
</tr>
<tr>
<td>Caucasians</td>
<td>2%</td>
</tr>
<tr>
<td>Asians</td>
<td>2%</td>
</tr>
<tr>
<td>Arabs</td>
<td>&lt; 1%</td>
</tr>
</tbody>
</table>
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In questionable cases, the CMR is also very important in the differentiation between the athlete’s heart and heart diseases, given that, in the athlete’s heart, there is no delayed enhancement, no hypersignal in T1, or myocardial fat (this final concept in the differentiation from right ventricular arrhythmogenic CMP).

According to the characteristics observed in each “athlete’s heart”, the diagnostic differentiation should be performed with hypertrophic CMP, dilated CMP, arrhythmogenic right ventricular CMP, and/or non-compacted myocardium. In some specific cases, this differentiation can be more challenging, since it can exist in a “grey area” with similar characteristics among the different heart diseases and the exercise-induced adaptations (Figure 2).

Below are some points that aid in the differentiation in relation to each of the following heart diseases (Chart 6).

**Conclusion**

The regular and intense practice of physical activity can induce a wide range of cardiovascular adaptations, including electric, structural, and functional adaptations. These exercise-induced adaptations can often coincide with alterations observed in some heart diseases. Cardiac imaging plays a key role in the differentiation between the “athlete’s heart” and pathological cardiovascular alterations, particularly in structural and functional evaluations.

**Author Contributions**

Conception and design of the research, acquisition of data, analysis and interpretation of the data, statistical analysis, obtaining financing, writing of the manuscript, critical revision of the manuscript for intellectual content: Mancuso FJN.

**Chart 6 – Elements that help in the differentiation between adaptation to exercise and cardiomyopathies**

- Dilated myocardial thickness (13 to 16 mm) – Findings that suggest hypertrophic CMP
  - Normal or reduced LVDD (< 54 mm)
  - Segmental hypertrophy of the LV
  - Obstruction of the left ventricular outflow tract
  - Alteration of the diastolic function (wave e’ < 8.0 cm/s and/or E/A relation < 1.0)
  - Left atrial dilation disproportional to the left ventricular dilation
  - Delayed enhancement in the CMR

- Heart chamber dilation – Findings that suggest dilated CMP
  - Left ventricular dilation disproportional to the other heart chambers
  - Left ventricular diastolic dysfunction
  - Alteration in segmental contractility
  - Reduced systolic volume
  - Delayed enhancement in the CMR

- Prominent Trabeculation in the LV – Findings that suggest non-compacted myocardium
  - Left ventricular dilation disproportional to the other heart chambers
  - Left ventricular systolic dysfunction
  - Trabeculation associated with the thinning of the myocardial wall
  - Delayed enhancement in the CMR

- Heart chamber dilation – Findings that suggest arrhythmogenic right ventricular CMP
  - Right ventricular dilation disproportional to the other heart chambers
  - Alteration in segmental contractility of the right and/or LV
  - Delayed enhancement in the right and/or LV in the CMR

CMR: cardiac magnetic resonance; LV: left ventricle; LVDD: left ventricular diastolic diameter.
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References